Australian Government
National Health and Medical Research Council
Natural Resource Management Ministerial Council

National Water Quality Management Strategy

Australian Drinking Water Guidelines 6 2011

Version 3.4 Updated October 2017

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AUSTRALIAN DRINKING WATER GUIDELINES 6

2011

VERSION 3.4

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TABLE OF UPDATES

Amendment type	Amendment detail	Date updated	Version number
Development of a new Chemical Fact Sheet - *lanthanum*	Chemical Fact Sheet on lanthanum has been developed following a Secondary Notification risk assessment by the National Industrial Chemicals Notification and Assessment Scheme.	September 2017	3.4
Text added to Chapter 6	Addition of guidance on rounding for guideline values. Addition of guidance on parent compounds and environmental transformation products. Addition of guidance on the use of screening assays.	November 2016	3.3
Text added to Chapter 10	Addition of guidance for comparing analytical results with rounded guideline values.	November 2016	3.3
Minor amendments	Editorial changes have been made to correct minor errors, and update references.	November 2016	3.3
Removal of text from Chapter 5 – Microbial Quality of Drinking Water	Historical overview text removed, duplicative text removed. Information on viruses deleted for consistency with other waterborne pathogen sections.	November 2016	3.3
Removal of Appendix 3 – National Water Quality Management Strategy	This Appendix is significantly out of date and has been deleted.	November 2016	3.3
Removal of Appendix 4 – Process Report	This Appendix is the process report for review of the ADWG prior to 2011.It has been published separately for historical purposes. References to Appendix 4 in the body of the ADWG has been deleted.	November 2016	3.3
Text added to Chapter 6 Minor amendments to Chapter 8, sections 8.5 to 8.8, and Table 8.2.	Interim guideline values for chemicals that have been detected in drinking water. Clarification of approval process for new drinking water chemicals, and modifications to inconsistencies to section 8.5 to 8.8.	February 2016	3.2
Minor amendments to Chapters 8 and 10, Tables 8.4 and 10.5, Information Sheets 1.4, 1.6 and 2.1, and Fact Sheets on *Campylobacter*, *Salmonella* and *Vibrio*.	Editorial changes have been made to correct minor errors, provide further clarification and context, and update references and contact details.	March 2015	3.1

Amendment type	Amendment detail	Date updated	Version number
Review and update of three Chemical Fact Sheets – *chloral hydrate, monochloramine* and *chlorine*	Chemical Fact Sheets on chloral hydrate (originally endorsed in 1996), monochloramine (last endorsed in 2011) and chlorine (last endorsed in 2011) have been reviewed and updated following a review of recent literature, including the World Health Organization guidelines for drinking water quality and to correct a rounding error. The guideline value for chloral hydrate has changed. The equivalent guideline value for monochloramine as Cl_2/L has changed, which also resulted in a consequential change to the chlorine Fact Sheet.	19 December 2014	3
Minor amendments to contents page and Table 10.5 in relation to changes to the Fact Sheets for chloral hydrate, monochloramine and chlorine	Editorial changes have been made to provide further clarification and update guideline values and naming conventions.	19 December 2014	3
Review and update of eight information sheets for water treatment operators on water disinfection. Includes: *Introduction to water treatment, Overview of water disinfection, Disinfection with chlorine, Disinfection with chloramine, Disinfection with chlorine dioxide, Disinfection with ozone, Disinfection with ultra-violet light, Other disinfectant*	These Information Sheets replace the previous Information Sheets which were developed in 2004 and were very general in nature. In revising the Information Sheets, the Water Quality Advisory Committee considered the information on water disinfection already included in the 2011 ADWG, international standards including the WHO Drinking-water Guidelines, and reviewed the recent literature on water disinfection. This information was integrated to produce the revised Information Sheets on disinfection of drinking water, which include additional information on practical aspects of water disinfection.	13 December 2013	2
Review and update of four chemical Fact Sheets – *benzene, toluene, ethylbenzene and xylenes*	Chemical Fact Sheets on Benzene, Toluene, Ethylbenzene and Xylenes (originally endorsed in 1996) have been reviewed and updated following a review of recent literature, including the World Health Organization guidelines for drinking water quality. The guideline values for these chemicals have not been changed as a result of the review.	13 December 2013	2
New resource – *Guidance for issuing and lifting a Boil Water Advisory*	A new resource has been developed to assist health and environment department officials determine when to issue and lift boil water notices following a drinking water contamination incident. This document has been developed by the WQAC at the request of jurisdictions, following recent natural disasters which led to possible drinking water contamination.	13 December 2013	2

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CHAPTER 1 INTRODUCTION

Chapter 1 Introduction

Safe drinking water is essential to sustain life. Therefore, every effort needs to be taken to ensure that drinking water suppliers provide consumers with water that is safe to use.

The ***Australian Drinking Water Guidelines*** (the ADWG) are intended to provide a framework for good management of drinking water supplies that, if implemented, will assure safety at point of use. The ADWG have been developed after consideration of the best available scientific evidence. They are designed to provide an authoritative reference on what defines safe, good quality water, how it can be achieved and how it can be assured. They are concerned both with safety from a health point of view and with aesthetic quality.

The ADWG are not mandatory standards; however, they provide a basis for determining the quality of water to be supplied to consumers in all parts of Australia. These determinations need to consider the diverse array of regional or local factors, and take into account economic, political and cultural issues, including customer expectations and willingness and ability to pay.

The ADWG are intended for use by the Australian community and all agencies with responsibilities associated with the supply of drinking water, including catchment and water resource managers, drinking water suppliers, water regulators and health authorities.

1.1 Guiding principles

The ADWG contain a great deal of information about management of drinking water systems, monitoring and the vast array of contaminants that may be present in drinking water. An ever-increasing knowledge base means that the document has continued to grow in both detail and complexity. Although the increased information needs to be included, a danger is that the fundamental principles vital to ensuring safe drinking water quality become obscured in the detail. These fundamental principles, described below, should always be remembered.

The greatest risks to consumers of drinking water are pathogenic microorganisms. Protection of water sources and treatment are of paramount importance and must never be compromised.

Waterborne pathogens can cause outbreaks of illness affecting a high proportion of the community and, in extreme cases, causing death. How much treatment is needed will depend on the level of protection of water supplies. Completely protected groundwater may not require treatment, but all other supplies will require continuous disinfection. If water supplies are not completely protected from human and livestock waste, filtration is likely to be required.

Disinfection is the single process that has had the greatest impact on drinking water safety. There is clear evidence that the common adoption of chlorination of drinking water supplies in the 20th century was responsible for a substantial decrease in infectious diseases. Disinfection will kill all bacterial pathogens and greatly reduce numbers of viral and most protozoan pathogens. Combined with protection of water sources from human and livestock waste, disinfection can ensure safe drinking water. In the absence of complete protection of source water, filtration is likely to be required to improve the removal of viruses and protozoa.

All waterborne disease outbreaks are avoidable. Pathogens can only cause disease and death in humans if water source protection, pathogen removal by disinfection or filtration, or integrity of distribution systems fail.Chemical by-products of disinfection have been suggested as potential health risks. However, the possibility of such health risks remains highly uncertain in comparison to the well-established risks

from inadequate disinfection and contamination of water supplies with pathogens. Therefore, although concentrations of by-products should be kept as low as possible, efforts to achieve this should never jeopardise effective disinfection.

The drinking water system must have, and continuously maintain, robust multiple barriers appropriate to the level of potential contamination facing the raw water supply.

The multiple barrier approach is universally recognised as the foundation for ensuring safe drinking water. No single barrier is effective against all conceivable sources of contamination, is effective 100 per cent of the time or constantly functions at maximum efficiency. Robust barriers are those that can handle a relatively wide range of challenges with close to maximum performance and without suffering major failure.

Although it is important to maintain effective operation of all barriers, the advantage of multiple barriers is that short-term reductions in performance of one barrier may be compensated for by performance of other barriers. Prevention of contamination provides greater surety than removal of contaminants by treatment, so the most effective barrier is protection of source waters to the maximum degree practicable. Knowing how many barriers are required to address the level of potential contamination in individual systems is important. This requires a thorough understanding of the nature of the challenges and the vulnerabilities of the barriers in place. In terms of reliability, there is no substitute for understanding a water supply system from catchment to consumer, how it works and its vulnerabilities to failure.

Finally, a robust system must include mechanisms or failsafes to accommodate inevitable human errors without allowing major failures to occur.

Any sudden or extreme change in water quality, flow or environmental conditions (e.g. extreme rainfall or flooding) should arouse suspicion that drinking water might become contaminated.

Disease outbreaks from drinking water are almost invariably linked to changes in measurable water quality parameters or to the failure of treatment processes to cope with extreme weather events such as high rainfall and flooding. Water treatment processes generally function best under steady state conditions, and performance can seriously deteriorate when there are major fluctuations in quality or flow. It is vitally important that water quality after treatment remain as constant as possible, no matter how much the quality of the source water varies. Operators and managers need to be aware of normal operating requirements, the measurement criteria that define normal operation and the enormous risks that can be associated with operating outside normal limits.

System operators must be able to respond quickly and effectively to adverse monitoring signals.

Sudden changes in water quality or flow are likely to be a sign of imminent problems; such variations should always trigger appropriate responses. Wherever possible, key processes should be monitored continuously. Operators and managers must have the knowledge and appropriate responsibility to implement the necessary responses, which could range from modifying treatment processes to, in extreme cases, advising health regulators to consider issuing public advice such as 'boil water' notices or shutting down water supplies.

Previous water quality failures or 'close calls' should be studied so that operators are aware of the relationship between operational indicators and subsequent water quality failures. Even seemingly small faults should be addressed because these can accumulate and lead to a serious incident. Many waterborne disease outbreaks are caused by a combination of faults.

System operators must maintain a personal sense of responsibility and dedication to providing consumers with safe water, and should never ignore a consumer complaint about water quality.

Consumers are the ultimate assessors of water quality. Consumers may not be able to detect trace concentrations of individual contaminants, but their ability to recognise change should not be discounted. In some cases, consumer complaints may provide valuable information on potential problems not detected by testing water quality or monitoring treatment processes. Water quality testing has limitations and there are many possibilities for contamination of water in reticulation systems after treatment. All consumer complaints should be investigated to ensure that otherwise undetected problems that might compromise drinking water safety have not occurred. Meeting reasonable consumer expectations and maintaining confidence in the water supply is vitally important.

Ensuring drinking water safety and quality requires the application of a considered risk management approach.

The process of keeping drinking water safe is one of risk management. This requires steering a sensible course between the extremes of failing to act when action is required and taking action when none is necessary. Lack of action can seriously compromise public health, whereas excessive caution can have significant social and economic consequences. Corrective action or system upgrades should be undertaken in a considered, measured and consultative manner. Failure to act when required (e.g. failing to shut down a system when disinfection is not working effectively) may lead to an outbreak of waterborne disease. Acting when not required (e.g. issuing a 'boil water' notice when that is not necessary) is usually less severe in the short term, but repeated occurrences waste resources and are likely to cause complacency in the long term, leading to failure to respond when it is truly necessary. Similarly, failing to install a treatment process when required could lead to waterborne disease; however, installing treatment processes that are not required could have a high financial cost and divert funds needed elsewhere.

Risk management is about taking a carefully considered course of action. As the obligation is to ensure safe water and protect public health, the balancing process must be tipped in favour of taking a precautionary approach.

1.2 About the ADWG

1.2.1 SCOPE OF THE ADWG

Drinking water is defined as water intended primarily for human consumption, either directly, as supplied from the tap, or indirectly, in beverages, ice or foods prepared with water. Drinking water is also used for other domestic purposes such as bathing and showering.

With the exception of bottled or packaged water, the ADWG apply to any water intended for drinking irrespective of the source (municipal supplies, rainwater tanks, bores etc) or where it is consumed (the home, restaurants, camping areas, shops etc). Bottled water and packaged water are subject to the ***Food Standards Code***[1]. The ADWG do not address water used for specialised purposes such as renal dialysis and some industrial purposes where water of a higher quality than that specified in the Guidelines may be required.

1 Food Standards Australia New Zealand (2011) *Food Standards Code. Standard 2.6.2 Non-Alcoholic Beverages and Brewed Soft Drinks* [http://www.foodstandards.gov.au/_srcfiles/Standard_2_6_2_Non_alco_bev_v110.pdf]

1.2.2 PURPOSE OF THE ADWG

The ADWG provide the authoritative Australian reference for use within Australia's administrative and legislative framework to ensure the accountability of drinking water suppliers (as managers) and of state and territory health authorities (as auditors of the safety of water supplies). The ADWG are not, however, mandatory legally enforceable standards.

With appropriate consultation with the community, the ADWG may be used directly as agreed levels of service or they may form the basis for developing local levels of service. In the case of health-related water quality characteristics, there is less latitude for variation because the safety of drinking water is paramount. However, with regard to aesthetic characteristics, what is acceptable or unacceptable depends on public expectations and can therefore be determined by water authorities in consultation with consumers, taking into account the costs and benefits of further treatment of the water. The ADWG provide a starting point for that process. The ADWG may also be used by a standards body for defining quality processes suitable for third party accreditation of a quality management system.

1.2.3 STRUCTURE OF THE ADWG

The remainder of this document is divided into five parts.

Part I deals with the management of drinking water quality.

- Chapter 2 summarises a preventive strategy for the management of drinking water quality. It outlines a Framework for developing the approach; explains the need for water suppliers to work in partnership with other agencies in implementing the Framework; describes the purpose, structure, benefits and application of the Framework; and illustrates how the Framework is related to other management approaches such as Hazard Analysis Critical Control Point (HACCP) and ISO 9001.
- Chapter 3 details the 12 elements of the Framework.
- Chapter 4 considers how the Framework can be applied to small water supplies.

Part II considers the characteristics of water.

- Chapters 5–7 present overviews of the microbial, physical and chemical, and radiological characteristics, respectively, that determine water quality.
- Chapter 8 provides information on chemicals commonly used in treatment of drinking water and how they affect water quality.

Part III considers the monitoring of the drinking water system.

- Chapter 9 provides an overview of monitoring.
- Chapter 10 details monitoring procedures for specific characteristics in drinking water.

Part IV presents information sheets for disinfection of drinking water, sampling and statistics.

Part V presents fact sheets on a wide range of individual water quality characteristics, arranged by category and alphabetically within each category. Each fact sheet contains, where appropriate, the guideline values (aesthetic or health-related, or both) and their derivation, a general description of the characteristic, typical values in Australian drinking water, methods for removing the characteristic from drinking water, measurement techniques and health considerations.

An Appendix gives additional guidance on certain elements of the Framework for Management of Drinking Water Quality. The Appendix is located at the end of the ADWG, together with a glossary.

1.3 Water quality characteristics

1.3.1 INTRODUCTION

The ADWG are concerned with the safety and aesthetic quality of drinking water for consumers. Drinking water does not need to be absolutely pure to be safe. Because water is such a good solvent, pure water containing nothing else is almost impossible to attain. What is required is that drinking water be safe to drink for people in most stages of normal life, including children over six months of age and the very old. It should contain no harmful concentrations of chemicals or pathogenic microorganisms, and ideally it should be aesthetically pleasing in regard to appearance, taste and odour.

The Guidelines are derived so as to take account of the needs of an individual through a normal lifetime, including changes in sensitivity that may occur between life stages. Those at greatest risk of waterborne disease are infants and young children, people who are debilitated or living under insanitary conditions, and the elderly. Sensitive sub-populations (including those who are severely immuno-compromised) should seek further medical advice.

A wide range of measurable characteristics, compounds or constituents can be found in water and may affect its quality. They fall into several categories:

- physical
- microbial
- chemical, including
  - inorganic chemicals
  - organic compounds
  - pesticides
- radiological.

Appearance, taste and odour are useful indicators of quality because they are generally the characteristics by which the public judges water quality. However, water that is turbid or coloured, or has an objectionable taste or odour, may not be unsafe to drink. Conversely, the absence of any unpleasant qualities does not guarantee that water is safe.

The safety of water in public health terms is determined by its microbial, physical, chemical and radiological quality; of these, microbial quality is usually the most important.

1.3.2 GUIDELINE VALUES

The ADWG include two different types of guideline value:

- a **health-related guideline value**, which is the concentration or measure of a water quality characteristic that, based on present knowledge, does not result in any significant risk to the health of the consumer over a lifetime of consumption;
- an **aesthetic guideline value**, which is the concentration or measure of a water quality characteristic that is associated with acceptability of water to the consumer; for example, appearance, taste and odour.

The guideline values should be used in two separate but complementary ways: as action levels for the short-term verification of drinking water quality, and as a means to assess performance over the longer term (e.g. over a 12-month period). Using a guideline value for short-term verification entails assessing whether individual results conform to the requirements of good quality water. If a value is exceeded, some form of immediate corrective action will generally be initiated. For example, if a guideline value for a health-related characteristic is exceeded, the response should be to take immediate action to reduce the

risk to consumers, and, if necessary, to advise the health authority and consumers of the problem and the action taken. If the characteristic affects only aesthetic quality, the action may be to advise the community of deterioration in water quality.

When guideline values are used in assessing overall performance (e.g. as presented in an annual report), the aim is to assess whether management strategies are effective. The assessment is used to identify emerging problems and to determine priorities for improvement. Resulting actions will generally be applied in the longer term.

The guideline values relate to the quality of water at the point of use (e.g. kitchen or bathroom tap). They apply to reticulated water at the consumer's tap, rainwater for drinking, and source water if it is to be used without prior treatment. This does not, however, imply that the drinking water supplier is responsible for water quality problems caused by plumbing or other factors within a consumer's property. However, although it is not possible to control consumers' actions, suppliers should consider how drinking water quality may be affected in private plumbing systems and provide appropriate information to consumers.

The drinking water supplier should ensure that the quality of water in the reticulation mains meets the guideline values or agreed levels of service. The drinking water supplier would normally monitor quality in a service pipeline directly off a water main selected to represent the quality of water in the system. This is not usually within a private consumer's property. However, it may sometimes be necessary to check at the consumer's tap, either to confirm that chosen distribution sampling points are representative for microbial monitoring, to investigate specific problems such as leaching of metals into water, or as a consumer service.

The guideline values define water that, based on current knowledge, is safe to drink over a lifetime; that is, it constitutes no significant risk to health. For most of the water quality characteristics discussed, there is a grey area between what is clearly safe and what is clearly unsafe. Often the latter has not been reliably demonstrated and the guideline values always err on the side of safety. Therefore, for most characteristics, occasional excursions beyond the guideline value are not necessarily an immediate threat to health. The amount by which and the duration for which any health-related guideline value can be exceeded without raising concerns for public health depends on the particular circumstances. Exceeding a guideline value should be a signal to investigate the cause and, if appropriate, to take remedial action. If the characteristic is health related, the relevant health authority should be consulted.

Nevertheless, the ADWG provide the minimum requirements for drinking water of good quality, both aesthetically and from a public health viewpoint. Water suppliers should adopt a preventive risk management approach, as stipulated in the ADWG, to maintain the supply of water at the highest practicable quality. The guideline values should never be seen as a licence to degrade the quality of a drinking water supply to that level.

1.4 Community consultation

The ADWG are intended to provide consumers with safe and aesthetically pleasing water, and ultimately it is consumers who will be the final judges of water quality. It is vitally important that consumers are viewed as active partners in making decisions about drinking water quality and the levels of service to be adopted. Community expectations and willingness to pay must be considered. It is the responsibility of drinking water suppliers to keep the community fully informed about water quality, existing problems and needs for improvement.

Consumers also need to be informed about their responsibilities in relation to domestic plumbing and of any possible issues associated with the interaction of mains water with this plumbing.

1.5 Development of the Guidelines

National guidance on drinking water was first published by the National Health and Medical Research Council (NHMRC) in 1972 as ***Desirable Standards for Public Water Supplies in Australian Capital Cities***, adopting the Biennial Conference of Engineers ***Criteria and Objectives for Water Quality for Capital Cities*** (1969). The NHMRC standards were updated in 1975 as *Recommended Quality Criteria for Drinking Water* and in 1977 as *Desirable Quality for Drinking Water*. In 1980, *Desirable Quality for Drinking Water* was revised and jointly published with the Australian Water Resources Council (AWRC). This was considered a significant advance in water quality management because, for the first time, water supply and health authorities in Australia combined to produce a single guideline document. The 1980 guidelines were based on published criteria and standards recommended by overseas and international agencies, in particular the 1971 *International Standards for Drinking Water* of the World Health Organization (WHO).

Following a review of the 1980 Guidelines, and taking into consideration the 1984 WHO *Guidelines for Drinking-Water Quality*, the NHMRC and the AWRC published the *Guidelines for Drinking Water Quality in Australia* in 1987.

In 1996, the NHMRC and the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ, formerly AWRC) published the ADWG. The Guidelines were based on working papers and assessments prepared by the WHO expert panels, and reflected recent improvements in understanding problems of water quality. Referenced material included scientific papers, Guidelines published by overseas agencies, issues papers prepared by Australian water authorities, and assessments made by the NHMRC. Only the key references were cited, particularly those that were used as a basis for determining guideline values.

The guideline values in the 1996 ADWG were based primarily on the latest WHO recommendations, and any departures from these were detailed in the text. It should be noted, however, that the WHO *Guidelines for Drinking Water Quality* seek to define drinking water which, as well as being safe, is aesthetically ***acceptable***, whereas the emphasis in the Australian Guidelines is on producing drinking water that is safe and of ***good*** aesthetic quality.

During the development of the ADWG, it became evident that undertaking a major review of the ADWG in the future would be time consuming and resource intensive. To improve development and ensure that the Guidelines continued to represent the latest scientific evidence, the NHMRC and ARMCANZ agreed to initiate a 'rolling revision' process for the ADWG. Through this process, the Guidelines would remain under constant revision, with specific issues identified for review as required.

In 1998, NHMRC and ARMCANZ established a joint committee, the Drinking Water Review Coordinating Group, to oversee and manage the review process. In 2001–2002, ARMCANZ and the Australia and New Zealand Environment Conservation Council were replaced with the Natural Resource Management Ministerial Council (NRMMC) and the Environment Protection and Heritage Council. The ADWG continue to be developed under the auspices of the NHMRC and NRMMC.

A major revision of the 1996 ADWG was published as the 2004 ADWG. Specialist panels developed the *Framework for Management of Drinking Water Quality*, outlined in Chapters 2 and 3, and the sections on microorganisms, physical quality, inorganic chemicals, organic chemicals, radiological quality and pesticides. The specialist panels and the joint committee included representatives from the NHMRC, water authorities, private industry, universities, departments of health, departments of water resources and others.

Chapter 8, *Drinking Water Treatment Chemicals*, was subsequently incorporated into the ADWG in 2006, the aim was to ensure that the chemicals used to produce drinking water are safe and appropriate for the purpose, and to provide the water industry with guidance on drinking water treatment chemicals.

The 2011 edition of the ADWG supersedes the 2004 Guidelines, as amended in 2006. Major differences between the current ADWG and the 2004 edition include revisions to the monitoring chapters (9 and 10) together with the information sheets on sampling and statistics, to achieve closer alignment with the *Framework for Management of Drinking Water Quality* (Chapter 3).

The 2011 edition also includes new pesticide fact sheets and revision of some existing microbiological and chemical contaminant fact sheets.

The ADWG is part of the National Water Quality Management Strategy. The strategy aims to 'achieve sustainable use of the nation's water resources by protecting and enhancing their quality while maintaining economic and social development'. It provides information and tools to help communities manage their water resources to meet current and future needs.

A regulatory impact statement (RIS), including a cost-benefit evaluation of regulatory alternatives, was not undertaken as part of this review. The Productivity Commission has determined that the NHMRC is not required to undertake an RIS as the Guidelines do not have a regulatory status (Productivity Commission 2000). Implementation of the Guidelines by the states and territories is at the discretion of each state and territory health department, usually in consultation with water suppliers, and should include an appropriate economic analysis prior to implementation.

1.5.1 ACKNOWLEDGMENTS

The NHMRC and NRMMC express gratitude to all the people who provided input into the development of the ADWG. The work is usually performed on an honorary basis and in addition to their usual work commitments, and has been crucial in the continued development of the ADWG.

1.6 Future revisions of the ADWG

The ADWG will continue to be subject to regular review by NHMRC and NRMMC, with representatives from national health, water, environmental and community organisations, supported by specialist panels.

Submissions for updating the ADWG should be forwarded to:

Chief Executive Officer
National Health and Medical Research Council
GPO Box 1421
Canberra ACT 2601

1.7 References

Productivity Commission (2000) Arrangements for Setting Drinking Water Standards: International Benchmarking. Commonwealth of Australia, Canberra.

PART I MANAGEMENT OF DRINKING WATER QUALITY

CHAPTER 2 FRAMEWORK FOR MANAGEMENT OF DRINKING WATER QUALITY: OVERVIEW

Chapter 2 Framework for Management of Drinking Water Quality: overview

This chapter introduces the Framework for Management of Drinking Water Quality (the Framework) and describes its purpose, benefits and structure. It outlines how the Framework can be applied and explains the importance of various agencies working in partnership with drinking water suppliers to apply the Framework successfully.

2.1 A preventive strategy from catchment to consumer

The most effective means of assuring drinking water quality and the protection of public health is through adoption of a preventive management approach that encompasses all steps in water production from catchment to consumer.

In the Australian water industry, risk management and quality management are increasingly being used as a means of assuring drinking water quality by strengthening the focus on more preventive approaches. Some water authorities have implemented management systems based on ISO 9001 Quality Management, ISO 14001 *Environmental Management*, AS/NZS 4360:2004 Risk Management and the Hazard Analysis Critical Control Point (HACCP) system that has been adopted internationally by the food industry.

These available frameworks provide generic requirements for organisations undertaking a diverse range of activities. As such, they are not intuitively translated to management of drinking water quality, and therefore result in a range of interpretations and applications within the water industry. Furthermore, management of drinking water quality from catchment to consumer poses several challenges that are unique to the water industry and that may not be sufficiently addressed in these models.

The Framework was developed to guide the design of a structured and systematic approach for the management of drinking water quality from catchment to consumer, to assure its safety and reliability.

The Framework incorporates a preventive risk management approach; it includes elements of HACCP, ISO 9001 and AS/NZS 4360:2004, but applies them in a drinking water supply context to support consistent and comprehensive implementation by suppliers.

The Framework addresses four general areas, which are described below and illustrated in Figure 2.1:

- **Commitment to drinking water quality management.** This involves developing a commitment to drinking water quality management within the organisation. Adoption of the philosophy of the Framework is not sufficient in itself to ensure its effectiveness and continual improvement. Successful implementation requires the active participation of senior executive and a supportive organisational philosophy.
- **System analysis and management.** This involves understanding the entire water supply system, the hazards and events that can compromise drinking water quality, and the preventive measures and operational control necessary for assuring safe and reliable drinking water.
- **Supporting requirements**. These requirements include basic elements of good practice such as employee training, community involvement, research and development, validation of process efficacy, and systems for documentation and reporting.
- **Review.** This includes evaluation and audit processes and their review by senior executive to ensure that the management system is functioning satisfactorily. These components provide a basis for review and continual improvement.

2.2 Structure of the Framework

The Framework includes 12 elements considered good practice for system management of drinking water supplies (Table 2.1).

Figure 2.1 *Framework for management of drinking water quality*

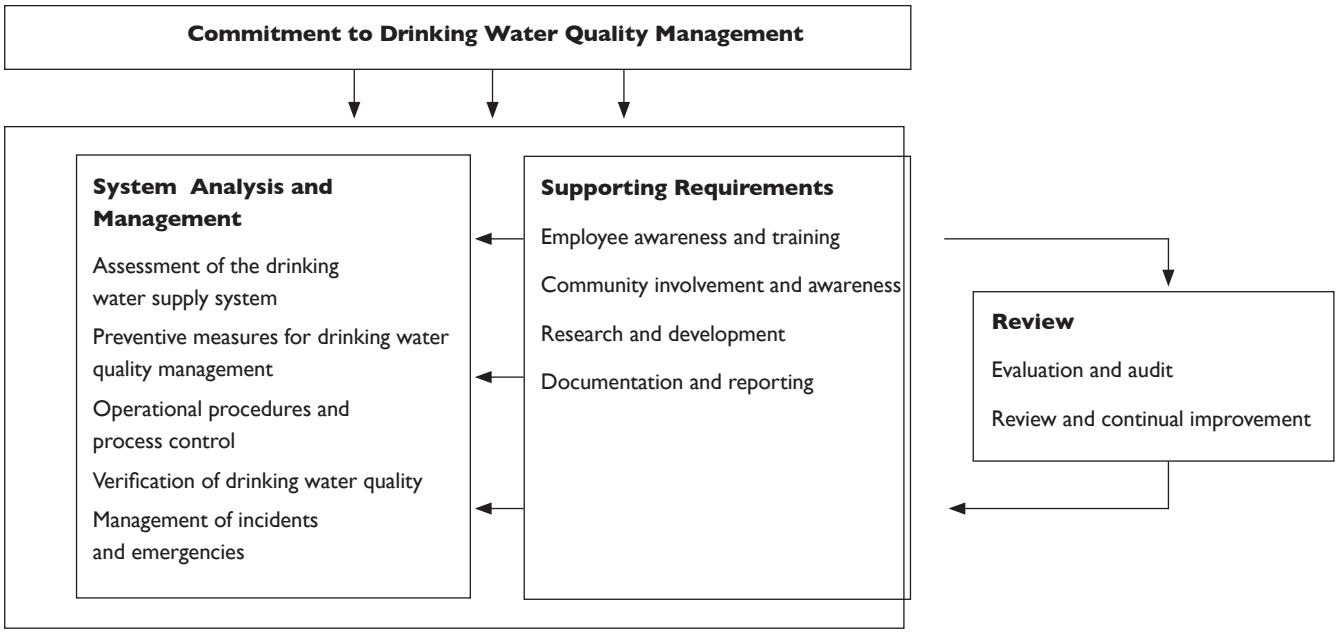


Table 2.1 *Framework for Management of Drinking Water Quality*

COMMITMENT TO DRINKING WATER QUALITY MANAGEMENT
Element 1 Commitment to drinking water quality management
Drinking water quality policy
Regulatory and formal requirements
Engaging stakeholders
SYSTEM ANALYSIS AND MANAGEMENT
Element 2 Assessment of the drinking water supply system
Water supply system analysis
Assessment of water quality data
Hazard identification and risk assessment
Element 3 Preventive measures for drinking water quality management
Preventive measures and multiple barriers
Critical control points
Element 4 Operational procedures and process control
Operational procedures
Operational monitoring
Corrective action
Equipment capability and maintenance
Materials and chemicals
Element 5 Verification of drinking water quality
Drinking water quality monitoring
Consumer satisfaction
Short-term evaluation of results
Corrective action
Element 6 Management of incidents and emergencies
Communication
Incident and emergency response protocols
SUPPORTING REQUIREMENTS
Element 7 Employee awareness and training
Employee awareness and involvement
Employee training
Element 8 Community involvement and awareness
Community consultation
Communication
Element 9 Research and development
Investigative studies and research monitoring
Validation of processes
Design of equipment
Element 10 Documentation and reporting
Management of documentation and records
Reporting
REVIEW
Element 11 Evaluation and audit
Long-term evaluation of results
Audit of drinking water quality management
Element 12 Review and continual improvement
Review by senior executive
Drinking water quality management improvement plan

Although listed as discrete components, the 12 elements are interrelated and each supports the effectiveness of the others. To assure a safe and reliable drinking water supply, these elements need to be addressed together because most water quality problems are attributable to a combination of factors.

The Framework outlines principles of management applicable to all water supply systems regardless of size and system complexity (i.e. both small and large supplies, ranging from those with minimal treatment to those with full treatment). To reflect the diversity of individual water supplies and the varying institutional arrangements (e.g. corporations, local authorities, wholesale, retail and contractors), the Framework is flexible. It provides generic guidance and the content should not be regarded as being prescriptive or exhaustive.

2.3 Benefits of the Framework

Management of drinking water quality through a comprehensive preventive strategy benefits the water industry by providing an overall framework that:

- promotes public health by assuring safer drinking water for consumers;
- enables an in-depth systematic evaluation of water systems, the identification of hazards and the assessment of risks;
- fosters a holistic approach to, and understanding of, management of drinking water quality;
- emphasises prevention and places drinking water quality monitoring in an appropriate verification role;
- introduces a common and standard approach throughout the industry, which establishes due diligence and credibility;
- provides the opportunity for various agencies and stakeholders to identify their areas of responsibility and become involved, and offers the outcome of a cooperative and coordinated approach with improved understanding of the responsibilities of all parties;
- provides a framework for communication with the public and with employees;
- addresses the uncertainties in setting accurate guideline values when insufficient scientific data are available;
- identifies future research needs for individual systems and throughout the water industry, and assists the development of improved risk assessment for specific hazards.

2.4 The need for multi-agency involvement

Restructuring of the water industry in Australia over recent years has increasingly transferred catchment and water resource management to agencies other than drinking water suppliers. These agencies may include water resource departments, natural resource and environment departments, agriculture departments, local governments, planning authorities, catchment water management boards, and community-based interest groups and organisations.

In some cases, restructuring has extended to dividing the traditional functions associated with the supply of drinking water, so that separate agencies are responsible for bulk water supply, water treatment and water reticulation. In addition, regulation of drinking water quality can take various forms. Health departments generally take a leading role in regulation; however, in some areas, specific water regulators may be established.

The Framework is intended to apply from catchment to consumer; as such, it addresses the necessity of inter-agency involvement. Drinking water suppliers are responsible for the quality of drinking water delivered to consumers and accordingly must show leadership in application of the Framework; however, implementation will generally require coordination and consultation with other agencies.

The range of agencies involved in individual water supply systems will need to be determined. Relevant agencies need to be encouraged to recognise their roles and responsibilities within the Framework, and to support drinking water suppliers through partnership agreements. The breadth and depth of partnership arrangements between agencies and the mechanisms by which they operate will vary between different jurisdictions, depending on the division of responsibilities and legislative authorities. If possible, a state- or territory-wide commitment to drinking water quality management and a formal coordination of responsible agencies should be developed (see Box 2.1).

Even where commitments and partnership agreements with other agencies are difficult to establish, the Framework should still be implemented. Gradually, as partnerships with other agencies are established, the Framework can be further improved and a more integrated approach developed.

***Box 2.1** Application of the Framework in Western Australia*

In Western Australia, drinking water quality management is a shared responsibility between the Water and Rivers Commission (WRC) and the Water Corporation of Western Australia (WCWA). The WRC is responsible for administration of catchment and source protection legislation; the WCWA is the major licensed drinking water supplier responsible for the collection, treatment and distribution of drinking water to consumers. Other key agencies in the supply of drinking water are the regulators, including the health authority, which provides interpretation and guidance on potential health impacts of drinking water quality.

A variation to the application of the Framework that is proposed by Western Australia is to apply the Framework at the state level using a whole-of-government approach, with each agency responsible for implementing the Framework within its areas of control and consulting with relevant partnership agencies. This approach requires a high level of commitment by all agencies, clear definition of accountabilities and responsibilities within the Framework, and increased communication and coordination of planning and management activities.

Under these circumstances, the WRC would be the lead agency to implement the catchment aspects of the Framework, with the WCWA a significant stakeholder. Downstream of the catchment, WCWA would be responsible for implementing the Framework in its areas of control. It is proposed that the Health Department, as the agency with responsibility for protecting public health, will have a key coordinating role in ensuring effective implementation and operation of the Framework.

2.5 Applying the Framework

Although the guidelines are not intended to be applied as standards, it is recognised that some jurisdictions may choose to regulate the guidelines through legislation or operating licences. In determining how the guidelines are translated into standards, operators and regulators should consider costs and benefits of these actions as well as developing an appropriate implementation timetable. The timetable should allow for endorsement of tools and processes used by water suppliers, and the establishment of mechanisms to ensure continual improvement. Just as important is an early determination and agreement on how the Framework will be monitored, audited and reported against. These aspects need clarification to ensure effective, unambiguous implementation.

Application of the Framework will vary depending on the arrangements for water supply within each jurisdiction; for example, in some states, water supply is managed by the one agency, whereas in other states it is managed locally by numerous water suppliers. This is likely to affect the manner and degree to which the Framework is implemented. However, all water suppliers and relevant government agencies should still be encouraged to use the Framework as a model for best practice.

How the Framework is applied will depend on the needs of the organisation, the separation of responsibilities and the institutional arrangements. Each organisation should develop an internal plan for implementing the Framework in a manner that suits its particular circumstances. The Framework can be applied as a stand-alone drinking water quality management system or can be integrated with an existing management system.

The time and resources required to develop a drinking water quality management system will depend on how many features of the Framework are already being practised and on how advanced existing management systems are. Current management applied by most drinking water suppliers and associated agencies will already incorporate many of the elements specified in the Framework. However, existing practices may not be sufficiently comprehensive to address fully the range of drinking water quality issues that can arise, and may not be systematically structured or sufficiently visible to ensure that all employees know and understand the system. In many instances, all that may be needed is to review, document and formalise these practices and address any areas where improvements are required.

The first step in initiating a drinking water quality management system based on the Framework is to identify appropriate personnel with defined roles and responsibilities. Establishing a core group with the necessary skills will help to ensure consistency throughout the implementation. This group can be supplemented by other expertise as necessary when dealing with specific issues. One option is to establish a water quality committee or water quality department with responsibility for the implementation and ongoing management of the overall system.

Some elements of the Framework will require more effort than others, and improvements may need to be prioritised and implemented sequentially. Additional guidance on two elements of the Framework – *Assessment of the water supply system* (element 2) and *Preventive measures for drinking water quality management* (element 3) – is provided in the Appendix. To assist with implementation of the Framework, users are encouraged to draw on the numerous sources providing detailed technical guidance (see Section A9 of the Appendix).

The most important step is getting started. Documenting current practice is often the most effective way to begin. However, in doing this it is important not to get involved in so much detail that making progress on implementing the Framework is inhibited. Documentation of the drinking water quality management system should make maximum use of existing documentation where that is adequate. A manual should be developed to provide an overview of the system and a summary of all relevant documentation.

Training personnel, including senior executives, in quality and risk management methods such as ISO 9001 and HACCP may assist in the development and implementation of a drinking water quality management system. Where necessary, help from outside experts should be sought to facilitate implementation of the Framework.

Effective management systems are not static and must be capable of accommodating change such as catchment developments, emerging issues, advances in technology or new institutional arrangements. Development should be an ongoing and iterative process whereby performance is continually evaluated and reviewed.

2.6 Correlations of the Framework with other systems

The Framework is not intended to duplicate or replace management systems that are adequately working; rather, it is intended to be compatible and complementary. The Framework includes principles of established systems such as HACCP, ISO 9001 and AS/NZS 4360:2004, and is sufficiently flexible to allow implementation to be built on programs and systems already present in an organisation. However, the relationships between the Framework and these systems should be understood.

The HACCP system was developed for the food industry and has become an internationally recognised risk management system to prevent or reduce the health risks from hazards associated with food processing. It is designed primarily as a preventive system of control to assure product safety while reducing reliance on end-product testing.

The application of the HACCP system to drinking water supplies has received increasing recognition due to the many parallel issues in food and drinking water supply. The HACCP system comprises seven principles. These principles and the equivalent Framework elements are shown in Table 2.2.

The HACCP system offers a systematic approach to the identification of hazards and their prevention, with a particular focus on process control to ensure that preventive measures are operating effectively. HACCP was not designed to be a fully comprehensive management system but was intended to be added on to existing good management practices. Thus, its scope and application are limited in several important areas of the Framework such as commitment, stakeholder involvement, emergency response, employee training, community consultation, and research and development. Furthermore, while HACCP is aligned quite readily to the treatment component of drinking water supply, its application may not transfer as easily to the important areas of catchment and distribution systems.

Table 2.2 *Correlations between HACCP and the Framework*

HACCP	Framework for Management of Drinking Water Quality
1. Hazard identification and preventive measures	Water supply system analysis, hazard identification and risk assessment (element 2)
	Preventive measures and multiple barriers (element 3)
2. Critical control points	Critical control points (element 3)
3. Critical limits	Operational monitoring (element 4)
4. Monitoring system for each critical control point	Operational monitoring (element 4)
5. Corrective actions	Corrective action (elements 4 and 5)
6. Verification / validation	Equipment capability and maintenance (element 4)
	Drinking water quality monitoring, consumer satisfaction (element 5)
	Validation of processes, design of equipment (element 9)
	Audit of drinking water quality management (element 11)
7. Documentation and record keeping	Management of documentation and records (element 10)

ISO 9001 provides a generic framework that specifies requirements for quality management systems to address customer satisfaction by assuring a consistent end product. The standard puts emphasis on continuous improvement; it adopts a process model approach that sets out the responsibilities, processes and resources needed to achieve specified objectives with respect to quality.

Table 2.3 lists the detailed ISO 9001 requirements and identifies links and correlations with the Framework. While the Framework and ISO 9001 are compatible, the structures of the two are somewhat different and correlations between them are not as close as those with HACCP. Table 2.3 shows correlations of general themes and areas.

Table 2.3 *Correlations between ISO 9001 and the Framework*

ISO 9001	Framework for Management of Drinking Water Quality
Quality management system	
General requirements	See Section 2.5 Applying the Framework
Documentation requirements	Management of documentation and records (element 10)
Management responsibility	
Management commitment	Drinking water quality policy, regulatory and formal requirements (element 1)
	Review by senior executive, drinking water quality management improvement plan (element 12)
Customer focus	Regulatory and formal requirements (element 1)
	Community consultation (element 8)
Quality policy	Drinking water quality policy (element 1)

Table 2.3 *Correlations between ISO 9001 and the Framework (continued)*

Planning	Regulatory and formal requirements (element 1)
	Operational monitoring (element 4)
	Drinking water quality monitoring (element 5)
Responsibility, authority and communication	See Section 2.5 Applying the Framework
Management review	Long-term evaluation of results, audit of drinking water quality management (element 11)
	Review by senior executive, drinking water quality management improvement plan (element 12)
Resource management	
Provision of resources	Drinking water quality management improvement plan (element 12)
Human resources	Employee awareness and involvement, employee training (element 7)
Infrastructure	Equipment capability and maintenance (element 4)
	Design of equipment (element 9)
Work environment	
Product realisation	
Planning of realisation processes	Preventive measures and multiple barriers, critical control points (element 3)
Customer-related processes	Community consultation, communication (element 8)
	Regulatory and formal requirements (element 1)
Design and development	Investigative studies and research monitoring, validation of processes, design of equipment (element 9)
Purchasing	Materials and chemicals (element 4)
Production and service provision	Operational procedures, operational monitoring, corrective action, equipment capability and maintenance (element 4)
	Validation of processes (element 9)
Control of measuring and monitoring devices	Equipment capability and maintenance (element 4)
Measurement, analysis and improvement	
General	
Monitoring and measurement	Operational monitoring (element 4)
	Drinking water quality monitoring, consumer satisfaction (element 5)
	Audit of drinking water quality management (element 11)
Control of nonconforming product	Corrective action (elements 4 and 5)
	Incident and emergency response protocols (element 6)
	Reporting (element 10)
Analysis of data	Operational monitoring (element 4)
	Short-term evaluation of results (element 5)
	Long-term evaluation of results (element 11)
Improvement	Review by senior executive, drinking water quality management improvement plan (element 12)

ISO 9001 includes several aspects of the Framework, but in a general sense, and it does not always provide a good fit to the specific requirements of drinking water quality management. The most important limitation of ISO 9001 is that it fails to address the preventive requirements of system analysis, hazard identification and control, and risk assessment, which are all critical for effective management of drinking water quality. There are other limitations in the areas of stakeholder involvement (for stakeholders other than consumers), research and development, management of large-scale emergencies, communication and reporting.

There is scope to implement the Framework within the structure of these established systems by expanding them to encompass all the necessary elements for drinking water quality management. For example, when integrated, HACCP and ISO 9001 can satisfy many of the key elements for drinking water quality management. However, if established management systems are applied to meet the requirements for management of drinking water quality as outlined in the Framework, then it should be ensured that all the necessary elements of drinking quality management are addressed.

Table 2.4 provides a general comparison indicating the applicability of established quality and risk management systems to the Framework.

Table 2.4 *Comparison of features from various management frameworks*

Framework for Management of Drinking Water Quality	HACCP	ISO 9001 (2000)	AS/NZS 4360 (2004)
Commitment to drinking water quality management			
Drinking water quality policy		+++	+++
Regulatory and formal requirements	+++	+++	
Engaging stakeholders			
Assessment of the drinking water supply system			
Water supply system analysis	+++		
Assessment of water quality data			
Hazard identification and risk assessment	+++		+++
Preventive measures for drinking water quality management			
Preventive measures and multiple barriers	+++	+	+++
Critical control points	+++		
Operational procedures and process control			
Operational procedures	+	+++	
Operational monitoring	+++	+++	
Corrective action	+++	+++	
Equipment capability and maintenance	+	+++	
Materials and chemicals	+	+++	
Verification of drinking water quality			
Drinking water quality monitoring	+++	+++	+++
Consumer satisfaction		+++	
Short-term evaluation of results		+++	+
Corrective action	+++	+++	
Management of incidents and emergencies			
Communication			
Incident and emergency response protocols			
Employee awareness and training			
Employee awareness and involvement		+++	
Employee training	+++	+++	
Community involvement and awareness			
Community consultation		+++	+++
Communication	+	+	+++

Table 2.4 *Comparison of features from various management frameworks (Continued)*

Framework for Management of Drinking Water Quality	HACCP	ISO 9001 (2000)	AS/NZS 4360 (2004)
Research and development			
Investigative studies and research monitoring			
Validation of processes	+++	+++	
Design of equipment		+++	
Documentation and reporting			
Management of documentation and records	+++	+++	+++
Reporting			+++
Evaluation and audit			
Long-term evaluation of results		+	
Audit of drinking water quality management	+++	+++	+++
Review and continual improvement			
Review by senior executive	+++	+++	+
Drinking water quality management improvement plan		+++	

Notes:
+++ Aspect explicitly stated
+ Aspect not explicitly stated but interpreted to include

CHAPTER 3 Framework for Management of Drinking Water Quality: the twelve elements

Chapter 3 Framework for Management of Drinking Water Quality: the twelve elements

This chapter details the 12 elements that make up the Framework for Management of Drinking Water Quality (the Framework). Each element includes an introduction and a list of the components that make up that element, which are then described in further detail. A 'summary of actions' box heads each component, providing an overview of the steps involved in implementation.

Some elements of the Framework are more complex than others, and therefore require further explanation. The Appendix (located at the end of the Guidelines) provides additional information and guidance for two elements – *Assessment of the drinking water supply system* (element 2) and *Preventive measures for drinking water quality management* (element 3).

3.1 Commitment to drinking water quality management (element 1)

Components:
- Drinking water quality policy
- Regulatory and formal requirements
- Engaging stakeholders

Organisational support and long-term commitment by senior executive is the foundation to implementation of an effective system for drinking water quality management.

Successful implementation requires:

- an awareness and understanding of the importance of drinking water quality management and how decisions affect the protection of public health;
- the development of an organisational philosophy that fosters commitment to continual improvement and cultivates employee responsibility and motivation;
- the ongoing and active involvement of senior executive to maintain and reinforce the importance of drinking water quality management to all employees as well as those outside the organisation.

Senior executive should ensure that its actions and policies support the effective management of drinking water quality (e.g. appropriate staffing, training of employees, provision of adequate financial resources, active participation and reporting to the board or chief executive).

3.1.1 DRINKING WATER QUALITY POLICY

Summary of actions

- Formulate a drinking water quality policy, endorsed by senior executive, to be implemented throughout the organisation.
- Ensure that the policy is visible and is communicated, understood and implemented by employees.

Development of a drinking water quality policy is an important step in formalising the level of service to which the drinking water supplier is committed and in increasing focus on water quality management throughout the organisation. The policy provides the basis on which all subsequent actions can be judged. It should define the organisation's commitments and priorities relating to drinking water quality.

The drinking water quality policy should provide a basis from which more detailed policies and implementation strategies can be developed. As such, it should be clear and succinct, and should address broad issues and requirements of the organisation's commitment and approach to drinking water quality management. The policy may cover issues such as:

- commitment to drinking water quality management;
- the level of service provided;
- the involvement of employees;
- compliance with relevant regulations and other requirements;
- liaison and cooperation with relevant agencies including health departments and other regulators;
- communication with employees and the public;
- intention to adopt best practice management and multiple barriers;
- continual improvement in the management of drinking water quality.

Box 3.1 provides an example of a generic drinking water quality policy.

In developing the drinking water quality policy, the opinions and requirements of employees, consumers and other stakeholders should be considered.

Management should ensure that the policy is highly visible, continually communicated, understood and implemented by all employees of the organisation. It is the responsibility of all employees to support this commitment.

***Box 3.1** Example of a drinking water quality policy*

The organisation is committed to managing its water supply effectively to provide a safe, high-quality drinking water that consistently meets the NHMRC/NRMMC *Australian Drinking Water Guidelines*, and consumer and other regulatory requirements.

To achieve this, in partnerships with stakeholders and relevant agencies, the organisation will:

- manage water quality at all points along the delivery chain from source water to the consumer;
- use a risk-based approach in which potential threats to water quality are identified and balanced;
- integrate the needs and expectations of our consumers, stakeholders, regulators and employees into our planning;
- establish regular monitoring of the quality of drinking water and effective reporting mechanisms to provide relevant and timely information, and promote confidence in the water supply and its management;
- develop appropriate contingency planning and incident response capability;
- participate in appropriate research and development activities to ensure continued understanding of drinking water quality issues and performance;
- contribute to the debate on setting industry regulations and guidelines, and other standards relevant to public health and the water cycle;
- continually improve our practices by assessing performance against corporate commitments and stakeholder expectations.

The organisation will implement and maintain a drinking water quality management system consistent with the *Australian Drinking Water Guidelines* to manage effectively the risks to drinking water quality.

All managers and employees involved in the supply of drinking water are responsible for understanding, implementing, maintaining and continuously improving the drinking water quality management system.

Dated Signed by Responsible Officer

3.1.2 REGULATORY AND FORMAL REQUIREMENTS

Summary of actions

- Identify and document all relevant regulatory and formal requirements.
- Ensure responsibilities are understood and communicated to employees.
- Review requirements periodically to reflect any changes.

Drinking water quality management may be subject to a range of regulatory and other formal requirements such as:

- federal, state or territory legislation and regulation;
- operating licences and agreements;
- contracts and agreed levels of service;
- memoranda of understanding;
- industry standards and codes of practice.

All regulatory and formal requirements should be identified and documented. Individual drinking water suppliers need to understand their responsibilities in supplying water for their particular jurisdictions. Relevant information should be communicated to employees and a registry of relevant regulations and other requirements should be readily accessible for reference. This registry should be regularly reviewed and updated as necessary to reflect any changes.

3.1.3 ENGAGING STAKEHOLDERS

Summary of actions

- Identify all stakeholders who could affect, or be affected by, decisions or activities of the drinking water supplier.
- Develop appropriate mechanisms and documentation for stakeholder commitment and involvement.
- Regularly update the list of relevant agencies.

Several aspects of drinking water quality management require involvement with other agencies. For example, collaboration with the appropriate agency is necessary where catchments and source waters are beyond the drinking water supplier's jurisdiction. Similarly, consultation with relevant health and other regulatory authorities is necessary for establishing many elements of drinking water quality management, such as monitoring and reporting requirements, emergency response plans and communication strategies.

The range of agencies involved in individual water supply systems will vary depending on local organisational and institutional arrangements. Agencies may include:

- health and environment protection authorities;
- catchment and water resource management agencies;
- local government and planning authorities;
- non-government organisations;
- community-based groups;
- industry associations.

An integrated management approach with collaboration from all relevant agencies is essential for effective drinking water quality management. All major stakeholders that could affect (e.g. regulators, catchment boards) or be affected by (e.g. consumers, industry, plumbers) decisions or activities of the drinking water supplier should be identified. The list of stakeholders should be regularly updated.

The various agencies involved should be encouraged to define their accountabilities and responsibilities to support the drinking water supplier and, where appropriate, to coordinate their planning and management activities. Appropriate mechanisms and documentation should be established for stakeholder commitment and involvement. This may include establishing working groups, committees or task forces, with appropriate representatives, and development of partnership agreements, including signed memoranda of understanding.

3.2 Assessment of the drinking water supply system (element 2)

Components: Water supply system analysis
Assessment of water quality data
Hazard identification and risk assessment

Assessment of the drinking water supply system is an essential prerequisite for subsequent steps in which effective strategies for prevention and control of hazards are planned and implemented. This includes understanding the characteristics of the drinking water system, what hazards may arise, how these hazards create risks, and the processes and practices that affect drinking water quality.

The drinking water supply system is defined as everything from the point of collection of water to the consumer and can include:

- catchments, including groundwater systems;
- source waters;
- storage reservoirs and intakes;
- treatment systems;
- service reservoirs and distribution systems;
- consumers.

Water quality can be affected at each of these points and because they are all interrelated, integrated management is essential. Generally, a drinking water supplier is only responsible for delivery of water to the consumer's meter. However, although it is not possible to control consumers' actions, suppliers should consider how drinking water quality may be affected in private plumbing systems and provide appropriate information to consumers.

Additional guidance on this element is provided in the Appendix.

3.2.1 WATER SUPPLY SYSTEM ANALYSIS

Summary of actions

- Assemble a team with appropriate knowledge and expertise.
- Construct a flow diagram of the water supply system from catchment to consumer.
- Assemble pertinent information and document key characteristics of the water supply system to be considered.
- Periodically review the water supply system analysis.

Effective system management requires, first and foremost, an understanding of the water supply system from catchment to consumer. Each element of the water supply system should be characterised with respect to drinking water quality and the factors that affect it. This characterisation promotes understanding of the water supply system, and assists with identification of hazards and assessment of risks to water quality.

A team with appropriate knowledge and expertise should be assembled to carry out the analysis. The team should include management and operations staff from the drinking water supplier as well as representatives from relevant agencies. In most cases, consultation with other agencies will be required for the analysis of catchments, which should include the potential impacts of land uses on water quality and stream and river flows. Health and other regulatory agencies should also be involved.

A generalised flow diagram should be constructed describing the water supply system from catchment to consumer. The diagram should:

- outline all steps and processes, whether or not they are under control of the drinking water supplier;
- summarise the basic characteristics of each component;
- make explicit any characteristics that are unique to the system;
- be verified by field audits and checked by those with specific knowledge of the system.

The water supply system analysis should be reviewed periodically to incorporate any changes that occur, for example in land use, treatment processes or consumer distribution.

3.2.2 ASSESSMENT OF WATER QUALITY DATA

Summary of actions

- Assemble historical data from source waters, treatment plants and finished water supplied to consumers (over time and following specific events).
- List and examine exceedances.
- Assess data using tools such as control charts and trends analysis to identify trends and potential problems.

A review of historical water quality data can assist in understanding source water characteristics and system performance both over time and following specific events such as heavy rainfall. This can help in identifying hazards and aspects of the drinking water system that need improvement.

Where available, water quality data should be assessed from monitoring of source waters, the operation of treatment processes, and drinking water as supplied to consumers. Trends analysis and control charts can be valuable tools for recognising potential problems or hazards and the accumulation of any gradual changes or cumulative effects.

Further information is provided in Section 10.3 and Information Sheet 3.2.

3.2.3 HAZARD IDENTIFICATION AND RISK ASSESSMENT

Summary of actions

- Define the approach and methodology to be used for hazard identification and risk assessment.
- Identify and document hazards, sources and hazardous events for each component of the water supply system.
- Estimate the level of risk for each identified hazard or hazardous event.
- Evaluate the major sources of uncertainty associated with each hazard and hazardous event and consider actions to reduce uncertainty.
- Determine significant risks and document priorities for risk management.
- Periodically review and update the hazard identification and risk assessment to incorporate any changes.

Effective risk management requires identification of all potential hazards, their sources and hazardous events, and an assessment of the level of risk presented by each. A structured approach is important to ensure that significant issues are not overlooked and that areas of greatest risk are identified.

In this context:

- A **hazard** is a biological, chemical, physical or radiological agent that has the potential to cause harm.
- A **hazardous event** is an incident or situation that can lead to the presence of a hazard (what can happen and how).
- **Risk** is the likelihood of identified hazards causing harm in exposed populations in a specified timeframe, including the severity of the consequences.

The distinction between hazard and risk is important: attention and resources need to be directed to actions selected primarily on the basis of level of risk, rather than just the existence of a hazard.

To give an example, the protozoan parasite *Cryptosporidium parvum* is a hazard; failure at a water treatment plant leading to *C. parvum* passing into the distribution system is a hazardous event; and the likelihood of the organism being present in source water and passing through the treatment plant in sufficient numbers to cause illness is a risk.

Realistic expectations of hazard identification and risk assessment are important. Rarely will enough knowledge be available to complete a detailed quantitative risk assessment. Hazard identification and risk assessment are predictive activities that will often include subjective judgments, and will inevitably contain uncertainty. Given these inherent limitations, flexibility is vital, to ensure an effective response when the unexpected occurs. Staff should have a realistic understanding of the limitations of these predictions, and this should also be conveyed to the public.

A consistent methodology should be established for both hazard identification and risk assessment. The methodology needs to be transparent and fully understood by everyone involved in the process. Staff should be included and need to be aware of the outcomes of the risk assessment.

Hazard identification

A comprehensive list of potential hazardous agents in drinking water is provided in Part V. Hazardous agents include microbial, chemical, physical and radiological agents. All potential hazards, sources and events that can lead to the presence of these hazards (what can happen and how) should be identified and documented for each component of the water supply system, regardless of whether or not the component is under the direct control of the drinking water supplier. This includes point sources of

pollution (e.g. human and industrial waste discharges) as well as diffuse sources (e.g. those arising from agricultural and animal husbandry activities). Continuous, intermittent or seasonal pollution patterns should also be considered, as well as extreme and infrequent events such as droughts or floods.

The hazard identification and risk assessment should be reviewed and updated periodically because changing conditions may introduce important new hazards or modify risks associated with identified hazards.

Risk assessment

Once potential hazards and their sources have been identified, the level of risk associated with each hazard or hazardous event should be estimated so that priorities for risk management can be established and documented. Although there are numerous contaminants that can compromise drinking water quality, not every potential hazard will require the same degree of attention.

The level of risk for each hazard or hazardous event can be estimated by identifying the likelihood of occurrence (e.g. certain, possible, rare) and evaluating the severity of consequences if the hazard were to occur (e.g. insignificant, major, catastrophic). The aim should be to distinguish between very high and low risks.

An example of an approach to estimating the level of risk is provided in Tables 3.1–3.3. These tables have been adapted from AS/NZS 4360:2004 (Risk Management), and can be modified to meet the needs of an organisation.

A likely outcome of risk assessment is the identification of specific areas where further information and research is required (see Box 3.7 in Section 3.9).

Risk prioritisation

Based on the assessment of risks, priorities for risk management and application of preventive measures can be established. Risk should be assessed at two levels:

- **maximum risk** in the absence of preventive measures; and
- **residual risk** after consideration of existing preventive measures.

Assessing maximum risk is useful for identifying high priority risks, determining where attention should be focused and preparing for emergencies. Residual risk provides an indication of the need for additional preventive measures.

Unforeseen and rare events

In well managed systems, problems should be rare, making them more challenging to anticipate and possibly to counter. This highlights the need to learn constructive lessons from the experiences of other Australian and international drinking water suppliers and water agencies. Many problems are triggered by short periods of sudden change, such as heavy rainfall or equipment failure. There are catalogues of waterborne disease outbreaks and the events that caused them. Some of these events should have been foreseeable while others have been attributable to more unusual or rare events. Maintaining awareness of such incidents can enable preventive measures to be implemented, to safeguard against similar occurrences (see Box 3.3 in Section 3.4).

Table 3.1 *Qualitative measures of likelihood*

Level	Descriptor	Example description
A	Almost certain	Is expected to occur in most circumstances
B	Likely	Will probably occur in most circumstances
C	Possible	Might occur or should occur at some time
D	Unlikely	Could occur at some time
E	Rare	May occur only in exceptional circumstances

Table 3.2 *Qualitative measures of consequence or impact*

Level	Descriptor	Example description
1	Insignificant	Insignificant impact, little disruption to normal operation, low increase in normal operation costs
2	Minor	Minor impact for small population, some manageable operation disruption, some increase in operating costs
3	Moderate	Minor impact for large population, significant modification to normal operation but manageable, operation costs increased, increased monitoring
4	Major	Major impact for small population, systems significantly compromised and abnormal operation if at all, high level of monitoring required
5	Catastrophic	Major impact for large population, complete failure of systems

Table 3.3 *Qualitative risk analysis matrix: level of risk*

Likelihood	Consequences				
	1 Insignificant	2 Minor	3 Moderate	4 Major	5 Catastrophic
A (almost certain)	Moderate	High	Very high	Very high	Very high
B (likely)	Moderate	High	High	Very high	Very high
C (possible)	Low	Moderate	High	Very high	Very high
D (unlikely)	Low	Low	Moderate	High	Very high
E (rare)	Low	Low	Moderate	High	High

Uncertainty

There will always be uncertainty associated with hazard identification and risk assessment. Uncertainty can be caused by a lack of knowledge or by variability in parameters. While variability can only be better understood (e.g. by improved characterisation of a hazard), uncertainty due to lack of knowledge can be reduced through better measurement and research. For example, uncertainty in our ability to identify the source, human infectivity or infectious dose of *Cryptosporidium* oocysts can be addressed through increased research.

Characterising the major sources and types of uncertainty can provide a better understanding of the limitations of the hazard identification and risk assessment and how these limitations can be reduced. Investigative studies and research monitoring can often provide further information for the risk assessment process and help to reduce uncertainty (see Section 3.9).

3.3 Preventive measures for drinking water quality management (element 3)

Components: Preventive measures and multiple barriers
Critical control points

Prevention is an essential feature of effective drinking water quality management. Preventive measures are those actions, activities and processes used to prevent hazards from occurring or reduce them to acceptable levels.

Hazards may occur or be introduced throughout the water system and preventive measures should be comprehensive, from catchment to consumer. Many preventive measures may control more than one hazard, while, as prescribed by the multiple barrier approach, effective control of some hazards may require more than one preventive measure. Preventive measures should be applied as close to the source as possible, with a focus on prevention in catchments rather than sole reliance on downstream control.

Planning of preventive measures should always be based on system-specific hazard identification and risk assessment. The level of protection to control a hazard should be proportional to the associated risk. Assessment of preventive measures involves:

- identifying existing preventive measures from catchment to consumer for each significant hazard or hazardous event;
- evaluating whether the preventive measures, when considered together, are effective in reducing risk to acceptable levels (i.e. residual risk – Section 3.2.3);
- if improvement is required, evaluating alternative and additional preventive measures that could be applied.

If additional measures are required, factors such as level of risk, benefits, effectiveness, cost, community expectations and willingness to pay should be considered. Preventive measures often require considerable expenditure, and decisions about water quality improvements cannot be taken in isolation from other aspects of water supply that compete for limited financial resources. Priorities will need to be established and many improvements may need to be phased in over time.

All preventive measures are important and should be given ongoing attention. However, some can significantly prevent or reduce hazards and are amenable to greater operational control than others. These measures could be considered as critical control points (see Section 3.3.2).

Additional guidance on this element is provided in the Appendix.

3.3.1 PREVENTIVE MEASURES AND MULTIPLE BARRIERS

Summary of actions

- Identify existing preventive measures from catchment to consumer for each significant hazard or hazardous event and estimate the residual risk.
- Evaluate alternative or additional preventive measures where improvement is required.
- Document the preventive measures and strategies into a plan addressing each significant risk.

Identifying and implementing preventive measures should always be undertaken within the context of a multiple barrier approach, so that failure of one barrier will be compensated by effective operation of the remaining barriers. This minimises the likelihood that contaminants will pass through the entire treatment system to be present in sufficient amounts to cause harm to consumers.

Traditional preventive measures are incorporated as or within a number of barriers, including:

- catchment management and source water protection;
- detention in protected reservoirs or storages;
- extraction management;
- coagulation, flocculation, sedimentation and filtration;
- disinfection;
- protection and maintenance of the distribution system.

The types of barriers required and the range of preventive measures employed will be different for each water supply and will generally be influenced by characteristics of the source water and surrounding catchment (see Box 3.2). Selection of appropriate barriers and preventive measures will be informed by hazard identification and risk assessment.

***Box 3.2** Examples of multiple barriers*

Large parts of Melbourne are supplied with high-quality source water from a highly protected catchment. Melbourne Water focuses much of its attention and resources on maintaining prevention of contamination at the source. The series of barriers for the majority of the water supply system include:

- protected forested catchments for harvesting of water with no human or livestock access;
- large catchment reservoirs with long detention times;
- additional retention time in seasonal storage systems;
- disinfection of water before it enters the distribution system;
- closed distribution systems.

In contrast, Adelaide is supplied with surface water derived from multi-use catchments and the Murray River, where there is limited control over activities with potential impacts on water quality. As a result, the barriers applied are heavily weighted towards water treatment and downstream control to remove turbidity and microorganisms. Barriers include the use of multiple storage reservoirs, coagulation, flocculation, sedimentation, filtration and disinfection with long contact times before supply.

Provision of residual disinfectant through large parts of the distribution system is also an important barrier for both systems.

Catchment management and source water protection

Catchment management and source water protection provide the first barrier for the protection of water quality. Where catchment management is beyond the jurisdiction of drinking water suppliers, the planning and implementation of preventive measures will require a coordinated approach with relevant agencies such as planning authorities, catchment boards, environmental and water resources regulators, road authorities and emergency services.

Effective catchment management and source water protection include the following elements:

- developing and implementing a catchment management plan, which includes preventive measures to protect surface water and groundwater;
- ensuring that planning regulations include the protection of water resources from potentially polluting activities, and are enforced;
- promoting awareness in the community of the impact of human activity on water quality.

Whether water is drawn from surface catchments or underground sources, it is important that the characteristics of the local catchment or aquifer are understood, and the scenarios that could lead to water pollution are identified and managed. The extent to which catchment pollution can be controlled is often limited in practical terms by competition for water and pressure for increased development in the catchment.

Effective catchment management has additional benefits. By decreasing contamination of source water, the amount of treatment and quantity of chemicals needed is reduced. This may lead to health benefits through reducing the production of treatment by-products, and economic benefits through minimising operational costs.

In surface water catchments, preventive measures can include:

- selection of an appropriate source water (where alternatives exist);
- exclusion or limitations of uses (e.g. restrictions on human access and agriculture);
- protection of waterways (e.g. fencing out livestock, management of riparian zones);
- use of planning and environmental regulations to regulate potential water-polluting developments (e.g. urban, agricultural, industrial, mining and forestry);
- use of industry codes of practice and best practice management;
- regulation of community and on-site wastewater treatment and disposal systems;
- stormwater interception.

Groundwater from depth is generally microbiologically safe and chemically stable; however, shallow or unconfined aquifers can be subject to contamination from discharges or seepages associated with agricultural practices (pathogens, nitrates and pesticides), septic tank discharges (pathogens and nitrates) and industrial wastes. Preventive measures for groundwater supplies should include protecting the aquifer and the local area around the borehead from contamination and ensuring the physical integrity of the bore (surface sealed, casing intact etc).

Further information on integrated catchment management is provided in Appendix Section A6 Preventive Measures and Multiple Barriers (Box A1) and the *National Water Quality Management Strategy: Implementation Guidelines*[2] (1998).

Detention in reservoirs or storages

Detention of water in reservoirs can reduce the number of faecal microorganisms through settling and inactivation, including solar (ultraviolet) disinfection. Most pathogenic microorganisms of faecal origin (enteric pathogens) do not survive indefinitely in the environment. Substantial die-off of enteric bacteria will occur over three to four weeks. Enteric viruses and protozoa will survive for longer periods (weeks to months).

Detention also allows suspended material to settle, which makes subsequent disinfection more effective and reduces the formation of disinfection by-products.

Other preventive measures in reservoirs and storages include:

- reservoir mixing or destratification to reduce growths of cyanobacteria (taste, odour and toxin production);
- excluding or restricting human, domestic animal and livestock access;
- diversion of local stormwater flows.

Extraction management

Where a number of water sources are available, there may be flexibility in the selection of water for treatment and supply. In such a situation it may be possible to avoid taking water from rivers and streams when water quality is poor (e.g. following heavy rainfall) in order to reduce risk and prevent problems in subsequent treatment processes.

2 Available at http://www.environment.gov.au/water/policy-programs/nwqms/

Within a single water body, selective use of multiple extraction points can provide protection against localised contamination, either horizontally or vertically through the water column (e.g. cyanobacterial blooms).

Coagulation, flocculation, sedimentation and filtration

Coagulation, flocculation, sedimentation (or flotation) and filtration remove particles, including microorganisms (bacteria, viruses and protozoa). It is important that operations are optimised and controlled to achieve consistent and reliable performance.

As an alternative to conventional media-based processes, membrane filtration provides a direct physical barrier and generally achieves a greater removal of microorganisms.

Care should be taken in the selection and use of water treatment chemicals as they may contain undesirable contaminants. In addition, there can be variation in performance of the same chemical obtained from different sources.

Disinfection

The most commonly used disinfection processes are chlorination and chloramination, but ozone, ultraviolet irradiation and chlorine dioxide are also used. These methods are very effective in killing bacteria and can be reasonably effective in inactivating viruses (depending on type) and many protozoa, including *Giardia*. *Cryptosporidium* is not inactivated by the concentrations of chlorine and chloramines that can be safely used in drinking water, and the effectiveness of ozone and chlorine dioxide is limited with this organism. However, there is some evidence that ultraviolet light might be effective in inactivating *Cryptosporidium*, and that combinations of disinfectants can enhance inactivation.

Storage of water after disinfection and before supply to consumers can improve disinfection by increasing contact times. This can be particularly important for microorganisms, such as *Giardia* and viruses.

Providing a disinfectant residual throughout the distribution system can provide protection against contamination and limit regrowth problems; however, the issue of disinfection by-products needs to be considered. Chloramination has proved successful in controlling *Naegleria fowleri* in water and sediments in long pipelines.

Protection and maintenance of the distribution system

Water distribution systems should be fully enclosed and storages should be securely roofed with external drainage to prevent contamination. Backflow prevention policies should be applied and monitored. There should also be effective maintenance procedures to repair faults and burst mains in a way that will prevent contamination. Positive pressure should be maintained throughout the distribution system. Appropriate security needs to be put in place to prevent unauthorised access to, or interference with, water storages.

Corrosion of pipes, including those on customer premises, can result in leaching of metals, with implications for public health (e.g. copper, cadmium and lead) or aesthetic quality (e.g. copper, iron and zinc). This should be monitored.

Growth or persistence of biofilms should be minimised to reduce aesthetic problems, including off-tastes, odours and staining.

Adequate training of maintenance workers, including contractors, responsible for the distribution system is essential because of the potential for contamination during repairs and recommissioning.

3.3.2 CRITICAL CONTROL POINTS

Summary of actions

- Assess preventive measures from catchment to consumer to identify critical control points.
- Establish mechanisms for operational control (see Section 3.4).
- Document the critical control points, critical limits and target criteria.

From among the preventive measures, critical control points should be identified for those hazards that represent a significant risk and require elimination or reduction to assure supply of safe drinking water.

A critical control point is defined as an activity, procedure or process at which control can be applied and which is essential to prevent a hazard or reduce it to an acceptable level.

Not all preventive measures are amenable to selection as critical control points. A critical control point has several operational requirements, including:

- operational parameters that can be measured and for which critical limits can be set to define the operational effectiveness of the activity (e.g. chlorine residuals for disinfection);
- operational parameters that can be monitored frequently enough to reveal any failures in a timely manner (online and continuous monitoring is preferable);
- procedures for corrective action that can be implemented in response to deviation from critical limits.

Critical limits are performance criteria that separate acceptability from unacceptability in terms of hazard control and water safety. They should be chosen carefully and should not be confused with target criteria (see Section 3.4.2). Critical limits may incorporate a numerical value as well as a consideration of time (e.g. failure to provide a minimum chlorine residual for a specified time).

Deviation from critical limits indicates loss of control of the process or activity and should be regarded as representing a potentially unacceptable health risk. Such events should result in immediate notification of the appropriate health regulator. Discussion of target criteria and critical limits is included in Section 3.4.2, and more detailed explanation of critical control points and their requirements is provided in Chapter 9 Section 9.4.3, 9.4.6 and the Appendix.

3.4 Operational procedures and process control (element 4)

Components:
Operational procedures
Operational monitoring
Corrective action
Equipment capability and maintenance
Materials and chemicals

The effectiveness of preventive measures is highly dependent upon the design and implementation of associated process control programs. To consistently achieve a high-quality water supply it is essential to have effective control over the processes and activities that govern drinking water quality.

Periods of sudden change and sub-optimal performance in the drinking water supply system can represent a serious risk to public health, as illustrated by the examples given in Box 3.3. Therefore, it is vital to ensure that all operations are optimised and are continuously controlled, and that barriers are functional at all times.

Process control programs support preventive measures by detailing the specific operational factors that ensure that all processes and activities are carried out effectively and efficiently. This includes a description of all preventive measures and their functions, together with:

- documentation of effective operational procedures, including identification of responsibilities and authorities;
- establishment of a monitoring protocol for operational performance, including selection of operational parameters and criteria, and the routine review of data;
- establishment of corrective actions to control excursions in operational parameters;
- use and maintenance of suitable equipment;
- use of approved materials and chemicals in contact with drinking water.

Effective implementation of these programs relies on the skills and training of operations staff. Operators should be proficient, have the ability to interpret the significance of changes in water quality and treatment, and be able to respond appropriately in accordance with established procedures (see Section 3.7).

Process control programs should be documented in operations manuals, with controlled copies readily accessible to all appropriate personnel. One option is to organise each manual into sections dealing with the individual components of the water supply system.

Documentation should include a description of:

- preventive measures and their purpose;
- operational procedures for relevant activities;
- operational monitoring protocols, including parameters and criteria;
- schedules and timelines;
- data and records management requirements;
- corrective actions to be implemented;
- maintenance procedures;
- responsibilities and authorities;
- internal and external communication and reporting requirements.

***Box 3.3** Examples of outbreaks resulting from sub-optimal performance*

Walkerton outbreak (Canada, 2000)

Over 2000 cases of illness were reported, including 26 cases of haemolytic uremic syndrome and seven deaths. Public health investigations confirmed that the most severe illnesses were caused by *Escherichia coli* 0157 and *Campylobacter*. The shallow groundwater supply appears to have been contaminated by cattle waste following heavy rains and localised flooding. A large number of faults have been proposed as potential contributing factors to the outbreak, including:

- reliance on bores subject to the direct influence of surface run-off, with only chlorination for treatment;
- operation and monitoring on the assumption that the bores were secure, deep groundwater sources;
- inadequate protection of surface catchments near the water supply bores;
- deficient chlorination practice;
- inadequate regulatory oversight;
- unreliable chlorine residual monitoring;
- failure to respond to the detection of contamination;
- failure to communicate the results to regulatory authorities;
- inadequate operator training and corporate commitment.

A public inquiry into the outbreak and its implications for the safety of drinking water elsewhere in Ontario resulted (O'Connor 2002a, b).

Box 3.3 *Examples of outbreaks resulting from sub-optimal performance (Continued)*

Milwaukee outbreak (United States, McKenzie *et al.* 1994)

Assessments indicate that over 400,000 illnesses were caused, including 4400 hospitalised. Premature deaths of at least 69 immunocompromised persons (most HIV positive) were recorded. The source of the contamination was not identified but it is considered that increased flows in rivers supplying Lake Michigan could have carried oocysts from livestock wastes or human sewage. Turbidity of the water taken from the lake deteriorated in the weeks preceding the outbreak.

Operation of one of the treatment plants supplying Milwaukee was not under optimal control. Although coagulant doses were adjusted, this did not prevent turbidity fluctuations in filtered water produced at one filtration plant (0.1–2.7 nephelometric turbidity units). Inexperience with the use of polyaluminium chloride, which had been a recent introduction, could have been a contributing factor. In addition, monitors intended to optimise coagulant doses during changes in water quality were not being used due to improper installation, and filtered water turbidimeters were not being used. Turbidity measurements were being taken every eight hours.

Recycling of backwash water through the filtration process could also have had an impact on the numbers of oocysts passing through the plant.

Other water treatment deficiencies associated with outbreaks of cryptosporidiosis have included:

- failure to respond to deterioration in source water quality;
- poor coagulation;
- poor monitoring of chemical dosing;
- inadequate flocculation;
- filters brought on line without backwashing.

3.4.1 OPERATIONAL PROCEDURES

Summary of actions

- Identify procedures required for processes and activities from catchment to consumer.
- Document all procedures and compile into an operations manual.

Operational procedures formalise the activities that are essential to ensure the provision of consistently good quality water. Detailed procedures are required for the operation of all processes and activities (both ongoing and periodic) from catchment to consumer, including preventive measures, operational monitoring and verification procedures, and maintenance requirements.

Procedures are most effective when operations staff are involved in their development, documentation and verification. This participation will help to ensure that all relevant activities are included, enhance operator training and awareness, and create commitment to operational and process control.

3.4.2 OPERATIONAL MONITORING

Summary of actions

- Develop monitoring protocols for operational performance of the water supply system, including the selection of operational parameters and criteria, and the routine analysis of results.
- Document monitoring protocols into an operational monitoring plan.

Operational monitoring includes the planned sequence of measurements and observations to assess and confirm the performance of preventive measures. Observations could include activities such as regular inspections of the catchment (e.g. for integrity of fences), plant equipment, wellhead protection areas and bore construction. Measurements are of operational parameters that will indicate whether processes are functioning effectively.

The general intent of operational monitoring is different from that of drinking water quality monitoring (see Section 3.5.1). Operational monitoring is used to confirm that preventive measures implemented to control hazards are functioning properly and effectively. Data from operational monitoring can be used as triggers for immediate short-term corrective actions to improve drinking water quality.

Key elements of operational monitoring include:

- development of operational monitoring plans from catchment to consumer, detailing strategies and procedures;
- identification of the parameters and criteria to be used to measure operational effectiveness and, where necessary, trigger immediate short-term corrective actions;
- ongoing review and interpretation of results to confirm operational performance.

Further guidance on operational monitoring is provided in Chapter 9.

Operational parameters

Operational parameters should be selected that reflect the effectiveness of each process or activity, and provide an immediate indication of performance. Typically, operational monitoring should focus on parameters that can be readily measured and enable a rapid response. To fulfil these requirements, surrogates are often used as operational parameters rather than direct measurement of the hazards themselves. For example, turbidity may be used as a surrogate for *Cryptosporidium* and *Giardia*. More detail on surrogates is provided in Chapter 9.

Operational parameters should be monitored with sufficient frequency to reveal any failures in good time. Online and continuous monitoring should be used wherever possible, particularly at critical control points (see below). Examples of parameters that can be used for operational monitoring are provided in Table 9.1, Chapter 9.

Target criteria and critical limits

Once operational parameters are identified, target criteria (performance goals) should be established for each preventive measure. These criteria can be quantitative (numerical) or qualitative (descriptive). Any deviation of performance from established targets should be regarded as a trend towards loss of control of the process, and appropriate action should be taken to resolve potential problems.

For preventive measures identified as critical control points for the water supply system, critical limits must also be defined and validated. A critical limit is a prescribed tolerance that distinguishes acceptable from unacceptable performance at a critical control point. When a critical control point is operating within the prescribed limits, performance in terms of hazard removal is regarded as being acceptable. However, exceedance of or deviation from a critical limit represents loss of control of a process and indicates an unacceptable health risk. Corrective actions should immediately be instituted to resume control of the process, and the health regulator should be notified.

Setting target criteria that are more stringent than critical limits at critical control points will enable corrective actions to be instituted before an unacceptable health risk occurs. Exceedance of a target criterion at a critical control point would generally not require that the health regulator be notified, providing corrective action successfully prevented deviation from a critical limit.

Chapter 9 provides more explanation of target criteria, critical limits and monitoring at critical control points.

Analysis of results

Results must be reviewed frequently to confirm that records are complete and accurate, and that there are no deviations from critical limits or target criteria. Where results indicate that control has been lost, appropriate corrective actions and process adjustments should be instituted to maintain quality. Those responsible for interpreting and recording operational results should clearly understand how the results should be assessed.

A system should be established for regular reporting of operational monitoring results to relevant staff and departments. Methods such as graphs or trend charts can be used to facilitate the interpretation of operational monitoring results. More guidance on short-term evaluation of results for assessing drinking water safety is provided in Section 10.2.

3.4.3 CORRECTIVE ACTION

Summary of actions

- Establish and document procedures for corrective action to control excursions in operational parameters.
- Establish rapid communication systems to deal with unexpected events.

Procedures should be developed for immediate corrective action to re-establish process control following failure to meet target criteria or critical limits. The procedures should include instructions on required adjustments, process control changes and additional monitoring. Responsibilities and authorities, including communication and notification requirements, should be clearly defined.

After implementing a corrective action, its effectiveness will need to be verified. This usually requires additional monitoring. Secondary impacts of the corrective action, and whether adjustments or action is needed further along in the supply system, should also be considered.

Examples of possible corrective actions include:

- selection of an alternative raw water source if available;
- altering the plant flow rate (e.g. reducing loading);
- jar testing for coagulant control and optimisation;
- altering the mixing intensity;
- changing treatment chemicals;
- using auxiliary chemicals such as coagulant aids, flocculant aids, filtration aids;
- adjusting pH;
- varying chemical feed rates and feed points;
- adjusting filtration loading rate or operation;
- increasing disinfectant dose;
- secondary or booster disinfection;
- mains flushing, cleaning and localised disinfection.

Where possible, the underlying cause of the problem should be determined and measures implemented to prevent future occurrences. Analysis of the causes may identify possible solutions, such as modifying an operating procedure or improving training. Details of all incidents should be recorded and reported.

While advance planning is important, it will not always be possible to anticipate every type of event. Rapid communication systems should be established to deal with these events.

Incident and emergency responses should be prepared for times when normal corrective actions cannot re-establish operational performance quickly enough to prevent drinking water of unacceptable quality from reaching consumers.

Section 10.2 provides more discussion of corrective actions and incident and emergency responses.

3.4.4 EQUIPMENT CAPABILITY AND MAINTENANCE

Summary of actions

- Ensure that equipment performs adequately and provides sufficient flexibility and process control.
- Establish a program for regular inspection and maintenance of all equipment, including monitoring equipment.

The capability of equipment is an important consideration in maintaining process control. Equipment and infrastructure in a drinking water supply system need to be adequately designed and of sufficient capacity (size, volume, detention times) to handle all flow rates (peak and otherwise) without limiting performance. Processes should not be hydraulically overloaded or subjected to rapid changes in hydraulic loading, as these conditions may compromise performance.

Design features that can improve performance and process control include:

- online measuring devices that monitor operational parameters continuously;
- automated responses to changes in water quality;
- 24-hour monitored alarm systems that indicate operational failure;
- backup equipment, including power generators;
- variable control of flow rates and chemical dosing;
- effective mixing facilities.

Design of new equipment and processes should undergo validation through appropriate research and development (see Section 3.9.).

Equipment used to monitor process performance should also be selected carefully. Monitoring equipment needs to be sufficiently accurate and sensitive to perform at the levels required. Wherever possible, monitoring should be online and continuous, with alarm systems to indicate when operational criteria have been exceeded. Monitoring failures should not compromise the system and in some cases, particularly at critical control points, backup equipment should be considered.

Staff should understand the operation of monitoring equipment so that causes of spurious results can be recognised and rectified.

Regular inspection and maintenance of all equipment from catchment to consumer is required to ensure continuing process capability. A maintenance program should be established and documented, detailing:

- operational procedures and records for the maintenance of equipment, including the calibration of monitoring equipment;
- schedules and timelines;
- responsibilities;
- resource requirements.

3.4.5 MATERIALS AND CHEMICALS

Summary of actions

- Ensure that only approved materials and chemicals are used.
- Establish documented procedures for evaluating chemicals, materials and suppliers.

The selection of materials and chemicals used in water systems is an important consideration as potentially they may have an adverse effect on drinking water quality. Chemicals added to water include disinfectants, oxidants, coagulants, flocculants, algicides, antioxidants and chemicals for softening, pH adjustment, fluoridation and scale prevention.

All chemicals used should be evaluated for potential contamination. General considerations include data on impurities, chemical and physical properties, maximum dosages, behaviour in water, migration and concentration build-up. In addition, the potential impact of water treatment chemicals on materials used in treatment plants needs to be considered. For example, ferric chloride used as a coagulant is extremely corrosive and can have severe effects on commonly used grades of stainless steel.

Chemical suppliers should be evaluated and selected on their ability to supply product in accordance with required specifications. Documented procedures for the control of chemicals, including purchasing, verification, handling, storage and maintenance, should be established to assure the quality of the chemicals at the point of application. Responsibilities for testing and quality assurance of chemicals (supplier, purchaser or both) should be clearly defined in purchase contracts.

Contaminants may also be introduced when water comes into contact with materials such as filter media, protective coatings, linings and liners, joining and sealing products, pipes and fittings, valves, meters and other components. Materials used should comply with Australian Standard AS/NZS 4020 ***Products for use in contact with drinking water***.

The products used in water systems should be subjected to an audited system of quality control.

3.5 Verification of drinking water quality (element 5)

Components: Drinking water quality monitoring
Consumer satisfaction
Short-term evaluation of results
Corrective action

Verification of drinking water quality provides an assessment of the overall performance of the system and the ultimate quality of drinking water being supplied to consumers. This entails both monitoring drinking water quality and assessing consumer satisfaction.

Verification provides:

- a useful indication of problems within the water supply system (particularly the distribution system) and the necessity for any immediate short-term corrective actions or incident and emergency response;
- confidence for consumers and regulators regarding the quality of the water supplied.

Section 9.5 provides more information on verification of drinking water quality.

3.5.1 DRINKING WATER QUALITY MONITORING

Summary of actions

- Determine the characteristics to be monitored in the distribution system and in water as supplied to the consumer.
- Establish and document a sampling plan for each characteristic, including the location and frequency of sampling.
- Ensure monitoring data are representative and reliable.

Drinking water quality monitoring is a wide-ranging assessment of the quality of water in the distribution system and, importantly, as supplied to the consumer. It includes regular sampling and testing to assess whether water quality is meeting guideline values and any regulatory requirements or agreed levels of service.

Monitoring of drinking water quality should be regarded as the final check that, overall, the barriers and preventive measures implemented to protect public health are working effectively. The purpose of drinking water quality monitoring is different from that of operational monitoring and the two types of monitoring also differ in what, where and how often water quality characteristics are measured. As it is neither physically nor economically feasible to test for all drinking water quality parameters equally, monitoring effort and resources should be carefully planned and directed at significant or key characteristics.

Key characteristics related to health include:

- microbial indicator organisms;
- disinfectant residuals and any disinfection by-products;
- any health-related characteristic that can be reasonably expected to exceed the guideline value, even if occasionally;
- potential contaminants identified in analysis of the water supply system (Section 3.2.1) and hazard identification (Section 3.2.3).

In addition to characteristics related to health, those with significant aesthetic impact (e.g. taste, odour) may also need to be monitored. Where these frequently reach unacceptable levels, further investigation may be needed to determine whether there are problems with significance for health.

Sampling locations will depend on the water quality characteristic being examined. Sampling at the treatment plant or at the head of the distribution system may be sufficient for characteristics where concentrations do not change during delivery; however, for those that can change during distribution, sampling should be undertaken throughout the distribution system, including the point of supply to the consumer.

Frequency of testing for individual characteristics will depend on variability, and whether the characteristics are of aesthetic or health significance. Sampling should be frequent enough to enable the monitoring to provide meaningful information. Sampling and analysis are required most frequently for microbial constituents, and less often for organic and inorganic compounds. This is because even brief episodes of microbial contamination can lead to immediate illness in consumers, whereas, in the absence of a specific event (e.g. chemical overdosing at a treatment plant), episodes of chemical contamination that would constitute an acute health concern are rare. Guideline values for most chemical parameters are based on impacts of chronic exposure.

Once parameters and sampling locations have been identified, these should be documented in a consolidated monitoring plan. Monitoring data should be representative, reliable and fully validated (see Box 3.4). Procedures for sampling and testing should also be documented.

Section 9.5.2 provides more information on the monitoring of drinking water quality.

Box 3.4 *Reliability of data*

Monitoring is only as good as the data collected, so every effort should be made to ensure that the data are representative, reliable and fully validated. Appropriate procedures should be in place and the following need to be considered.

Sampling plan:

- parameters measured, sampling locations, sampling frequency;
- qualifications and training of personnel;
- approved sampling methods and techniques;
- quality assurance and validation procedures for sampling;
- statistical validity.

Analytical testing:

- qualifications and training of personnel;
- suitability of equipment;
- approved test methods and laboratories;
- quality assurance and validation procedures (e.g. positive and negative control samples, inter-laboratory comparisons);
- accreditation with an external agency such as the National Association of Testing Authorities.

Monitoring equipment:

- calibration and inspection procedures to ensure control of monitoring equipment.

3.5.2 CONSUMER SATISFACTION

Summary of actions

- Establish a consumer complaint and response program, including appropriate training of employees.

Monitoring of consumer comments and complaints can provide valuable information on potential problems that may not have been identified by performance monitoring of the water supply system. Consumer satisfaction with drinking water quality is largely based on a judgment that the aesthetic quality of tap water is 'good', which usually means that it is colourless, free from suspended solids and has no unpleasant taste or odour.

Changes from the norm are particularly noticeable to consumers, who may interpret aesthetic problems as indicating health risks. A consumer complaint and response program operated by appropriately trained personnel should be established. Response targets should be set and regularly reviewed. Complaints and responses should be recorded and, in the longer term, the types, patterns and changes in numbers of complaints received should be evaluated.

One proactive approach to gauge perception of drinking water quality is to establish a consumer-based taste panel. Participants, who should be sensitive to off-flavours, can be trained with common flavour-profile descriptors so that their feedback to the drinking water supplier is more useful for identifying and solving aesthetic water quality problems. This approach can be particularly helpful in identifying recurring seasonal episodes of poor aesthetic quality. The fact sheet on Taste and Odour, in Part V, discusses consumer panels.

Sections 9.5.1 and 10.3.4 provide additional information on consumer satisfaction.

3.5.3 SHORT-TERM EVALUATION OF RESULTS

Summary of actions

- Establish procedures for the daily review of drinking water quality monitoring data and consumer satisfaction.
- Develop reporting mechanisms internally, and externally, where required.

Short-term performance evaluation entails the daily reviewing of drinking water quality monitoring data and consumer satisfaction to verify that the quality of water supplied to consumers conforms with guideline values. If the quality does not conform, then immediate corrective actions and/or incident and emergency response should be implemented.

Those responsible for interpreting and recording results should clearly understand how results should be assessed and, if required, how and where they should be communicated. Monitoring results should be reviewed within appropriate timeframes, and compared with previous results, established guideline values, and any regulatory requirements or agreed levels of service. Procedures for performance evaluation and recording of results should be established and documented. Mechanisms and responsibilities should be identified for the reporting of results internally to operators and senior executives as well as externally, where required, to stakeholders such as regulators and consumers (see Section 3.10.2).

Section 10.2 provides further discussion on short-term evaluation of results.

3.5.4 CORRECTIVE ACTION

Summary of actions

- Establish and document procedures for corrective action in response to non-conformance or consumer feedback.
- Establish rapid communication systems to deal with unexpected events.

If the short-term evaluation of drinking water quality monitoring data indicates non-conformance with guideline values or other requirements, an investigation should be initiated and, if necessary, corrective action taken as quickly as possible. Failure to take prompt and effective action may lead to the development of a more serious situation, which could require incident and emergency response protocols to be instituted. Corrective action could also be required in response to consumer feedback.

Corrective actions should be developed in consultation with relevant regulatory authorities and other stakeholders. Examples include:

- disinfection of tanks;
- flushing and maintenance of the distribution system;
- temporary shutdown of a treatment plant if adequate storage is available;
- increased booster or secondary disinfection;
- enhanced filtration;
- investigative or sanitary surveys of distribution systems.

Significant system failures that could pose a health risk or adversely affect water quality for an extended period require an immediate response and should also be reported to the relevant health authority (see Section 3.6).

Corrective actions should be documented, responsibilities and authorities clearly defined, and staff trained in appropriate procedures.

Section 10.2 provides further discussion on response to monitoring results that are outside specification.

3.6 Management of incidents and emergencies (element 6)

Components: Communication
Incident and emergency response protocols

Considered and controlled responses to incidents or emergencies that can compromise the safety of water quality are essential for protecting public health, as well as maintaining consumer confidence and the organisation's reputation. Although preventive strategies are intended to prevent incidents and emergency situations from occurring, some events cannot be anticipated or controlled, or the probability of their occurring is so low that providing preventive measures would be too costly. For such incidents, there must be an adaptive capability to respond constructively and efficiently.

Wherever possible, emergency scenarios should be identified, and incident and emergency protocols, including communication procedures, should be planned and documented. Establishing procedures 'on the run' is a recipe for inefficiency, lack of coordination, poor response times and potential loss of public confidence.

The development of appropriate protocols involves a review of the hazards and events that can lead to emergency situations, such as:

- non-conformance with guideline values and other requirements;
- accidents that increase levels of contaminants (e.g. spills in catchments, incorrect dosing of chemicals);
- equipment breakdown and mechanical failure;
- prolonged power outages;
- extreme weather events (e.g. flash flooding, cyclones);
- natural disasters (e.g. fire, earthquakes, lightning damage to electrical equipment);
- human actions (e.g. serious error, sabotage, strikes).

3.6.1 COMMUNICATION

Summary of actions

- Define communication protocols with the involvement of relevant agencies and prepare a contact list of key people, agencies and businesses.
- Develop a public and media communications strategy.

Effective communication is vital in managing incidents and emergencies. Clearly defined protocols for both internal and external communications should be established in advance, with the involvement of relevant agencies, including health and other regulatory agencies. These protocols should include a contact list of key people, agencies and businesses, detailed notification forms, procedures for internal and external notification, and definitions of responsibilities and authorities. Contact lists should be regularly updated (e.g. six-monthly) to ensure they are accurate.

Maintaining consumer confidence and trust during and after an incident or emergency is essential, and this is largely determined by how incidents and emergencies are handled. A public and media communication strategy should be developed before any incident or emergency situation occurs.

Draft public and media notifications should be prepared in advance and formatted for the target audience. An appropriately trained and authoritative contact should be designated to handle all communications in the event of an incident or emergency. All employees should be kept informed during any incident, because they provide informal points of contact for the community.

Consumers should be told when an incident has ended and be provided with information on the cause and actions taken to minimise future occurrences. This type of communication will help allay community concerns and restore confidence in the water supply. Interviews and surveys of a representative portion of the community are valuable for establishing consumer perceptions of events and how they were managed.

3.6.2 INCIDENT AND EMERGENCY RESPONSE PROTOCOLS

Summary of actions

- Define potential incidents and emergencies and document procedures and response plans with the involvement of relevant agencies.
- Train employees and regularly test emergency response plans.
- Investigate any incidents or emergencies and revise protocols as necessary.

Incident and emergency response protocols should be regarded as a priority. Potential incidents and emergencies should be defined and response plans should be developed and documented in advance to respond to these events.

Plans should be developed in consultation with relevant regulatory authorities and other key agencies, and should be consistent with existing government emergency response arrangements. In an emergency situation there will not be time to establish confidence and goodwill if these have not been established during normal operation. An investment in advance in building trust and understanding with parties who will be partners in responding to an emergency will pay important dividends in the form of more effective action when an emergency arises.

Key areas to be addressed in incident and emergency response plans include clearly specified:

- response actions, including increased monitoring;
- responsibilities and authorities of internal parties;
- responsibilities and authorities of parties external to the organisation;
- plans for emergency water supplies;
- communication protocols and strategies, including notification procedures (internal, regulatory body, media and public);
- mechanisms for increased health surveillance.

Employees should be trained in emergency response to ensure that they can manage any potential incidents or emergencies effectively. Incident and emergency response plans should be regularly reviewed and practised. This improves preparedness and provides opportunities to improve the effectiveness of plans before an emergency occurs.

Following any incident or emergency situation, an investigation of the incident or emergency should be undertaken and all involved staff should be debriefed to discuss performance and address any issues or concerns. The investigation should consider factors such as:

- What was the initiating cause of the problem?
- How was the problem first identified or recognised?
- What were the most critical actions required?

- What communication problems arose and how were they addressed?
- What were the immediate and longer-term consequences?
- How well did the protocol function?

Appropriate documentation and reporting of the incident or emergency should also be established. The organisation should learn as much as possible from the incident, to improve preparedness and planning for future incidents. Review of the incident may indicate necessary amendments to existing protocols.

Box 3.5 provides a summary of an emergency response protocol.

***Box 3.5** Water incident communication and notification protocol*

In South Australia, a protocol has been established between the Department of Human Services, South Australia Water, the Environmental Protection Agency (EPA) and the Department of Water Resources to ensure effective communication between government agencies in the event of incidents associated with reticulated water supplies. The protocol includes notification to other relevant bodies such as catchment water management boards and local authorities.

Incidents are classified as:

- Type 1 – potentially serious with either human health or environmental risks, or
- Type 2 – lesser incidents representing a low risk to human health or possible low impact and localised environmental harm.

The protocol includes agreed criteria for both raw water (e.g. cyanobacterial blooms, high numbers of *Cryptosporidium,* unacceptable concentrations of health-related chemicals and detection of pesticides) and treated drinking water (e.g. high turbidity in filtered water, chlorinator failure, detection of high concentrations of health-related chemicals, pesticides, *Cryptosporidium, Naegleria fowleri* and persistent *E. coli*/coliform bacteria).

The protocol defines the role of a water incident coordinator placed in the Department of Human Services and specifies which minister and agency will take the lead in dealing with and communicating incidents (incidents with health concerns are led by Department of Human Services, those with environmental concerns by the EPA, and those with operational concerns by South Australia Water).

Reporting requirements for individual agencies are defined, as well as communication requirements and protocols for the agencies, the water incident coordinator, offices of the ministers, and the lead minister.

The testing agency is required to report all Type 1 incidents immediately to the water incident coordinator and provide written confirmation within 24 hours by email or fax. The water incident coordinator ensures that all appropriate agencies have been notified and that relevant ministers are notified by their agencies as soon as possible and in any event within 24 hours.

Type 2 incidents are normally only notified to relevant agencies and generally do not require ministerial advice.

The protocol includes a list of 24-hour contacts for all agencies. Copies of the protocol are provided to all emergency contacts and relevant officers. The protocol is updated and reissued every six months.

3.7 Employee awareness and training (element 7)

Components: Employee awareness and involvement
Employee training

The knowledge, skills, motivation and commitment of employees and contractors ultimately determine a drinking water supplier's ability to operate a water supply system successfully. It is vital that awareness, understanding and commitment to performance optimisation and continuous improvement are developed and maintained within the organisation.

3.7.1 EMPLOYEE AWARENESS AND INVOLVEMENT

Summary of actions

- Develop mechanisms and communication procedures to increase employees' awareness of and participation in drinking water quality management.

An understanding of drinking water quality management is essential, to enable and motivate employees to make effective decisions. All employees of the drinking water supplier should be aware of:

- the organisation's drinking water quality policy;
- characteristics of the water supply system and preventive strategies in place throughout the system;
- regulatory and legislative requirements;
- roles and responsibilities of employees and departments;
- how their actions can impact on water quality and public health.

Mechanisms and communication procedures should be developed to ensure awareness of, and commitment to, drinking water quality management throughout the organisation. Methods to increase employee awareness can include employee education and induction programs, newsletters, guidelines, manuals, notice boards, seminars, briefings and meetings.

Employee participation and involvement in decision making is an important part of establishing the commitment necessary for the continuous improvement of drinking water quality management. Employees should be encouraged to participate in decisions that affect their jobs and areas of responsibility. Such participation provides a sense of ownership for decisions made and their implications. Open and positive communication is a foundation to creating a participatory culture, and employees should be encouraged to discuss issues and actions with management.

3.7.2 EMPLOYEE TRAINING

Summary of actions

- Ensure that employees, including contractors, maintain the appropriate experience and qualifications.
- Identify training needs and ensure resources are available to support training programs.
- Document training and maintain records of all employee training.

Employees and contractors must be appropriately skilled and trained in the management and operation of water supply systems, as their actions can have a major impact on drinking water quality and public health (see Box 3.6).

Employees should have a sound knowledge base from which to make effective operational decisions. This requires training in the methods and skills required to perform their tasks efficiently and competently, as well as knowledge and understanding of the impact their activities can have on water quality. For example, treatment plant operators should understand water treatment concepts and be able to apply these concepts and adjust processes appropriately to respond to variations in water quality.

Training needs should be identified and adequate resources made available to support appropriate programs. Examples of relevant areas to address are:

- general water quality;
- water biology and water chemistry;
- specific training to optimise system performance in areas such as:

- coagulant control testing;
- proper filtration operation;
- disinfection system operation;
- reticulation management;
- sampling, monitoring and analysis;
- interpretation and recording of results;
- maintenance of equipment.

Employees should also be trained in other aspects of drinking water quality management, including incident and emergency response, documentation, record keeping, reporting, and research and development.

Commonly used training techniques and methods include formal training courses accredited by a national training body, in-house training, on-the-job experience, mentor programs, workshops, demonstrations, seminars, courses and conferences. Training programs should encourage employees to communicate and think critically about the operational aspects of their work.

Training should be documented, and records maintained of all employees who have participated in training. Mechanisms for evaluating the effectiveness of training should also be established and documented. Training is an ongoing process and requirements should be regularly reviewed to ensure that employees maintain the appropriate experience and qualifications. For those activities that have a significant impact on drinking water quality, periodic verification of the capability of operations staff is necessary.

Where possible, accredited training programs and certification of operators should be employed.

***Box 3.6** Contractors*

With the considerable restructuring of the water industry in recent years, there is now a heavy reliance on contractors to undertake work for drinking water suppliers. These include contractors for construction, operations and maintenance of bulk water, treatment and distribution systems, and sampling and analytical work.

Contractors need to have the same awareness, training and culture as the organisation's employees. Requirements for contractor acceptability should be established, and contractors should be evaluated and selected on the basis of their ability to meet the specified requirements.

A drinking water supplier should ensure that contractors are qualified and have undergone appropriate training related directly to their task or role. When contracting labour, provisions should be made within the organisation to conduct the necessary education and training of contractors on the requirements for adherence to the organisation's policy and protocols.

Conditions under which the contractor operates should be clear, accurate and achievable, with scope for ongoing review and improvement. Partnerships will be more successful where the drinking water supplier retains sufficient knowledge and technical expertise to manage the contract efficiently.

3.8 Community involvement and awareness (element 8)

Components: Community consultation
Communication

Community consultation, involvement and awareness can have a major impact on public confidence in the water supply and the organisation's reputation. A communication program is a long-term commitment, including both consultation and education, and should be designed to provide an active, two-way exchange of information. This will help to ensure that consumers' needs and expectations are understood and are being satisfied.

3.8.1 COMMUNITY CONSULTATION

Summary of actions

- Assess requirements for effective community involvement.
- Develop a comprehensive strategy for community consultation.

Decisions on drinking water quality made by a drinking water supplier and the relevant regulatory authorities must be aligned with the needs and expectations of consumers. Therefore, the community and appropriate industry sectors should be consulted and involved during decision-making processes.

Discussions should include the establishment of levels of service, costs, existing water quality problems, and the options for protection and improvement of drinking water quality, including constraints on land use and changes in treatment or infrastructure. Consumers should also be consulted on monitoring requirements and mechanisms for public reporting of system performance.

Decisions and agreed levels of service should be based primarily on estimates of risk and cost, together with local knowledge of the source water (including the degree of catchment protection), treatment processes employed, history of the distribution system and the management of water quality. Consumer needs and expectations will influence the extent to which each community will adopt guideline values. For example, one community may choose to tolerate aesthetic problems, while another may choose to pay for treatment to bring water quality within commonly accepted limits.

Decisions about drinking water quality cannot be taken in isolation from other aspects of water supply that compete for limited financial resources. Two major decisions to be made are the levels of service to be provided, and the timeframe within which those levels can be achieved. Priorities will depend on the impact of water quality improvements on public health and on aesthetic considerations (taste, colour and odour). Public health should take a higher priority than aesthetics.

Assessing what is required for effective community involvement can be a complex task, depending on the issues and the community involved. Developing a community consultation strategy entails:

- defining the scope of the issue and the potential links with wider issues or problems. This will provide an indication of the extent of consultation or education required;
- identifying specific interest and stakeholder groups that may be affected, and their needs, existing level of knowledge and attitudes on the issues. All groups should be able to participate in the consultation process irrespective of barriers of language, distance, technical knowledge or lack of resources;
- presenting factual information to the community, consumers and groups in a form that is accessible, understandable and suitable as a basis for informed discussion;
- providing adequate time for consultation. The community should understand and agree to the process proposed for the consultation;
- identifying or developing measures to evaluate the effectiveness of the community consultation process.

Community consultation might include:

- briefings targeted to specific groups with interests or responsibilities;
- workshops or seminars on key issues or for special groups;
- focus groups and market research or surveys to determine community views, knowledge and attitudes;
- customer councils or customer panels;
- informative media programs targeting print media, radio and television;
- community education or information exchange programs;

- school programs;
- preparation of technical issues papers;
- media advertising of activities and available papers;
- public hearings for major and controversial initiatives.

Communications and community involvement should be considered when setting up a community consultation process or when working with, or seeking advice from, professionals in the areas of survey research. Records should be kept of all community consultation.

3.8.2 COMMUNICATION

Summary of actions

- Develop an active two-way communication program to inform consumers and promote awareness of drinking water quality issues.

Effective communication to increase community awareness and knowledge of drinking water quality issues and the various areas of responsibility is essential. Communication helps consumers to understand and contribute to decisions about the service provided by a drinking water supplier or land-use constraints imposed in catchment areas. A thorough understanding of the diversity of views held by individuals in the community is necessary to satisfy community expectations.

Effective communication is particularly important in the event of an incident or emergency (see Section 3.6).

A coordinated consumer information program should include:

- discussion of issues on drinking water quality, public health and risk assessment, cost of treatment, and levels of service;
- details of the water supply system and the drinking water quality management system;
- incident and emergency response plans, including procedures for notification when drinking water quality poses a health risk;
- consumer responsibilities beyond the meter and how drinking water quality may be affected in household distribution and use (e.g. use of suitable plumbing materials, point-of-use treatment devices, prevention of backflow);
- the need for further treatment of water for special purposes (e.g. renal dialysis, some industrial uses);
- the role and responsibility of the community in protecting water supply catchments and water conservation;
- commercial and industrial consumer responsibilities beyond the meter (e.g. the responsibility for design, maintenance, education of managers, and development of codes of practice that include reporting procedures in the event of contamination in large buildings).

Although a drinking water supplier is generally responsible only for delivery of water to the consumer's meter, consumers should be informed about how drinking water quality may be affected in household distribution and use.

Procedures should be established for disseminating information to promote awareness of drinking water quality issues to the community. Possible methods include annual or other periodic water quality reports, newsletters, notices in bills, workshops, seminars or briefings, media programs targeting radio and television, websites, treatment plant tours, catchment signage and school education programs.

Additionally, mechanisms such as a service line or complaint handling system should be established to provide opportunities for consumers to communicate their needs and expectations.

3.9 Research and development (element 9)

Components:	Investigative studies and research monitoring
	Validation of processes
	Design of equipment

A corporate commitment to conduct and participate in research and development activities on drinking water quality issues is important. Such a commitment helps to ensure continual improvement and the ongoing capability to meet drinking water quality requirements.

Applied research and development may be directed towards:

- increasing the understanding of a water supply system and potential hazards;
- investigating improvements, new processes, emerging water quality issues and new analytical methods;
- validation of operational effectiveness of new products and processes;
- increasing the understanding of the relationship between public health outcomes and water quality.

Research at a local level increases understanding of the specific characteristics of individual water supply systems. Local research could include, for example, detailed analysis of temporal and spatial variations in source water quality parameters. Research and development activities should also investigate mechanisms to improve and optimise plant performance, evaluate treatment processes (including the validation of critical limits and target criteria) and design new equipment. These activities should be carried out under controlled conditions by qualified staff, and all protocols and results should be documented and recorded.

Additionally, participation in research and development activities through partnerships and industry-wide cooperation can be a cost-effective way to address broader issues associated with water quality and treatment, including the development and evaluation of new technologies. Box 3.7 describes an example of a research activity. Opportunities for collaboration and initiation of joint research and development projects should be identified. Partnership organisations may include health and environment agencies, industry associations, other drinking water suppliers, university departments, cooperative research centres and community groups.

***Box 3.7** The Melbourne water quality study*

The Melbourne water quality study is an example of a large-scale, high-quality research study made possible through the collaboration of several organisations. The study was carried out by university researchers with the involvement of four water utilities, and was funded jointly by the water industry and the health regulator.

The study investigated the effect of microbial water quality on rates of community gastroenteritis in Melbourne by measuring the difference in the levels of illness among two population groups, each comprising approximately 300 households. One group consumed normal tap water and the other consumed water that was filtered and disinfected with ultraviolet radiation.

The principal aim of the study was to determine whether additional treatment of drinking water was necessary for an area served by a disinfected but unfiltered water supply drawn from protected catchments. The study used stringent epidemiological methodology and found no measurable difference in illness rates between the normal tap water group and the filtered water group, thus demonstrating that Melbourne's unfiltered drinking water does not make a significant contribution to gastroenteritis rates.

This groundbreaking project successfully addressed a water quality issue of international importance by shifting the focus from testing the microbial quality of drinking water to studying the health effects of drinking water. The study was a major undertaking but was completed for less than 1 per cent of the cost of building a water treatment plant.

Information from this research will enable better informed decisions about the management of Melbourne's water. Furthermore, the study has established a new methodology to assess the health effects of drinking water quality that is being employed in other cities to answer similar questions for different types of water supplies.

Source: Hellard *et al.* (2001)

3.9.1 INVESTIGATIVE STUDIES AND RESEARCH MONITORING

Summary of actions

- Establish programs to increase understanding of the water supply system.
- Use information to improve management of the water supply system.

Investigative studies and research monitoring include strategic programs designed to increase understanding of a water supply system, to identify and characterise potential hazards, and to fill gaps in knowledge. Improved understanding of the factors affecting water quality characteristics allows suppliers to anticipate periods of poor water quality and respond to them effectively.

Examples could include:

- baseline monitoring of parameters or contaminants or testing of potential new water sources to identify water quality problems;
- source water monitoring to understand the temporal and spatial variability of water quality parameters;
- developing early warning systems to improve the management of poor water quality;
- event-based monitoring to determine the magnitude of impacts (duration and maximum concentrations);
- examining mixing effects within a water storage;
- evaluating characteristics of an aquifer through pumping tests and analyses;
- studying the movement of water within reservoirs to determine short-circuiting effects;
- examining backwash return water and its effect in increasing microorganism load.

In addition, monitoring could provide input into predictive modelling of source water quality or assist in the selection of management and treatment approaches.

Careful consideration should be given to the selection of water quality characteristics to be analysed, use of statistical techniques, collection of samples (frequency and location), use of appropriate sampling and testing procedures, evaluation and management of results (see Information Sheets 2.1 to 3.5).

Tracing the cause of taste and odour problems often initiates investigations. Box 3.8 illustrates one such investigation and highlights the importance of investigative studies in assisting with evaluating risk to public health.

Box 3.8 *Cyanotoxin investigation in South Australia*

In April 2000, water quality problems were experienced at the Upper Paskeville Reservoir, a key water supply facility to the Yorke Peninsula in South Australia. Complaints of poor tastes and odours in the drinking water supplied to consumers were investigated. The problem was traced to the presence of high concentrations of 2-methyl isoborneol produced by the blue-green benthic cyanobacterium *Phormidium*, which was found in the reservoir entangled in strands of a submerged aquatic plant, water milfoil.

In view of the taste and odour complaints, Paskeville Reservoir was taken out of service. At the time, existing knowledge of *Phormidium* toxicity suggested that there would be no health concerns to the consumers of the Yorke Peninsula, but scientists at the Australian Water Quality Centre recommended that toxicity tests of cyanobacterial material be carried out as a precaution. The material was found to be toxic.

Following these results, the South Australian Department of Human Services issued advice that, due to the potential health risk, people should not use the mains water for drinking or cooking. Temporary supplies of bottled water were distributed to Yorke Peninsula communities by South Australia Water. Hospitals, nursing homes, caravan parks, food businesses and the like were notified individually and in some cases provided with carted water. The state primary industries department advised that the mains supply should not be used for stock water.

Subsequent testing confirmed that chlorination and boiling inactivated the toxin. On this basis the public was advised that the water could be used after boiling, and the strategy for cleaning the supply was changed from flushing to a mixture of chlorination and flushing. Chloramination, which is normally used to disinfect the supply, did not inactivate the toxin.

In addition to extending monitoring in similar storages in South Australia to determine the presence of *Phormidium* in benthic cyanobacterial growths, research is being undertaken to characterise the toxin further. Results so far have shown that the toxic effect is associated with cell-bound material and that the toxin is only sparingly soluble, thereby reducing its potential risk to human health.

Source: Baker *et al.* (2001)

3.9.2 VALIDATION OF PROCESSES

Summary of actions

- Validate processes and procedures to ensure that they are effective in controlling hazards.
- Revalidate processes periodically or when variations in conditions occur.

Validation involves evaluating the scientific and technical information that is available on processes and then, where necessary, undertaking further investigations, in order to validate system-specific operational procedures, critical limits and target criteria. The aim of process validation is to ensure effective operation and control. Historical data and operational experience can also be useful sources of information.

Processes should be revalidated on a regular basis or when variations occur (e.g. seasonally). Any new processes should be tested using benchtop, pilot-scale or full-scale experimental studies to confirm that the process and operational criteria produce the required results under the conditions specific to the individual water supply system.

Section 9.8 provides more information on validation of processes.

3.9.3 DESIGN OF EQUIPMENT

Summary of actions

- Validate the selection and design of new equipment and infrastructure to ensure continuing reliability.

Research and development should be undertaken to validate the selection and design of new equipment and infrastructure, or to confirm design changes necessary to improve plant performance and control systems. New technologies require pilot-scale research and evaluation before full-scale implementation. Design specifications should be established to ensure that new equipment will be able to meet the intended requirements and provide necessary process flexibility and controllability (see Section 3.4.4).

Other considerations for ensuring the reliability of water treatment systems include designing equipment and facilities to withstand natural disasters (e.g. earthquakes and flooding) and providing backup systems for emergency use (e.g. alternative power generation). Consideration of these factors during the design phase will reduce the risk that equipment failures will cause major disruptions in service.

3.10 Documentation and reporting (element 10)

Components: Management of documentation and records
Reporting

Appropriate documentation provides the foundation for the establishment and maintenance of effective drinking water quality management systems. Documentation should:

- demonstrate that a systematic approach is established and is implemented effectively;
- develop and protect the organisation's knowledge base;
- provide an accountability mechanism and tool;
- facilitate review and audits by providing written evidence of the system;
- establish due diligence and credibility.

Documentation provides a basis for effective communication within the organisation as well as with the community and various stakeholders. A system of regular reporting, both internal and external, is important to ensure that the relevant people receive the information needed to make informed decisions about the management or regulation of drinking water quality.

3.10.1 MANAGEMENT OF DOCUMENTATION AND RECORDS

Summary of actions

- Document information pertinent to all aspects of drinking water quality management.
- Develop a document control system to ensure current versions are in use.
- Establish a records management system and ensure that employees are trained to fill out records.
- Periodically review documentation and revise as necessary.

Documentation pertinent to all aspects of drinking water quality management is required. Documents should describe activities that are undertaken and how procedures are performed. They should also include detailed information on:

- preventive measures;
- critical control points, including specific operational procedures and criteria, monitoring and corrective actions;
- incident and emergency response plans;
- training programs;
- procedures for evaluating results and reporting;
- communication protocols.

Documentation should be visible and readily available to employees. Mechanisms should be established to ensure that employees read, understand and adhere to the documents.

Operation of systems and processes leads to the generation of large amounts of data that need to be recorded. Efficient record keeping is an essential tool for indicating and forewarning of potential problems, and providing evidence that the system is operating effectively.

Activities that generate records include:

- operational and drinking water quality monitoring;
- corrective actions;
- incident and emergency responses;
- training;
- research and development;
- assessment of the water supply system (flow diagrams, potential hazards etc);
- community consultation;
- performance evaluations, audits and reviews.

Documentation and records systems should be kept as simple and focused as possible. The level of detail in the documentation of procedures should be sufficient to provide assurance of operational control when coupled with a suitably qualified and competent operator. Retention of corporate memory should also be considered in documentation of procedures.

Mechanisms should be established to review documents periodically and, where necessary, to revise them to reflect changing circumstances. Documents should be assembled in a way that will allow any necessary modifications to be made easily. A document control system should be developed to ensure that current versions are in use and obsolete documents are discarded.

Records of all activities pertaining to the performance of drinking water quality management should be stored so that they can be easily accessed and reviewed. Storage should provide protection against damage, deterioration or loss. A system should be in place to ensure that employees are properly trained to fill out records, and that records are regularly reviewed by a supervisor, signed and dated.

Documents and records can be stored in a variety of forms, such as written documents, electronic files and databases, video and audiotapes, and visual specifications (flow charts, posters etc). Computer-based documentation should be considered to allow for faster and easier access as well as to facilitate updating.

3.10.2 REPORTING

Summary of actions

- Establish procedures for effective internal and external reporting.
- Produce an annual report to be made available to consumers, regulatory authorities and stakeholders.

Reporting includes the internal and external reporting of activities pertinent to drinking water quality management.

Internal reporting supports effective decision making at the various levels of the organisation, including operations staff and management, senior executive and the board of directors. It also provides a way to communicate information on decisions to employees throughout the organisation.

Internal reporting requirements should be defined and a system developed for communication between the various levels and functions of the organisation. Documented procedures (including definition of responsibilities and authorities) should be established for regular reporting (daily, weekly, monthly etc). These reports should include summaries of monitoring data, performance evaluation and significant operational problems that occurred during the reporting period. Results from audit and management reviews should also be communicated to those within the organisation responsible for performance.

External reporting ensures that drinking water quality management is open and transparent. It includes reporting to regulatory bodies, consumers and other stakeholders in accordance with requirements. External reporting requirements should be established in consultation with consumers and the relevant regulatory authorities; procedures for information dissemination should also be developed.

Agreement should be reached with health and other relevant regulators on requirements for:

- regular reports summarising performance and water quality data;
- event reports on significant system failures that may pose a health risk or adversely affect water quality for an extended period (see Section 3.6.2).

Reports should be provided to regulatory authorities on incidents defined in agreed incident and emergency response protocols. If necessary, the health authority can then ensure that public health concerns are reported to the community.

An annual report should be produced and made available to consumers, regulatory authorities and stakeholders. The annual report should:

- summarise drinking water quality performance over the preceding year against numerical guideline values, regulatory requirements or agreed levels of service, and identify water quality trends and problems;
- summarise any system failures and the action taken to resolve them;
- specify to whom the drinking water supplier is accountable, statutory or legislative requirements, and minimum reporting requirements;
- indicate whether monitoring was carried out in accordance with the principles of risk management set out in the *Australian Drinking Water Guidelines*, standards set by the regulator and any requirements contained in agreed levels of service.

Annual reports should contain sufficient information to enable individuals or groups to make informed judgments about the quality of drinking water and provide a basis for discussions about the priorities that will be given to improving drinking water quality. The annual report represents an opportunity to canvass feedback, and it should therefore encourage consumers and stakeholders to provide comment.

3.11 Evaluation and audit (element 11)

Components: Long-term evaluation of results
Audit of drinking water quality management

Long-term evaluation of drinking water quality results and audit of drinking water quality management are required to determine whether preventive strategies are effective and whether they are being implemented appropriately. These reviews enable performance to be measured against objectives and help to identify opportunities for improvement.

3.11.1 LONG-TERM EVALUATION OF RESULTS

Summary of actions

- Collect and evaluate long-term data to assess performance and identify problems.
- Document and report results.

The systematic review of monitoring results over an extended period (typically the preceding 12 months or longer) is needed to:

- assess overall performance against numerical guideline values, regulatory requirements or agreed levels of service;
- identify emerging problems and trends;
- assist in determining priorities for improving drinking water quality.

There will inevitably be occasions of non-conformance with operational criteria or numerical guideline values. Each event will need to be assessed and responses determined.

Mechanisms for evaluation of results should be documented, with responsibilities, accountabilities and reporting requirements defined. Useful tools to enhance the interpretation of data sets include statistical evaluation of results and graphs or trend charts using a 'control chart' format (see Information Sheets 3.1 to 3.4).

Evaluation of results should be reported internally to senior executive, and externally to consumers, stakeholders and regulatory authorities in accordance with established requirements (see Section 3.10.2). Providing assurance that data are reviewed regularly and that improvements are made in response to identified problems will contribute to consumer confidence.

Section 10.3 provides further more guidance on assessing long-term system performance.

3.11.2 AUDIT OF DRINKING WATER QUALITY MANAGEMENT

Summary of actions

- Establish processes for internal and external audits.
- Document and communicate audit results.

Auditing is the systematic evaluation of activities and processes to confirm that objectives are being met. It includes assessment of the implementation and capability of management systems. Auditing provides valuable information on those aspects of the system that are effective, as well as identifying opportunities to improve poor operational practices.

Periodic auditing of all aspects of the drinking water quality management system is needed to confirm that activities are being carried out in accordance with defined requirements and are producing the required outcomes.

Internal audits are important for maintaining a functional drinking water quality management system and for identifying areas for improvement. Internal audits will involve trained staff and should include a review of the management system and associated operational procedures, monitoring programs, and the records generated. The aim is to ensure that the system is being implemented correctly and is effective.

The frequency and schedule of audits should be defined, as should the responsibilities, requirements, procedures and reporting mechanisms. The audit process can take place over time but it should be comprehensive.

Drinking water agencies should consider mechanisms for establishing external auditing. Such auditing can be useful in establishing credibility and maintaining consumer confidence. External auditing could be achieved by peer review or be undertaken by an independent third party. External audits should focus on confirming implementation and results of internal audits.

External audits could be conducted on:

- the management system;
- operational activities;
- drinking water quality performance;
- the effectiveness of incident and emergency response or other specific aspects of drinking water quality management.

Audit results should be documented and communicated to management and personnel responsible for the department or function being audited. Results of audits should also be considered as part of the review by senior executive (see next section).

Section 10.3 provides additional information on review and continual improvement.

3.12 Review and continual improvement (element 12)

Components: Review by senior executive
Drinking water quality management improvement plan

Senior executive support, commitment and ongoing involvement are essential to the continual improvement of the organisation's activities relating to drinking water quality. Senior executive should regularly review its approach to drinking water quality management, develop action plans, and commit the resources necessary to improve operational processes and overall drinking water quality performance.

3.12.1 REVIEW BY SENIOR EXECUTIVE

Summary of actions

- Senior executive review of the effectiveness of the management system.
- Evaluate the need for change.

In order to ensure continual improvement, the highest levels of the organisation should maintain oversight of the effectiveness of the drinking water quality management system and evaluate needs for change.

Senior executive should review reports from audits, drinking water quality performance and previous management reviews. The review should also consider concerns of consumers, regulators and other stakeholders, and evaluate the suitability of the drinking water quality policy, objectives and preventive strategies in relation to changing internal and external conditions such as:

- changes to legislation, expectations and requirements;
- changes in the activities of the organisation;
- advances in science and technology;
- outcomes of drinking water quality incidents and emergencies;
- reporting and communication.

The review by senior executive should be documented.

3.12.2 DRINKING WATER QUALITY MANAGEMENT IMPROVEMENT PLAN

Summary of actions

- Develop a drinking water quality management improvement plan.
- Ensure that the plan is communicated and implemented, and that improvements are monitored for effectiveness.

An improvement plan should be developed to address identified needs for full implementation of the drinking water quality management system. The improvement plan should be endorsed by senior executive. Improvement plans may encompass a wide range of issues such as:

- capital works;
- training;
- enhanced operational procedures;
- consultation programs;
- research and development;
- incident protocols;
- communication and reporting.

Improvement plans can include short-term (e.g. one year) or long-term programs. Short-term improvements might include actions such as enhanced mains flushing programs, increased staffing, and the development of community awareness programs. Long-term capital works projects could include covering of water storages or enhanced coagulation and filtration.

Improvement plans should include objectives, actions to be taken, accountability, timelines and reporting. They should be communicated throughout the organisation and to the community, regulators and other agencies.

Implementation of improvement plans will often have significant budgetary implications and therefore may require detailed cost–benefit analysis and careful prioritisation in accord with the outcomes of risk assessment (see Section 3.2.3). Implementation of plans should be monitored to confirm that improvements have been made and are effective.

3.13 References

ARMCANZ, ANZECC (1998) *Implementation Guidelines, Paper 3 National Water Quality Management Strategy,* Agriculture and Resource Management Council of Australia and New Zealand, Australian and New Zealand Environment and Conservation Council, Commonwealth of Australia, Canberra

Baker PD, Steffensen DA, Humpage AR, Nicholson BC, Falconer IR, Lanthois B, FergussonKM, Saint CP (2001). Preliminary evidence of toxicity associated with the benthic cyanobacterium Phormidium in South Australia. *Environmental Toxicology*, 16(6 Special Issue SI):506–11.

Hellard ME, Sinclair MI, Forbes AB, Fairley CK (2001). A randomized, blinded, controlled trial investigating the gastrointestinal health effects of drinking water quality. *Environmental Health Perspectives*, 109(8):773–8.

MacKenzie WR, Hoxie NJ, Proctor ME, Gradus MS, Blair KA, Peterson DE, Kazmierczak JJ, Addiss DG, Fox KR, Rose JB, David JP (1994). A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *The New England Journal of Medicine*, 331(3):161-167.

O'Connor DR (2002a). *Report of the Walkerton Inquiry, Part 1. The events of May 2000 and related issues*. The Attorney General of Ontario, Toronto, The Walkerton Inquiry.

O'Connor DR (2002b). *Report of the Walkerton Inquiry, Part 2. A strategy for safe drinking water.* The Attorney General of Ontario, Toronto, The Walkerton Inquiry.

CHAPTER 4 Framework for the Management of Drinking Water Quality: application to small water supplies

Chapter 4 Framework for the Management of Drinking Water Quality: application to small water supplies

4.1 Introduction

For the purposes of this document, small water supplies are those serving fewer than 1000 people; they include supplies to facilities such as caravan parks, school camps, tourist attractions, roadhouses, and individual household supplies. The sources of these supplies can include groundwater, surface water and rainwater.

For small supplies, it may not be economically feasible or practical to carry out all the recommendations of the ***Australian Drinking Water Guidelines*** (ADWG); however, there is a range of basic measures that can be implemented to provide reasonable assurance of safety. This chapter provides guidance on methods that are suited to small communities and that should give an adequate degree of confidence that safe water is being supplied.

4.2 Applying the Framework

The ADWG provide a Framework for management of drinking water quality based on a preventive, risk management approach; Chapter 2 gives an overview of the Framework, and Chapter 3 details its 12 elements. Those responsible for small water supplies should adhere to this approach as far as possible; however, it may not be practical or necessary to implement all aspects of the Framework. One of the major difficulties for small communities, particularly those in remote areas, is the implementation of regular monitoring programs (both in terms of cost and the practicalities of transporting samples to testing laboratories). The advantage of the Framework is that it places emphasis on a preventive approach to managing water quality, with less reliance on water testing.

The principal risk to human health from drinking water is the presence of pathogenic microorganisms. Thus, to ensure safe water, the focus in small supplies should be on regular inspection of the system to check for any direct or potential sources of contamination, and on the use of a clean and unpolluted water source. The following sections explain how these requirements for small water supplies can be achieved in the context of the Framework.

4.2.1 ASSESSMENT OF THE DRINKING WATER SUPPLY

Analysis of the water supply system, identification of potential hazards and risk assessment (described in detail in Section 3.2) are essential for good management of all supplies.

In the case of small supplies, initial steps would be to develop a simple flow diagram of the main features of the system (water sources, treatment or disinfection, service tanks and major piping) and to determine basic water quality characteristics. If groundwater is the source of supply, then chemical quality should be assessed as a priority. In some parts of Australia, concentrations of naturally occurring elements such as arsenic, fluoride and uranium, or nitrates from agricultural land uses, may exceed safe levels.

The water system should be inspected to identify likely sources of hazards. The greatest sources of microbial hazards are human and livestock wastes, and water systems should be inspected to determine the likelihood that this type of contamination will affect water quality. The discharge of septic waste and access of livestock to watercourses, or the proximity of either to supply bores, are likely sources of contamination.

Potential sources of hazards for water supplies can include:

- septic waste from on-site or communal wastewater systems;
- human wastes from tourists, campers and others having access to water catchments;
- animal faeces or dumped animal carcases;
- effluent from factories, milking sheds and urban stormwater drains (which may contain partially treated sullage and toilet wastes);
- leakage or seepage from rubbish tips and landfill sites;
- agricultural pesticides and fertilisers;
- naturally occurring elements;
- mining industry wastes.

Risk assessment, described in detail in Section 3.2.3, involves estimating the likelihood that a hazard will occur and the consequences if it does. The aim is to distinguish between high and low risks so that attention and resources can be directed towards those hazards that are most threatening. The risks associated with all hazards identified for a small water supply system should be assessed.

4.2.2 PREVENTIVE MEASURES FOR DRINKING WATER QUALITY MANAGEMENT

Where there are hazards that represent high risks, preventive measures (described in detail in Section 3.3) will be required to remove the hazard or to reduce it to an acceptable level. The effectiveness of existing measures should be assessed, but if these are not sufficient, alternative measures will need to be identified. As with all systems, assessment of preventive measures should include consideration of the important principle of the multiple barrier approach. The types of barriers and the preventive measures required will depend on the characteristics of the source water and the associated catchment.

Groundwater

In most cases, contamination of groundwater supplies can be prevented by a combination of simple measures. Groundwater in confined or deep aquifers will generally be free of pathogenic microorganisms and, providing the water is protected during transport from the aquifer to consumers, microbial quality should be assured. The local vicinity of the borehead should be protected from livestock access, and buffer zones should be established between the bore and disposal or discharge of septic wastes. Bores should be encased to a reasonable depth and boreheads should be sealed to prevent ingress of surface water or shallow groundwater.

Once the groundwater is pumped out of the aquifer, protection can be achieved by delivering the water through enclosed water systems. Storage tanks should be roofed, pipelines should be intact and cross-connections should be protected by the installation of backflow prevention devices.

Rainwater

Rainwater systems, particularly those involving storage in above-ground tanks, generally provide a safe supply of water. The principal sources of contamination are birds, small animals and debris collected on roofs. The impact of these sources can be minimised by a few simple measures: guttering should be cleared regularly; overhanging branches should be kept to a minimum, because they can be a source of debris and can increase access to roof catchment areas by birds and small animals; and inlet pipes to tanks should include leaf litter strainers. First-flush diverters, which prevent the initial roof-cleaning wash of water (20–25 L) from entering tanks, are recommended. If first flush diverters are not available, a detachable downpipe can be used to provide the same result.

Further information on rainwater tanks is provided in ***Guidance on the use of rainwater tanks*** (enHealth 2004).

Surface water

Assurance of quality from surface water sources is more difficult than from most groundwater or rainwater systems. In general, surface waters will require at least disinfection, and in some cases filtration, to assure microbial safety. However, as for groundwater systems, the first barrier is to prevent contamination at source by minimising contamination from human waste, livestock and other hazards as discussed above. The greater the degree of protection of the water source, the less the reliance on treatment and disinfection. After treatment or disinfection, water should be protected during delivery to consumers in the same manner as groundwater, by ensuring that distribution systems are enclosed.

4.2.3 IMPLEMENTATION OF OPERATIONAL PROCEDURES AND PROCESS CONTROL

Section 3.4 provides a detailed description of the implementation of operational processes and process control.

Operational procedures

Operational procedures should be developed and clearly documented. The procedures should provide clear protocols for activities and processes such as:

- regular inspections of raw water sources and storages for sources of contamination (animals, birds, drainage inflows);
- checking the integrity of groundwater bores and protection of bores from surface contamination;
- inspection and cleaning of rainwater catchments and tanks;
- inspection and maintenance of all equipment and plant.

Operational monitoring

Operational monitoring includes both regular inspections and testing. In small and remote systems, greater attention should be given to inspections of systems, to check that the preventive measures used to protect water supplies (e.g. denying livestock access, keeping out human waste) are functioning.

The frequency of sanitary inspections of a catchment will depend on the characteristics of each site, the source of raw water, the time the water remains in storage (allowing natural die-off of pathogens to occur), and the subsequent treatment that is provided. As well as regular inspections in the immediate vicinity of the off-take site, every catchment where there is habitation or free public access should be comprehensively inspected at least once a year for potential sources of pollution. Wherever possible, measurements should be undertaken at the site. Test kits are available for a range of parameters, including disinfectant residuals and pH. In some cases, online monitoring might be used; for example, the operation of pumps and disinfection equipment can be monitored using 24-hour telemetry systems that include remote alarms.

Where catchments and supplies are beyond the water supplier's jurisdiction, exchange of information and collaborative assessment of the quality of source waters is advocated.

Corrective action

Where problems occur, corrective action should be taken as quickly as possible. Potential impacts on water quality will need to be assessed and, where necessary, discussed with the local health authority.

If health risks are considered unacceptable, responses could include using an alternative source of water (if available), or issuing advice to the public to either to boil water before consumption (in the case of microbial contamination) or avoid use (in the case of chemical contamination). In the latter case, an alternative water supply will be needed.

Equipment capability and maintenance

The equipment and plant incorporated in the water supply system should be maintained in good condition. In particular, equipment used in water treatment (e.g. for disinfection or microfiltration) should be inspected regularly and should be adequately maintained.

Materials and chemicals

Materials and chemicals used in water systems should be suitable for use with drinking water. Chemicals such as disinfectants and coagulants should be evaluated for suitability. Where expertise is limited, small communities are encouraged to seek advice from larger suppliers, or state/territory or local governments. All materials should comply with Australian Standard AS/NZS 4020 ***Products for use in contact with drinking water***.

4.2.4 VERIFICATION OF DRINKING WATER QUALITY

Verification of drinking water quality is described in detail in Section 3.5. Testing of water in small and remote supplies can present both economic and logistic difficulties, particularly for microbial samples that need to be transported to testing laboratories within 12–24 hours of collection. Application of the Framework decreases reliance on drinking water quality testing; however, testing is still important as a means of verifying that, overall, the barriers and preventive measures implemented to protect public health are working effectively.

Small systems should be monitored on the basis that it is more effective to test for a narrow range of characteristics as frequently as possible than to analyse comprehensively less often.

Microbial quality is the most important factor in determining the ongoing safety of water supplies for human consumption. Therefore, wherever possible, a regular testing program should be instituted for the indicator ***E. coli.*** As stated in Chapters 9 and 10, a minimum of one microbial sample per week is generally recommended; however, in small systems this is not always practical. Where sampling is less frequent than recommended, sanitary inspections should be more frequent, to provide assurance on the integrity and normal operation of the system.

In systems where disinfection is used, evidence of continuous operation is very important in providing assurance of microbial quality. Disinfection is very effective against bacterial pathogens but less so against viruses and enteric protozoa (e.g. ***Giardia*** and ***Cryptosporidium***). The presence of viruses and protozoa can be minimised by protecting water supplies from human and livestock waste.

If chlorination is used, the presence of a free chlorine residual in the distribution system provides evidence of initial disinfection and protection against recontamination from backflow, pipeline breaks or other causes. The amount of chlorine required varies with the flow rate, the quality of the raw water and other factors. Generally, a free chlorine residual of between 0.2 and 0.5 mg/L is adequate.

- At least daily testing of chlorine residuals should be carried out to check the effectiveness of the disinfection system. This can be done using a simple diethyl-phenylenediamine (DPD) colour comparator.

4.3 Individual household supplies

For an individual household supply, the emphasis should be on selecting the best quality source water available, and on protecting its quality by the use of barrier systems and maintenance programs. Whatever the source (ground, surface or rainwater tanks), householders should assure themselves that the water is safe to drink. Generally, surface water or shallow groundwater should not be used as a source of drinking water without treatment. Information on the quality of surface and groundwater may be available from state or local governments, which may monitor the particular source water as part of a water monitoring program. Alternatively, an individual household should consider having the water tested for any key health characteristics identified as being of local concern. Where the raw water quality does not meet the ADWG, a point-of-use device may be useful.

The quality of water from rainwater tanks can be affected by roofing and tank materials, paints, atmospheric contaminants, leaves, dust, and animal and bird droppings. However, providing that the system is reasonably well maintained, rainwater can generally provide a safe supply of drinking water. Further information on rainwater tanks is provided in ***Guidance on the use of rainwater tanks*** (enHealth 2004), and brochures and other material are provided by state and local government authorities.

4.4 Reference

enHealth Council (2004). ***Guidance on use of rainwater tanks***. National Public Health Partnership, Canberra.

PART II DESCRIPTION OF WATER QUALITY

CHAPTER 5 Microbial Quality of Drinking Water

Chapter 5 Microbial Quality of Drinking Water

5.1 Introduction

This chapter discusses the microbial characteristics of water quality. It describes the microorganisms found in drinking water that can be harmful to health and discusses the risk of disease from waterborne pathogens. It also discusses the 'nuisance organisms' that may affect the taste, odour or appearance of water but do not cause disease. Advice on when and how to measure the characteristics and how to interpret the results is provided in Part III.

5.2 Microorganisms in drinking water

The microbial guidelines seek to ensure that drinking water is free of microorganisms that can cause disease. The provision of such a supply is of paramount importance to the health of a community.

The most common and widespread health risk associated with drinking water is contamination, either directly or indirectly, by human or animal excreta and the microorganisms contained in faeces. If the contamination is recent, and those contributing to the contamination include carriers of communicable enteric diseases (diseases of the gut), some of the microorganisms that cause these diseases may be present in the water. Drinking such contaminated water or using it in food preparation may cause new cases of infection. Those at greatest risk of infection are infants and young children, people whose immune system is suppressed, the sick, and the elderly.

Pathogenic (disease-causing) organisms of concern include bacteria, viruses and protozoa; the diseases they cause vary in severity from mild gastroenteritis to severe and sometimes fatal diarrhoea, dysentery, hepatitis, cholera or typhoid fever.

The classic waterborne diseases are caused by organisms originating in the gut of humans or other animals. However, many organisms of environmental origin that are not normally associated with the gastrointestinal system are found in water, and some of these organisms may, under certain circumstances, cause disease in humans. Such organisms include the protozoan ***Naegleria fowleri***, a number of bacteria, including ***Pseudomonas, Klebsiella*** and ***Legionella*** spp, and some species of environmental mycobacteria.

Infection is the main, but not the only, problem associated with microorganisms in drinking water. For instance, certain algae and bacteria can produce toxins that affect humans; the toxins may remain in the water even when the organisms responsible have been removed. Other 'nuisance organisms' can cause problems of taste, odour or colour, or promote deposition and corrosion.

The supply of safe drinking water involves the use of multiple barriers to prevent the entry and transmission of pathogens. The effectiveness of these barriers should be monitored by a program based on operational characteristics and testing for microbial indicators (see Sections 3.4 and 3.5).

5.3 Waterborne pathogens

5.3.1 BACTERIAL PATHOGENS

Excreted pathogens

The human bacterial pathogens that can be transmitted by consuming contaminated drinking water, and that present a serious risk of disease, include *Salmonella* spp, *Shigella* spp, enterovirulent *E. coli*, *Vibrio cholera*, *Yersinia enterocolitica*, *Campylobacter jejuni* and *C. coli*.

After being excreted in faeces from the body of their host, bacterial pathogens gradually lose viability and the ability to cause infection. The rate of decay varies with different bacteria; it is usually exponential, and after a certain period a pathogen will become undetectable. The most common waterborne pathogens are those that are highly infectious or highly resistant to decay outside the body. Pathogens with a low persistence (i.e. those that do not survive long outside the host) must rapidly find a new host and are more likely to be spread by person-to-person contact or by poor personal or food hygiene than by drinking water.

If drinking water is faecally contaminated, bacterial pathogens are likely to be widely and rapidly dispersed. Outbreaks of waterborne disease are therefore frequently characterised by infection across a whole community.

Pathogens growing in water supplies

Various bacteria that occur naturally in the environment may cause disease opportunistically in humans. Those most at risk are people with impaired local or general defence mechanisms, such as the elderly, the very young, people with burns, people who have undergone recent surgery or who have suffered serious injury, and people with severely compromised immune systems. In such individuals, if water used for drinking or bathing contains large numbers of opportunistic pathogens, it can occasionally produce infections of the skin, and of the mucous membranes of the eye, ear, nose and throat. Examples of such opportunistic agents are Pseudomonas aeruginosa, species of Klebsiella and Aeromonas, and certain slow-growing mycobacteria.

Legionellosis, commonly caused by the free-living bacterium Legionella pneumophila, is a serious illness resulting from inhalation of water in which the causative organisms have been able to multiply because of warm conditions and the presence of nutrients.

Part V contains fact sheets on the bacterial pathogens that may contaminate the water supply.

5.3.2 PROTOZOA

The great majority of protozoa in freshwater are natural aquatic organisms of no significance to health.

They generally feed on other microorganisms such as bacteria, cyanobacteria or algae. The greatest diversity of protozoa is found in open surface waters, including water supply sources, but some species can colonise piped water supplies; the extent to which this occurs depends on bacterial activity in these supplies.

The protozoa that may occur in drinking water and cause adverse health effects fall into two functional groups:

- enteric protozoa that occur widely as parasites in the gut of humans and other mammals;
- free-living organisms that are opportunistic pathogens in humans and are responsible for serious cerebral and eye diseases (there are very few such organisms).

Since pathogenic protozoa are of both enteric and environmental origin, and since different species vary in their responses to water treatment, control strategies need to be specifically tailored to the biology of individual species.

Enteric protozoa

Enteric protozoa, like enteric bacteria and viruses, may be found in water following direct or indirect contamination with human or animal faeces. Transmission by drinking water is one of several mechanisms for completing the faecal–oral cycle for these organisms. Enteric protozoa occur in water as dormant infectious cysts; the cysts have natural mortality rates that are probably determined by temperature and incident ultraviolet light.

In principle, removal or disinfection at the water source should be sufficient to prevent contamination of drinking water by enteric protozoa, provided adequate measures are in place to prevent later recontamination. In practice, this may be difficult because protozoan cysts are generally more resistant to water disinfectants than most bacteria and viruses.

Cryptosporidium and *Giardia* species are likely to be the most important enteric protozoa in water in Australia, although infection by *Entamoeba histolytica* is also endemic in some communities. All these organisms cause moderate to severe enteritis in susceptible people; in Australia, they seem to be transmitted mostly by direct contact with a carrier. Outbreaks of *Cryptosporidium* in drinking water supplies are associated with contamination from by human or livestock (particularly cattle), and faulty or inadequate treatment. There is evidence that Giardia infections in Australia may result from contact with septic-tank waste or from recent faecal contamination of drinking water.

Free-living protozoa

Two groups of free-living amoebae, *Naegleria* and *Acanthamoeba*, have been responsible for human infections in Australia. Infection is opportunistic, and generally results from contact during recreational bathing, or domestic uses of water other than drinking. Public water supplies can contaminate swimming pools. The occurrence of these organisms is unrelated to faecal contamination, and their ecology in aquatic environments is more complex than that of enteric protozoa.

Cerebral infection by *Naegleria fowleri* is strictly waterborne and, although rare, is usually fatal. Since these amoebae are able to colonise piped water supplies, disinfection at the water source may not adequately control them unless the disinfectant pervades the whole distribution system.

Acanthamoeba species cause both cerebral and corneal disease. An environmental source of infection has rarely been identified with certainty. Since *Acanthamoeba* species are among the most common protozoa in soil, as well as occurring in freshwater and seawater, the source of infection may often be soil or airborne dust.

Both *Acanthamoeba* and *Naegleria* species are known to support symbiotic growth of *Legionella* species within the cell, and the presence of these amoebae in cooling-tower water can indicate conditions that favour Legionella.

Part V contains fact sheets on the protozoan pathogens that may contaminate the water supply.

5.3.3 VIRUSES

The viruses of most significance for drinking water are those that multiply in the human intestine and are excreted in large numbers in the faeces of infected individuals. Although they cannot multiply outside the tissues of infected hosts, some enteric viruses can survive in the environment and remain infective for long periods. Human enteric viruses occur in water largely as a result of contamination with sewage and human excreta. The numbers of viruses present and their species distribution will reflect the extent to which they are being carried by the population; however, the use of different analytical methods

can also lead to wide variations in calculations of the numbers of viruses found in sewage. Sewage treatment may reduce numbers by a factor of 10 to 10,000, depending upon the nature and degree of treatment; however, even tertiary treatment of sewage will not eliminate all viruses. As sewage mixes with receiving water, viruses are carried downstream; the length of time they remain detectable depends on temperature, their degree of adsorption to particulate matter, penetration of sunlight into the water and other factors. Consequently, enteric viruses can be found at the intakes to water treatment plants if the water is polluted by sewage. However, proper treatment and disinfection should produce drinking water that is essentially virus-free.

Recent methodological advances have revolutionised the diagnosis of viral diarrhoeal diseases, and waterborne outbreaks due to viruses have now been identified in both developed and developing countries all over the world, with many different strains of viruses isolated from raw and treated drinking water. Isolation of viruses from water indicates that a hazard exists, but it does not prove beyond doubt that water is a vehicle for transmission of disease.

Epidemiological proof of waterborne transmission of viral diseases is very difficult to establish, for a variety of reasons. Symptoms may not resemble those of typical waterborne diseases, and many of those infected will show no symptoms. Some infections, for example the hepatitis A virus, are difficult to trace to a source because of long incubation periods. Water is often only one of various routes of transmission, it is not always the major route, and adequately sensitive methods for detecting the infectious agent in water are often not available.

Part V contains fact sheets on the viral pathogens that may contaminate the water supply.

5.3.4 HELMINTHS

The major helminth (worm) parasites of humans listed by the World Health Organization as being transmitted by water do not occur in Australia, apart from their rare incidence in recent immigrants or Australians returning from areas where the organisms are endemic. The eggs of enteric nematodes such as Trichurus may enter water, but waterborne transmission is generally regarded as unimportant. Nematodes seen as adult worms or larvae in microscopic examination of material from water supplies are likely to belong to free-living groups such as Turbatrix or Rhabditis, which, like free-living protozoa, colonise systems that support other microorganisms.

Infective enteric helminths should not be present in drinking water; however, it is impracticable to set guidelines due to the low prevalence of these agents in Australia.

5.3.5 CYANOBACTERIA

Cyanobacteria are true bacteria, although they are often called 'blue-green algae' because they resemble green algae in morphology, habitat and photosynthetic ability. They occur as single cells, filaments or colonies, and their buoyancy enables them to migrate towards the surface of water in response to light. Cyanobacteria inhabit all natural waters, and become a problem only when present in excessive numbers (blooms). This is more likely to occur when temperatures are high, with long sunny days, high levels of plant nutrients in the water, low stream flows, and calm conditions that permit the cells to migrate to the surface. These conditions occur sporadically in late spring through to autumn in many parts of Australia. In addition, eutrophication (nutrient enrichment) associated with increased agriculture and urbanisation has increased the occurrence of cyanobacterial blooms.

They are of concern in drinking water primarily because of the intracellular toxins they produce, which are of three main types:

- hepatotoxins, which damage liver cells;
- neurotoxins, which damage nerve cells;
- cylindrospermopsin, which can damage the liver, kidney, gastrointestinal tract and blood vessels.

No human deaths have been recorded from ingesting the toxins of cyanobacteria but gastroenteritis may result from drinking water containing toxic species and extended exposure may lead to more serious impacts. Deaths have been attributed to the presence of microcystin in water used for renal dialysis in Caruara, Brazil (Jochimsen *et al.* 1998).

Direct contact with toxic or non-toxic species of cyanobacteria may cause skin rashes or eye irritation due to adverse reactions to components in the cell walls of the organisms. This could occur through showering or bathing in water containing blooms or scums.

Part V contains fact sheets on toxic cyanobacteria.

5.4 Risk of disease from waterborne pathogens

Drinking water is only one of several means by which many infectious agents can be transmitted. It can, however, be of considerable importance, and many pathogens that are excreted in faeces have caused epidemics through contaminated water. The significance of a particular organism in water can vary considerably; for example, a potentially pathogenic organism will not always cause symptomatic disease in a particular individual. The chances of waterborne infections occurring in a community depend on:

- the concentration of pathogenic organisms in the water;
- the virulence of the strain;
- the per capita intake of contaminated water;
- the infectious dose of the particular pathogen;
- the susceptibility of individuals;
- the incidence of the infection in the community (which will determine the numbers of pathogens being excreted).

The occurrence of disease is also related to the relative level of immunity in the community. If, for example, the water supply has been repeatedly contaminated, the community may have become immune to some waterborne pathogens. Such a situation can be seen in some developing countries where the prevalence of pathogens is high and the standard of tap water is less than optimal. Visitors who drink the water frequently become ill, while the local community, especially adults, appear to suffer minimal morbidity. The immunity of the local population may, however, be acquired at the expense of the health of more susceptible individuals in that community, including children, the aged and people already in poor health.

Thus, a community consuming water with indicators of faecal pollution may show no discernible disease. Such a situation, however, is unstable. Apart from the risk to visitors, faecal pathogens affecting the locals may be introduced from, for instance, an immigrant or a seasonal outbreak of a disease such as cryptosporidiosis resulting from cattle in the catchment. When illness occurs in a community, the route of infection needs to be confirmed by epidemiological investigation, even when the disease-causing organism is found in a suspect water supply.

5.5 Nuisance organisms

Nuisance organisms comprise a morphologically and physiologically diverse collection of organisms.

They include:

- prokaryotic bacteria such as planktonic and benthic cyanobacteria (blue-green algae);
- iron, manganese and sulfur bacteria;
- actinomycetes and fungi;
- eukaryotic organisms such as algae, crustacea and protozoa.

Problems occur when the conditions in source waters, reservoirs or distribution systems support the growth of a particular nuisance organism or group of nuisance organisms. Excessive quantities of organic matter, for instance, will support the growth of bacteria and fungi, and these will maintain populations of protozoa and crustacea. Many invertebrate animals can feed on bacteria, fungi and protozoa.

In addition, a particular nuisance organism may show morphological characteristics or produce some extracellular product that gives the organism a competitive advantage over other aquatic inhabitants. This may include a 'holdfast' (i.e. a mechanism for anchoring the organism) or sheath (in the case of some iron bacteria) or the ability to produce antibiotic substances (as in some fungal species).

Raw water does not usually contain sufficient numbers of nuisance organisms to create problems; however, the water treatment process may assist their growth. Nuisance organisms concentrate on the surface of filters and inside the filter bed, and on mains and water reservoir surfaces, where they lyse and release cellular compounds responsible for colour, turbidity, taste and odour. Activated carbon filters will, after a period, contain high amounts of organic matter; this may affect taste and odour, and increase turbidity, providing an excellent substrate for bacteria. Poorly operated filter systems, including activated carbon-based domestic filter systems, can be the source of tastes and odours.

It is not practicable to specify a quantitative limit for nuisance microorganisms.

5.5.1 ORGANISMS CAUSING TASTE AND ODOUR PROBLEMS

Objectionable tastes and odours can result from compounds produced by certain types of algae, cyanobacteria (blue-green algae), bacteria and sometimes protozoa. Actinomycetes and cyanobacteria, for instance, produce geosmin and methylisoborneol (MIB), which have an earthy taint, and a taste and odour threshold of approximately 0.00001 mg/L (10 ng/L).

Several groups of protozoa produce odorous compounds in culture. Certain species of the amoeba genera *Vannella*, *Saccamoeba* and *Ripidomyxa* that carry rather dense bacterial symbionts also produce either geosmin or MIB. Most previously described sources of these compounds have been cyanobacteria or actinomycetes, so it seems likely that the symbionts are the immediate source. While the mechanism of symbiont contribution to odours in waters is unknown, they should be considered as the likely source of a problem if no other biological source of these strongly smelling compounds can be identified.

Free-swimming ciliates, such as *Climacostomum* and certain *Stentor* species that bear the algal symbionts zoochlorelle, can contribute to odours in water if they reach high densities, although such incidents are not often reported.

Consumers often detect taste and odour problems before analytical methods have detected the compounds responsible. It is therefore advisable to use trained panels to detect taste and odour, and undertake remedial measures before a problem becomes significant. Section 3.5.2 and the fact sheet on Taste and Odour in Part V discuss such panels.

Another method to pre-empt taste and odour problems is to use microscopy to examine regularly the type and number of organisms present in the water. When a group of organisms known to cause taste or odour problems is dominant, measures should be taken to overcome the problem.

5.5.2 ORGANISMS CAUSING COLOUR PROBLEMS

Excessive growths of some algae, cyanobacteria and other bacteria can produce undesirable 'blooms' in source waters, and this may affect colour in the distribution system.

Blooms of algae and cyanobacteria may be controlled by judicious application of copper sulfate or other algaecide to the source water, provided that the cyanobacterial genus is not toxic.

When pigmented organisms such as cyanobacteria and algae are crushed on filters, colour problems can result. This type of problem can be exacerbated by the passage of microalgae through the filters causing an increase in turbidity.

5.5.3 DEPOSITS DUE TO IRON AND MANGANESE BACTERIA

Nuisance iron-oxidising organisms may cause problems in groundwater sources by encrusting bore screens, causing loss of yield and impairing the aesthetic quality of the supply. The presence of these organisms may also indicate organic pollution of the aquifer.

Manganese-oxidising organisms (bacteria, fungi and, very rarely, protozoa) may be responsible for deposits in aquifers, wells and water conduits. The deposits can reduce yield, clog slots in the bore pipes, slow the flow in pipes by increasing turbulence, damage equipment for measuring water flows, and produce black water that stains laundry and disrupts food-handling establishments. Bacteria can attach to the deposits; if disturbed, these will increase the heterotrophic colony count of the water. These problems will generally not occur if the concentration of manganese is below 0.1 mg/L. (See fact sheet on Manganese).

In water containing ferrous or manganous salts, iron or manganese bacteria can oxidise these compounds to form rust-coloured or black deposits in tanks and on the walls of pipes in slow-flowing parts of the distribution system. Changes in water flow can then release the deposits into the supply system, staining laundry and plumbing fittings, and adversely affecting the appearance of drinking water. The slurry may also contain organic deposits that can break down to cause odour problems. (See fact sheets on Colour, Iron, and Manganese.)

Although these nuisance organisms can impair water quality, it is not practicable to monitor for them routinely because of their diverse nature and unpredictable occurrence. Consumer complaints, together with local knowledge of the water supply system and water sources, should be a trigger for action.

5.5.4 CORROSION PROBLEMS DUE TO IRON AND SULFUR BACTERIA

Iron and sulfur bacteria contribute to the corrosion of iron and steel well pipes and drinking-water mains, with corrosion starting from either inside or outside. Microorganisms may cause corrosion by:

- depleting dissolved oxygen;
- preventing corrosive metabolites;
- producing sulfuric acid from sulfides or elemental sulfur;
- participating in the cathodic process.

The presence of these organisms in water may indicate a potential for corrosion of cast iron mains and storage tanks. It can also indicate biodeterioration of certain construction materials, including non-metallic materials such as plastics, rubber jointing compounds and pipe lining materials, which provide organic nutrients and thus encourage the growth of microorganisms such as *Pseudomonas aeruginosa*.

5.5.5 PROBLEMS CAUSED BY LARGE NUMBERS OF MICROORGANISMS

Large numbers of aerobic heterotrophic bacteria in treated water can interfere with the interpretation of tests for the coliform group by masking their presence, thus yielding false-negative results. Strains of Aeromonas species that produce acid and gas with coliform media, even at 44°C, present a particular problem.

Most of these organisms can be controlled relatively easily by water treatment processes, including disinfection. Nutrient-rich raw water should be avoided if water treatment cannot be applied.

5.5.6 NUISANCE INVERTEBRATES

Invertebrate animals often infest shallow open wells, warm shallow storage tanks and small supplies, but problems are uncommon in large public supplies. These invertebrates derive their food from bacteria, algae and protozoa that are present in the water or on slimes. They include freshwater sponges of the phylum Porifera (*Spongilla* spp and *Ephydatia* spp), a coelenterate (*Cordylophora* spp), bryozoans (*Plumatella* spp and *Fredericella* spp) and molluscan bivalves and snails (e.g. *Corbiculina* spp).

For control purposes, the types of animal can be divided into two groups:

- free-swimming organisms, such as the crustacea *Gammarus pulex* (freshwater shrimp), *Crangonyx pseudogracilis*, *Cyclops* species and *Chydorus sphaericus*;
- animals that either move along surfaces or are anchored to them, such as *Asellus aquaticus* (water louse), snails, *Dreissena polymorpha* (the zebra mussel) and other bivalve molluscs, the bryozoan *Plumatella* species, or animals that inhabit slimes, such as *Nais* species, nematodes, and larvae of chironomids.

In warm weather, slow sand filters can sometimes discharge larvae of midges and mosquitoes into the water. This occurs if the top layer of the bed collapses, causing unfiltered water to be drawn down.

Nuisance invertebrates are more likely to penetrate water filtration plant and mains when low-quality raw waters and high-rate filtration processes are used. Prechlorination destroys the invertebrates and thereby assists their removal by filtration; however, the use of high concentrations of chlorine may produce high levels of chlorination by-products. Infestation can usually be prevented by maintaining chlorine residuals in the distribution system, producing high-quality water, and cleaning water mains regularly by flushing or swabbing.

5.6 References

Jochimsen EM, Carmichael WW, An JS, Cardo DM, Cookson ST, Holmes CE, Antunes MB, Filho DA, Lyra TM, Barreto VS (1998). Liver failure and death after exposure to microcystins at a hemodialysis center in Brazil. New England Journal of Medicine, 338:873-878.

CHAPTER 6 Physical and Chemical Quality of Drinking Water

Chapter 6 Physical and Chemical Quality of Drinking Water

6.1 Introduction

This chapter discusses both the physical characteristics of water quality and the chemical characteristics, including organic and inorganic chemicals and pesticides. It explains the rationale for deriving guideline values. The principles used in both cases are very similar and a number of common assumptions have been made.

6.2 Physical quality of drinking water

6.2.1 AN OVERVIEW OF PHYSICAL CHARACTERISTICS

The appearance, taste, odour, and 'feel' of water determine what people experience when they drink or use water and how they rate its quality; other physical characteristics can suggest whether corrosion and encrustation are likely to be significant problems in pipes or fittings. The measurable characteristics that determine these largely subjective qualities are:

- true colour (the colour that remains after any suspended particles have been removed);
- turbidity (the cloudiness caused by fine suspended matter in the water);
- hardness (the reduced ability to get a lather using soap);
- total dissolved solids (TDS);
- pH;
- temperature;
- taste and odour;
- dissolved oxygen.

Colour and turbidity influence the appearance of water. Taste can be influenced by temperature, TDS, and pH. The 'feel' of water can be affected by pH, temperature, and hardness. Rates of corrosion and encrustation (scale build-up) of pipes and fittings are affected by pH, temperature, hardness, TDS and dissolved oxygen.

Each of the physical characteristics is discussed separately in the fact sheets in Part V. However, there is some overlap with organic compounds, microorganisms and, most notably, the inorganic constituents of water; when this occurs, it is noted and cross-referenced.

6.2.2 APPROACH USED IN DERIVATION OF GUIDELINES VALUES FOR PHYSICAL CHARACTERISTICS

In general, the physical characteristics of water are not of direct public health concern, but they do affect the aesthetic quality of the water, which largely determines whether or not people are prepared to drink it. If water is unpalatable or appears to be of poor quality, even though it may be quite safe to drink, the consumer may seek other water sources, and these may not be as safe.

Each guideline value is set at a level that ensures good quality water – that is, water that is aesthetically pleasing and safe, and that can be used without detriment to fixtures and fittings. The values are determined by considering water quality guidelines used by other countries and international bodies, assessing any health implications, and then deciding on a point beyond which the quality of the water might no longer be regarded as good. Factors taken into account include:

- taste and odour thresholds (i.e. the smallest concentration or amount that would be just detected by a trained group of people);

- the concentration or amount that would produce noticeable stains on laundry or corrosion and encrustation of pipes or fittings;
- the concentration or amount that would be just noticeable in a glass of water and lead to a perception that the water was not of good quality.

The physical guideline values are not absolute; they are value judgments determined from an often wide range of values that may be broadly classed as acceptable – that is, there is no one right answer. Consequently, small, short-term excursions beyond a physical guideline value do not necessarily mean that the water will be unacceptable. What is aesthetically acceptable or unacceptable depends on public expectations, and must ultimately be determined by water authorities in consultation with consumers, taking into account the costs and benefits of further treatment. The *Australian Drinking Water Guidelines* (ADWG) provide a starting point for this process.

6.3 Chemical quality of drinking water

A number of chemicals, both organic and inorganic, including some pesticides, are of concern in drinking water from the health perspective because they are toxic to humans or are suspected of causing cancer. Some can also affect the aesthetic quality of water.

The presence of chemical in drinking water may result from:

- natural leaching from soils, rocks and mineral deposits into source waters;
- land-use activities in catchments leading to exacerbation of natural processes such as mobilisation of salts;
- run-off from agricultural operations within drinking water catchments;
- biological processes including growth of cyanobacteria and algae in waterways and reservoirs;
- contamination of source water by treated effluent discharge and other point sources within the catchment;
- carry-over of small amounts of treatment chemicals;
- addition of chemicals such as chlorine and fluoride;
- corrosion and leaching of pipes and fittings.

6.3.1 INORGANIC CHEMICALS

Inorganic chemicals in drinking water usually occur as dissolved salts, principally carbonates, chlorides and sulfates, attached to suspended material such as colloids and clay particles, or as complexes with naturally occurring organic compounds.

Unless otherwise stated, the guideline value refers to the total amount of the substance present, regardless of its form (e.g. in solution or attached to suspended matter).

6.3.2 ORGANIC COMPOUNDS (*REVISED 2011*)

Organic compounds are usually present in drinking water in very low concentrations. They may occur either naturally or as a result of human activities. By-products of disinfection are the most commonly found organic contaminants in Australian drinking water supplies. Pesticides and petroleum products are occasionally detected in source water or treated drinking water in Australia, but rarely at concentrations above health-based guideline values.

Disinfection by-products

The by-products of disinfection are the products of reactions between disinfectants, particularly chlorine, and naturally occurring organic material such as humic and fulvic acids, which result from the decay of vegetable and animal matter. Of these disinfection by-products, the trihalomethanes (THMs) are produced in the highest concentrations.

Most disinfectants used to render drinking water safe from pathogenic microorganisms will produce by-products in the disinfection process. Factors affecting the formation of disinfection by-products include:

- the amount of natural organic matter present;
- the disinfectant used;
- the disinfectant dose;
- pH;
- temperature;
- the time available for reaction (C.*t* or contact time).

Chlorine is the most common disinfectant; in the chlorination process it reacts with naturally occurring organic matter to produce a complex mixture of by-products, including a wide variety of halogenated compounds (i.e. organic by-products of chlorination). The main by-products are the THMs and chlorinated acetic acids. Many other by-products can be produced, but concentrations are generally very low (usually <0.01 mg/L and often <0.001 mg/L).

Other disinfectants can produce different types of by-products: for example, ozone is known to produce formaldehyde and other aldehydes.

Known disinfection by-products are considered individually in the fact sheets in Part V. It is possible, however, that other disinfection by-products for which no health data are available are present at extremely low concentrations. It is also possible that when these compounds (both known and unknown) are ingested together, their combined effects on health may be different from their individual effects. Epidemiological studies examine disinfection by-products as a generic group, and can be useful in determining overall effects.

A number of epidemiological studies have suggested an association between water chlorination by-products and various cancers (Michaud *et al.* 2007, Villanueva *et al.* 2007). This association has been most consistent in relation to cancer of the bladder and rectum, but there are insufficient data to determine concentrations at which chlorination by-products might cause an increased risk to human health.

In experiments with laboratory mice, when concentrates derived from chlorinated drinking water were applied to the skin, there was no increase in the incidence of skin tumours compared with concentrates derived from unchlorinated supplies. Similarly, oral administration of chlorinated humic acids in drinking water did not increase the incidence of tumours compared with animals receiving unchlorinated humic acids, or with saline-treated controls (IARC 1991).

Studies have shown that concentrates of some chlorinated drinking water supplies are mutagenic to some strains of test bacteria. These effects were consistently found with samples of surface water that had a high content of natural organic compounds at the time of chlorination. A significant proportion of the increased mutagenicity has been attributed to a chlorinated furanone known as MX (Kronberg and Vartiainen 1988).

The International Agency for Research on Cancer has reviewed the available data and concluded that there is inadequate evidence to determine the carcinogenicity of chlorinated drinking water to humans (IARC 1991).

Action to reduce the concentration of disinfection by-products is encouraged, but disinfection itself must not be compromised: the risk posed by disinfection by-products is considerably smaller than the risk posed by the presence of pathogenic microorganisms in water that has not been disinfected.

Further information on disinfection of drinking water is contained in the information sheets (Part IV) and fact sheets (Part V).

Pesticides

For the purpose of the ADWG the term 'pesticides' includes agricultural chemicals such as insecticides, herbicides, nematicides, rodenticides and miticides.

The Australian Pesticides and Veterinary Medicines Authority (APVMA) is responsible for assessing all pesticides prior to registration to allow sale and use in Australia. For registration, data required on the pesticide include information on the proposed use, the toxicity and the residues that might result from proper use. When the pesticide is registered, a safe level of exposure, conditions of use and maximum levels of residues for water are determined. This mechanism allows the formulation of appropriate guideline values for pesticides in drinking water and a process for their revision, which includes public consultation.

The use of pesticides in Australia is regulated by the states and territories, though this is the subject of a COAG reform and may change in the future. The APVMA provides label requirements for the approved use and application of pesticides and these labels are required to be followed by all users of registered pesticides, with enforcement the responsibility of the states and territories. These label requirements are intended, in part, to minimise pesticide contamination of waterways. Consistent with this, pesticides should not be found in water supplies above safe levels and if they are, investigations must be undertaken to determine how they came to be there. These investigations should then be followed by corrective action aimed at the prevention of pesticide contamination of drinking water supplies.

Within the context of aiming to minimize pesticide contamination of drinking water, it should be noted that a small number of pesticides have been approved by the APVMA for the management and control of pests including insects and insect larvae in drinking water supplies. An example is s-methoprene, which has been approved for use as a larvacide in rainwater tanks. In circumstances where pesticides are intentionally applied to drinking water supplies, drinking water concentrations should be monitored to ensure that concentrations are within safe levels.

Contamination of drinking water by pesticides may occur occasionally as a result of accidental spills, misadventure, or emergency use of pesticides. In such cases, prompt action may be required by public health officials.

The health-based guideline values are derived from the acceptable daily intake (ADI) and are set at about 10 per cent of the ADI for an adult weight of 70 kg and a daily water consumption of 2 litres. The health values are very conservative, and include a range of safety factors, which always err on the side of safety.

In earlier versions of the *Australian Drinking Water Guidelines*, the guideline value for many pesticides was set at the practical analytical detection limit for the particular chemical substance. This approach was used to reflect the philosophy that good water quality management should aim to prevent the contamination of drinking water supplies by pesticides (regardless of potentially negligible health implications). While this management philosophy still applies, the approach to setting guidelines has been revised and analytical detection limits are no longer used as guideline values for pesticides.

The revised approach has been adopted for two main reasons. The first is that analytical detection limits are constantly changing (decreasing) as a result of on-going technological advancements. This means that in order to keep up-to-date, a detection limit-based guideline would also need to be continuously revised downward, which is an impractical situation from a human health perspective. The second reason is the desirability of a scientifically-consistent approach to guideline setting across all chemicals.

Wherever possible, guideline values for all other chemicals are based on human health considerations and toxicological data. Accordingly, it is appropriate that the guideline values for pesticides be addressed in the same way.

As noted above, this change in guideline setting for pesticides does not change the general philosophy regarding the management of pesticides in drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

Pharmaceuticals and endocrine-disrupting chemicals

Pharmaceuticals

Pharmaceuticals comprise a large class of predominantly organic compounds. They are administered to humans and animals to achieve a variety of benefits including prevention and treatment of disease. The large variety of compounds in use and their importance to physiology, along with their widespread use and chemical characteristics contributing to persistence suggest the potential for their similarly widespread distribution in the environment and the potential for contamination of potable water supplies.

Virtually all pharmaceuticals administered to humans are excreted in varying degrees and discharged directly into the sewerage system. These compounds are then affected by treatment processes in municipal sewage treatment plants, before discharge to the environment. Depending on chemical properties including aqueous solubility, volatility, lipophilicity and susceptibility to biodegradation, pharmaceutical residues may be removed in varying degrees during conventional sewage treatment processes prior to environmental discharge.

Synthetic pharmaceutical compounds were first observed and reported in sewage during the 1970s. Since then, over 100 pharmaceutical drugs and metabolites have been identified in environmental samples, primarily in Europe and North America. Reported compounds include analgesic, anti-inflammatory, beta-blocker, lipid regulator, antiepileptic, β2-sympathomimetic, antineoplastic, antibiotic and contraceptive drugs.

No definitive link has been reported or established between pharmaceutical exposure in drinking waters and human health risk. Furthermore, current evidence does not support a general requirement for additional or specialised drinking water treatment to reduce concentrations of pharmaceuticals. Routine monitoring is not recommended, but targeted, well designed and quality controlled investigative studies could provide more information on potential human exposure from drinking water. Nonetheless, concern for the potential implications of exposure to mixtures of these biologically active chemicals exists and worldwide investigations are ongoing.

Specific concerns have been raised by some scientists that the presence of antibiotic agents in water supplies may facilitate the development of resistant organisms, with implications for public health (Kummerer 2009). While this may be a valid hypothesis, studies are yet to demonstrate that the presence of antibiotics in water supplies has any impact on the development of resistance.

It is not common international practice to regulate or provide guidelines for pharmaceuticals in drinking water. However, the *Australian National Guidelines for Water Recycling (Phase 2)* have taken a pro-active approach and do provide guideline concentrations (and an approach for further developing guidelines) that are applicable to potable water supplies intentionally augmented by recycled municipal effluents (NRMMC, EPHC and NHMRC 2008). Use of these guideline values should be considered for supplies where the risk assessment identifies significant contribution of municipal effluent, whether it is intentional or unintentional.

Endocrine-disrupting chemicals

During the last few decades, reports of hormonally related abnormalities in a wide range of species have accumulated. Chemical contaminants are believed to be responsible for many of these abnormalities, acting via mechanisms leading to alteration in endocrine function. This phenomenon, known generally as 'endocrine disruption', has been identified by the World Health Organization as an issue of global concern (Damstra *et al.* 2002). The chemicals implicated have been collectively termed 'endocrine-disrupting chemicals' (EDCs), or simply 'endocrine disruptors' (Damstra *et al.* 2002).

A particularly well documented form of endocrine disruption has been the induction of biochemical hormonal responses in freshwater fish, which can cause significant behavioural and morphological dysfunctions and lead in the worse cases to sterility (Tyler and Jobling 2008). A growing number of natural and synthetic environmental chemicals have been implicated as causative agents of these observed disruptions. However, in terms of potency, the most significant have been natural and synthetic steroidal hormones. Some steroidal hormones have been observed to cause disruption of the endocrine system of fish at ambient concentrations less than 0.000001 mg/L (1 ng/L).

Environmental exposure to oestrogenic hormones has been shown to cause feminisation of male fish (Tyler and Jobling 2008, Rempel *et al.* 2006. More recently, exposure to androgens has been implicated in the masculinisation of fish (Jensen 2006). Furthermore, scientists suspect that anthropogenic estrogens, androgens and progestins may act as reproductive pheromones in fish, thus adversely affecting reproduction (Kolodziej *et al.* 2004).

Much attention has focused on the discharge of hormonal steroids from municipal sewage treatment plants. Municipal sewage effluents have been generally characterised as being 'oestrogenic' in nature, due largely to trace concentrations of oestrogenic steroidal hormones as well as some other natural and synthetic chemicals.

While some EDCs have been detected in some drinking water supplies, concentrations have been generally insignificant compared to other dietary sources of estrogenic activity.

Box 6.1 *The Black Mountain Declaration (2007) on Endocrine Disrupting Chemicals in Australian Waters*

Humans, as mammals, have very similar endocrine systems to other species for which impacts of environmental EDCs have been observed. There is clear evidence that humans have been severely impacted by some EDCs when exposed to significant doses in the form of medications or extreme occupational exposure. However, exposure to EDCs via water (either through recreation or consumption) is considered relatively insignificant compared to other sources such as occupational or dietary exposure.

Despite the valid reasons for concern, evidence of impacts to humans from environmental exposure to EDCs is yet to be established. This includes a lack of evidence of impacts via exposure from water supplies, food products and air. Given the observed susceptibility of other species and the ultimate importance of protecting public health, a precautionary approach towards minimising unnecessary exposure to EDCs in water, food and air is warranted.

It is not common international practice to regulate or provide guidelines for EDCs in drinking water. However, the ***Australian National Guidelines for Water Recycling (Phase 2)*** have taken a pro-active approach and do provide guideline concentrations (and an approach for further developing guidelines) that are applicable to potable water supplies intentionally augmented by recycled municipal effluents (NRMMC, EPHC and NHMRC 2008). Use of these guideline values should be considered for supplies where the risk assessment identifies significant contribution of municipal effluent, whether it is intentional or unintentional.

Other organic compounds

Naturally occurring organic compounds are not generally of human health concern, except for certain specific toxins (see fact sheets on Toxic Cyanobacteria). Other than disinfection by-products, organic contaminants resulting from human activity are not normally detected in Australian drinking water.

They have, however, been detected at times in supplies in North America and Europe, usually following an accidental spill or discharge into a water source or, on rare occasions, from rain contaminated by airborne pollutants. Fact sheets and guideline values are provided in case similar incidents should occur in Australia.

6.3.3 APPROACH USED IN DERIVATION OF GUIDELINE VALUES FOR CHEMICALS

The guideline value for each organic and inorganic chemical is the concentration that, based on present knowledge, does not result in any significant risk to the health of the consumer over a lifetime of consumption and is consistent with water of good quality.

The health-related guideline values are very conservative, and are calculated using a range of safety factors. They always err on the side of safety, particularly where scientific data are inconclusive or where the only data available are from animal studies.

Where aesthetic considerations, including taste and odour, corrosion, and stains on sanitary ware and laundry, dictate a more stringent guideline than that required to protect health, both values are quoted. Health considerations may be of less concern in such cases (although they must still be considered), because water that is aesthetically unacceptable is less likely to be consumed.

For most chemicals, it has not been possible to estimate the higher concentrations that would affect health over shorter periods, so short-term guideline values have generally not been set. However, given the very conservative nature of the guidelines, deviations from the guideline values over a short period do not necessarily mean that the water is unsuitable for consumption. The amount by which and the period for which any guideline value could be exceeded without causing concern will depend on the chemical involved and other factors, such as the risks and benefits to public health.

Each excursion beyond a guideline value should, however, be a trigger for further action.

Chemicals fall into two categories based on health effects:

- those where the effects are observed only above a certain threshold dose, with no effects observed at doses below this threshold;
- those that do not appear to have a threshold.

Sources of data used

Human data

There is little information on the effects of human exposure to organic and inorganic compounds, including pesticides, at the concentrations likely to occur in water. Occasionally, there are useful epidemiological data, and where available, these have been the primary consideration in setting the guideline value.

Animal data

In the absence of human data, experiments on laboratory animals provide toxicological data on the effects of exposure to chemical agents. Ideally, these are long-term studies involving ingestion of the compound dissolved in water or present in food, rather than inhalation or dermal exposure studies. For expediency, such studies are conducted at concentrations that are relatively high in comparison to the concentrations likely to be found in drinking water. Furthermore, the most sensitive animal species, and the most sensitive group within that species, are used in order to increase the likelihood of observing a toxicological effect.

Effects of exposure to chemicals in experimental animals are generally classified in the following broad categories:

- organ-specific;
- neurological/behavioural;
- reproductive/developmental;
- carcinogenic/mutagenic.

Effects may be prolonged or short term, reversible or irreversible, immediate or delayed, single or multiple. The nature, number, severity, incidence and prevalence of specific effects generally increase with increasing dose. Adequately designed and conducted experimental studies in animals can usually provide an exposure level below which adverse effects are not seen.

Interpreting these data and extrapolating from them to human populations can be difficult, as health effects vary with dose, route of exposure (e.g. ingestion, inhalation, skin absorption), frequency or duration of exposure, and the species, sex and age of the exposed population. This can require appropriate expertise and prudent judgment (e.g. see IPCS 1978).

Derivation of guideline values for substances for which a threshold exists

Where appropriate human data are available, these have been used in the derivation of the guideline value.

In the absence of human data, the guideline value is generally based on the highest dose that causes no adverse effects in long-term experiments on laboratory animals. It is calculated using the following formula:

$$\text{Guideline value} = \frac{\text{animal dose} \times \text{human weight} \times \text{proportion of intake from water}}{\text{volume of water consumed} \times \text{safety factor}}$$

In using this equation, it is necessary to make assumptions about the amount of water consumed per day, the average body weight and the proportion of total intake that can be attributed to water consumption, and to decide on an appropriate safety factor. Clearly the figures selected will all affect the guideline value, and varying one or more of them could raise or lower the resultant value by a factor of 10 or more. Any guideline value will thus have a degree of 'fuzziness' surrounding it; however, the assumptions made in calculating these guideline values are generally very conservative, and always err on the side of safety.

Animal dose

The animal dose is usually the 'no observed effect level' (NOEL); that is, the highest amount of the compound that does not cause observable effects in repeat dose studies on experimental animals. If this is not available, then the dose often used is the 'lowest observed effect level' (LOEL); that is, the lowest amount of the compound that causes observable effects in studies on experimental animals. (In some cases, the 'no/lowest observed *adverse* effect level, NOAEL/LOAEL, is used – that is, the highest amount of the compound that causes no observable adverse effects, or the lowest amount that causes observable effects, in repeat dose studies on experimental animals). If the LOEL (or LOAEL) is used, an extra safety factor is applied.

The dose data can come from drinking water studies or feeding or force-feeding studies. Dose is expressed as milligrams of compound per kilogram of animal body weight per day.

Human weight

It has been assumed that the average weight of an Australian adult is 70 kg. This is the figure used in Canada and other developed countries. The World Health Organization (WHO) uses a value of 60 kg, which reflects the lower adult weights in developing countries. The heavier weight assumed here will slightly increase the magnitude of the guideline value.

Where there is a specific need to protect young children, the average weight of a child at 2 years of age is assumed to be 13 kg. The same figure is used in other developed countries, such as Canada. The WHO uses 10 kg.

Proportion of intake from water

The animal dose data are assumed to encompass all sources of exposure. It is thus necessary to estimate the proportion of total human intake of a compound that is derived from water. Intake from air is generally negligible compared with other sources, but intake from food, pharmaceuticals and other products can be significant.

For chemicals that are used commercially or industrially, it is assumed, in the absence of other information, that water contributes 10 per cent of intake. For compounds that are not used commercially or industrially, a higher proportion of intake (usually 20 per cent but sometimes 80 per cent or 100 per cent) is assumed to come from drinking water. These figures are regarded as conservative (assuming a higher proportion deriving from drinking water would result in raising the guideline value), and the approach is consistent with that adopted by the WHO and by other countries.

Although exposure to chemical agents in water is predominantly through drinking the water, skin absorption during bathing or inhalation in a shower can also occur. Such exposures may increase the proportion of the chemical derived from drinking water, but the lower proportion (10 per cent or 20 per cent) is used for calculating the guideline value because it provides a higher margin of safety.

Volume of water consumed

The amount of water consumed by an adult each day is assumed to be 2 L. If the guideline value is based on the weight of a child, 1 L per day is assumed. Consumption can vary with season and climate; however, both figures, which are the same as those used by the WHO, are believed to be appropriate, on average, for Australian conditions. Some colder countries use different values: Canada, for example, uses 1.5 and 0.75 L per day.

Safety factor

Safety factors are used because of the uncertainty inherent in extrapolating from animal studies to human populations, or from a small human group to the general population. Safety factors generally applied are:

- a factor of 10 for variations between animals of the same species (because some animals within a species may be more sensitive to the effects of a chemical than the group tested);
- a factor of 10 for variations between species (because the animal species tested may be less sensitive than humans, and in many cases human sensitivity is unknown);
- a factor of 10 if data from a sub-chronic study are used in the absence of reliable data from chronic studies (this factor can be less if chronic studies are available and indicate that no other effects occur, or that other effects are mild);
- a factor of up to 10 if adverse effects have been observed at the lowest doses (usually the data used are based on the highest dose at which no adverse effects are seen).

The individual factors for each of the points listed above are multiplied together to give an overall safety factor. A safety factor of 100 to 1000 is common; higher values may be used on occasions.

Occasionally, individual safety factors lower than 10 are used where there is additional information to justify a reduction. This can occur, for instance, where information is available to clarify the mechanism of the effects on humans, where human epidemiological data are available, where the adverse effects observed are regarded as being relatively minor, or where large amounts of animal and human data are available.

Guideline values for carcinogenic compounds that act only above a threshold dose are determined in the same way as for non-carcinogenic compounds, but with an additional safety factor for carcinogenic effects.

Derivation of guideline values for substances where no threshold has been demonstrated

With compounds for which no threshold can be demonstrated, it can be expected that, as the level of exposure decreases, the resultant hazard similarly decreases. The risk associated with exposure to very low concentrations may be extrapolated using a risk assessment model, often over many orders of magnitude, from the dose–response relationship observed at higher doses. A number of uncertainties are involved, but the calculations used tend to overestimate rather than underestimate the risk, and so provide a greater margin of safety: it is possible that the actual risk from exposure to low concentrations may, in fact, be lower than the estimated values by more than an order of magnitude.

This approach can be applied for genotoxic carcinogenic compounds, and has been used by the WHO for this purpose.

Benchmark dose (BMD) approach

In a few cases, a slight variation to the above approaches for setting guideline values has been used. This variation, known as the benchmark dose (BMD) approach, has been used in dealing with both cancer and non-cancer end points. It is described in Environmental Health Criteria 170 (WHO 1994) and a modified version for use with carcinogenic soil contaminants was described by the NHMRC (1999).

The benchmark dose corresponds to a predetermined increase (between 1 and 10% but commonly 5%) of a defined effect in a test population. Mathematically it is the statistical lower confidence limit on the dose that corresponds to that predetermined increase, although some agencies are using a best estimate rather than a lower confidence limit (IEH, 1999).

Guidance on rounding

The vast majority of numerical guideline values in the ADWG are rounded to a single significant figure. Consistent with standard rounding convention, mid-way values are rounded up. For example, 1.5 is rounded to 2 and 25 is rounded to 30. Trailing zeros in numbers where there is no decimal point should not be taken as significant (e.g., nitrate, 50 mg/L).

Practically all of the health-based guideline values were established using data and assumptions with a precision of one significant figure (e.g., volume of water consumed by an adult = 2 L/day). Furthermore, the vast majority include the incorporation of safety factors, which are applied at the precision of 'order of magnitude' (e.g., 10 for interspecies extrapolation and 10 for intra-species variation).

Quoting more significant figures misrepresents the degree of calculated precision and may lead to unfounded concern when guidelines are exceeded at the second or third significant figure.

It is noted that exceptions to this may be necessary for some chemicals. These will be considered on a case-by-case basis and the reasons for the deviation from the convention of rounding to a single significant figure will be explained in the fact sheet.

It is noted that aesthetic guidelines are generally based on direct information on palatability to consumers, including appearance, taste and odour, and so do not need to be rounded.

Interaction between chemicals

Guideline values are calculated for individual chemicals without specific consideration of the potential for each to interact with others in the water. Normally, the majority of chemicals will not be present in concentrations at or near the guideline value, and the large margin of safety incorporated in the majority of the guideline values is considered to be sufficient to account for potential interactions with other substances.

Guidance on parent compounds and environmental transformation products

Fact sheets and health-based guideline values are established for the form(s) of the chemical that may be present in drinking water. Where the chemical form present in drinking water is an environmental transformation product, toxicological data on the transformation product(s) should be evaluated to derive a health-based guideline value if available.

Use of screening assays

For some chemicals, the accepted analytical method is a screening assay that is able to detect any of a group of chemicals, with the measured value derived from the members of the group that are present. For example, in analytical chemistry, a *residue method* involves breaking down the sample and measuring a breakdown product (for example, subjecting the sample to acid digestion and measuring carbon disulphide, CS_2). Also, some bioanalytical methods detect groups of chemicals based on their biological activity, for example estrogenic compounds. Using such methods, it may not be possible to determine the parent compound(s) present in the original sample. In this case, the result should be expressed in units of the most potent compound (or a well-established method reference compound) and compared to the relevant guideline value, or follow-up analytical techniques should be applied that measure the chemical(s) of interest directly.

6.4 Differences between Australian and WHO guideline values

The guideline values in the ADWG take as their point of reference the WHO *Guidelines for Drinking-water Quality* (2004) and subsequent addenda in 2006 and 2008. When the guideline values derived for chemicals in the ADWG differ from those recommended by the WHO, the difference usually arises in one of two ways:

- The ADWG use an average adult weight of 70 kg, consistent with developed countries such as Canada, whereas the WHO figure is 60 kg to cater for lighter body weights in developing countries. The use of a higher average weight can sometimes yield slightly higher guideline values, but the difference is not significant given the large safety factors used.
- For genotoxic carcinogenic compounds, WHO uses a risk assessment calculation, with the guideline value set at the concentration that would give rise to a risk of one additional cancer per 100,000 people. The Australian guideline values for these types of compounds are based on a consideration of:
  - the limit of determination based on the most common analytical method;
  - the concentration, calculated by the WHO using a risk assessment model, that could give rise to a risk of one additional cancer per million people, if water containing the compound at that concentration were consumed over a lifetime;
  - a value based on a threshold effect calculation, with an additional safety factor for potential carcinogenicity.

Frequently the values determined from these two types of calculations are very similar. The balance between these considerations is assessed as follows:

- If the limit of determination gives an adequate degree of protection (i.e. is within a factor of 10 of values determined from health considerations), it has been used as the guideline value. If the limit of determination is much lower than values determined from health considerations, then the lower of the two calculated values has been used. If, conversely, the calculated value is much lower than the limit of determination, then the calculated value is used, but with a note that it is lower than the practical limit of determination. Improved limits of determination are required for such compounds.
- The approach used for carcinogenic compounds in the ADWG is believed to lead to a more balanced assessment of the health risks, and is similar to that adopted in other countries (e.g. Canada). Whether the assumed risk should be one in 100,000 or one in a million is a value judgment. However, the greater degree of protection afforded by a risk of one in a million is generally consistent with calculations based on a threshold approach, and is in line with the high expectations of Australian consumers.

6.5 National and international guideline values (2016)

For some chemical substances, an Australian Drinking Water Guideline value may not be available. It is recommended that water suppliers seek advice from the appropriate state or territory health regulatory agency when chemicals that do not have a guideline value in the ADWG are detected in drinking water. In such cases, interim water quality advice may be obtained from alternative sources.

The following list details a hierarchy of documents in which national and international drinking water guideline values can be found. The sources are listed in order of preference of acceptance, based on recommendations from the NHMRC and the Environmental Health Committee (enHealth) in relation to risk assessment of environmental hazards (enHealth 2012). The recommendations are derived from the relevance to the Australian context and the methodologies used to calculate guideline values. Starting at the top of the hierarchy, the most recent final version of the document should be consulted until a suitable interim guideline value is identified:

- Australian Guidelines for Water Recycling – Phase 2: Augmentation of Drinking Water Supplies (Environment Protection and Heritage Council (EPHC), 2008)
- WHO Guidelines for Drinking-Water Quality, fourth edition (WHO, 2011)
- Drinking Water Standards for New Zealand, New Zealand Ministry of Health, New Zealand (New Zealand Ministry of Health, 2008)
- Guidelines for Canadian Drinking Water Quality (Health Canada, 2014)
- Health Advisories for Drinking Water Contaminants (United States Environmental Protection Agency (US EPA) Office of Water, various dates)
- Drinking Water Contaminants Lists (US EPA Office of Water, 2007)
- Public Health Goal for Chemical Substances in Drinking Water (Office of Environmental Health Hazard Assessment, California Environmental Protection Agency 2014)

6.6 References

Black Mountain Declaration on Endocrine Disrupting Chemicals in Australian Waters 2007 (2007), Canberra, Australia. http://www.clw.csiro.au/conferences/ourwater/EDC-conference-declaration.pdf

Damstra T, Barlow S, Bergman A, Kavlock R, Van Der Kraak G (Eds) (2002). *Global Assesment of the State-of-the-Science of Endocrine Disruptors*. Word Health Organization, International Programme on Chemical Safety, Geneva, Switzerland.

EPHC NHMRC NRMMC (2008) Australian Guidelines for Water Recycling – Phase 2: Augmentation of Drinking Water Supplies http://www.environment.gov.au/system/files/resources/9e4c2a10-fcee-48ab-a655-c4c045a615d0/files/water-recycling-guidelines-augmentation-drinking-22.pdf (Accessed 19 January 2016)

enHealth (2012). *Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards*, enHealth Council of Australia and Department of Health and Ageing, Commonwealth of Australia.

Health Canada (2014). *Guidelines for Canadian Drinking Water Quality*. http://www.hc-sc.gc.ca/ewh-semt/water-eau/drink-potab/guide/index-eng.php (Accessed 19 January 2016)

IARC (International Agency for Research on Cancer) (1991). *IARC monographs on the evaluation of carcinogenic risks to humans: chlorinated drinking water; chlorination by-products; some other halogenated compounds; cobalt and cobalt compounds*. WHO, IARC, 52, Lyon.

IEH (Institute for Environment and Health) (1999). Risk assessment approaches used by UK Government for evaluating human health effects of chemicals. Leicester: IEH.

IPCS (International Programme on Chemical Safety) (1978). *Principles and methods for evaluating the toxicity of chemicals: Part I*. Environmental Health Criteria, 6. WHO, IPCS.

Jensen K M, Makynen EA, Kahl MD, Ankley GT (2006). Effects of the feedlot contaminant 17 α-trenbolone on reproductive endocrinology of the fathead minnow. *Environmental Science and Technology,* 40(9):3112-3117.

Kolodziej EP, Harter T, Sedlak DL (2004). Dairy wastewater, aquaculture, and spawning fish as sources of steroid hormones in the aquatic environment. *Environmental Science and Technology,* 38(23):6377-6384.

Kronberg L, Vartiainen T (1988). Ames mutagenicity and concentration of the strong mutagen 3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone and its geometric isomer E-2-chloro-3-{dichloromethyl)-4-oxo-buteinoic acid in chlorine treated tap water. *Mutation Research,* 206:177-82.

Kummerer K (2009). Antibiotics in the aquatic environment: A review, Part II. ***Chemosphere***, 75(4):435-441.

Michaud DS, Kogevinas M, Cantor KP, Villanueva CM, Garcia-Closas M, Rothman N, Malats N, Real FX, Serra C, Garcia-Closas R, Tardon A, Carrato A, Dosemeci M, Silverman DT (2007). Total fluid and water consumption and the joint effect of exposure to disinfection by-products on risk of bladder cancer. *Environmental Health Perspectives,* 115(11):1569-1572.

New Zealand Ministry of Health (2008). *Drinking Water Standards for New Zealand*, New Zealand Ministry of Health, New Zealand. http://www.health.govt.nz/publication/guidelines-drinking-water-quality-management-new-zealand (Accessed 19 January 2016)

NHMRC (National Health and Medical Research Council) (1999). Toxicity assessment for carcinogenic soil contaminants. Canberra: Commonwealth of Australia.

NRMMC (National Resource Management Ministerial Council), EPHC (Environment Protection and Heritage Council), NHMRC (National Health and Medical Research Council) (2008). *Australian Guidelines for Water Recycling: Managing Health and Environmental Risks – Augmentation of Drinking Water Supplies*. NWQMS Guideline document #22. www.ephc.gov.au/sites/default/files/WQ_AGWR_GL__ADWS_Corrected_Final_%20200809.pdf

Office of Environmental Health Hazard Assessment (2014). *Public Health Goal for Chemical Substances in Drinking Water*. California Environmental Protection Agency. http://www.oehha.ca.gov/water/phg/allphgs.html (Accessed 19 January 2016)

Rempel MA, Reyes J, Steinert S, Hwang W, Armstrong J, Sakamoto K, Kelley K, Schlenk D (2006). Evaluation of relationships between reproductive metrics, gender and vitellogenin expression in demersal

flatfish collected near the municipal wastewater outfall of Orange County, California, USA. *Aquatic Toxicology,* **77**(3):241-249.

Tyler CR, Jobling S (2008). Roach, sex, and gender-bending chemicals: the feminization of wild fish in English rivers. *Bioscience,* 58(11):1051-1059.

United States Environmental Protection Agency (US EPA) (2007). *Drinking Water Contaminants Lists*. Office of Water, US EPA. http://water.epa.gov/drink/contaminants/index.cfm (Accessed 19 January 2016)

United States Environmental Protection Agency US EPA (Various dates). *Health Advisories for Drinking Water Contaminants*. Office of Water, US EPA. http://water.epa.gov/drink/standards/hascience.cfm (Accessed 19 January 2016)

Villanueva CM, Cantor KP, Grimalt JO, Malats N, Silverman D, Tardon A, Garcia-Closas R, Serra C, Carrato A, Castano-Vinyals G, Marcos R, Rothman N, Real FX, Dosemeci M, Kogevinas M (2007). Bladder cancer and exposure to water disinfection by-products through ingestion, bathing, showering, and swimming in pools. *American Journal of Epidemiology,* 165(2):148-156.

WHO (World Health Organization) (1994). *Assessing human health risks of chemicals: derivation of guidance values for health based exposure limits*. Environmental Health Criteria 170. IPCS/WHO. Geneva: WHO.

WHO (World Health Organization) (2011). *Guidelines for Drinking-Water Quality*, fourth edition. http://www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/ (Accessed 19 January 2016)

CHAPTER 7 Radiological Quality of Drinking Water (Revised 2004)

Chapter 7 Radiological Quality of Drinking Water (Revised 2004)

7.1 Introduction

This chapter describes the sources of radiation in the environment and in drinking water, the health effects of radiation, how people are exposed to radiation and how radiation exposure is measured. It also explains how the guideline values provided in Chapter 10 are derived.

7.2 Sources of radiation in the environment and in drinking water

Radioactive materials occur naturally in the environment (e.g. uranium, thorium and potassium). Some radioactive compounds arise from human activities (e.g. from medical or industrial uses of radioactivity) and some natural sources of radiation are concentrated by mining and other industrial activities.

By far the largest proportion of human exposure to radiation comes from natural sources – from external sources of radiation, including cosmic radiation, or from ingestion or inhalation of radioactive materials. A very low proportion of the total human exposure comes from drinking water. Radiological contamination of drinking water can result from:

- naturally occurring concentrations of radioactive species (e.g. radionuclides of the thorium and uranium series in drinking water sources);
- technological processes involving naturally radioactive materials (e.g. the mining and processing of mineral sands or phosphate fertiliser production);
- manufactured radionuclides, which might enter drinking water supplies from the medical and industrial use of radioactive materials.

7.3 Health effects of radiation

There is evidence from both human and animal studies that radiation exposure at low to moderate doses may increase the long-term incidence of cancer. There is also evidence from animal studies that the rate of genetic disorders may be increased by radiation exposure.

Acute health effects of radiation, ranging from skin burns to nausea, vomiting, diarrhoea, reduced blood cell counts and death, occur at much higher doses and therefore are not a concern for water supplies except in extreme accident situations.

7.4 Exposure to radiation

Several different forms of radiation can be emitted by radioactive species (alpha particles, beta particles and positrons, gamma rays and x-rays). Each form has different biological effects. Alpha particles have very low penetration of tissue but cause considerable cell damage over a short range. Radionuclides that emit alpha particles are therefore only a hazard if they are taken into the body (internal irradiation). Beta particles are more penetrating than alpha particles but on external exposure do not penetrate to internal organs. Gamma radiation and x-rays, on the other hand, are highly penetrating and radioactive sources of these types of radiation are an external radiation hazard.

Humans are irradiated internally if they ingest radioactive substances in food and water or inhale radioactive components in air. Radionuclides that enter the body in this way can remain in a particular organ or tissue for a long time, resulting in exposure over many months or, in some cases, years. Exposure to radiation from contaminated water comes from internal radiation by ingested radionuclides.

7.5 Units of radioactivity and radiation dose measurement

7.5.1 UNITS OF RADIOACTIVITY AND RADIATION DOSE

The International System of Units (SI) unit of radioactivity is the becquerel, where 1 Bq = 1 disintegration per second.

The radiation dose resulting from ingestion of a radionuclide depends on a number of chemical and biological factors. These include the fraction of the intake that is absorbed from the gut, the organs or tissues to which the radionuclide may be transported and deposited, and the time that the radionuclide might remain in the organ or tissue before excretion. The nature of the radiation emitted on decay and the sensitivity of the irradiated organs or tissues to radiation must also be considered.

The absorbed dose refers to how much energy is deposited in material by the radiation. The SI unit for absorbed dose is the gray (Gy). The equivalent dose is the product of the absorbed dose and a factor related to a particular type of radiation. The equivalent dose of radiation received by a person can be further quantified as the effective dose, which, in simple terms, is the sum of the equivalent doses received by all tissues or organs, weighted to account for the different sensitivities to radiation of different organs and tissues in the human body. The SI unit for effective dose is the sievert (Sv).

To reflect the persistence of radionuclides in the body, once ingested, the 'committed effective dose' is a measure of the total effective dose received over a lifetime (50 years) following intake of a radionuclide (internal exposure).

The term 'dose' is used as a general term to mean either absorbed dose (Gy) or effective dose (Sv), depending on the situation. For monitoring purposes, however, 'doses' are determined from the concentration of the radionuclide, which in the case of water is described in terms of Bq/L. This value is converted to an effective human dose per year using a dose conversion factor and the average annual consumption of water.

7.5.2 DOSE CONVERSION FACTORS

The dose arising from the intake of 1 Bq (by ingestion) of radioisotope in a particular chemical form can be estimated using a dose conversion factor. Data for age-related dose conversion factors for ingestion of radionuclides have been published by the International Commission on Radiological Protection (ICRP 1996). Table 7.1 shows the conversion factors or dose per unit intake (mSv/Bq) for radionuclides (naturally occurring or arising from human activities) that could be found in water supplies.

Table 7.1 *Dose per unit intake by ingestion for adult members of the public (ICRP 1996)*

Category	Radionuclide	Dose per unit intake (mSv/Bq)
Natural uranium series	Uranium-238	4.5×10^{-5}
	Uranium-234	4.9×10^{-5}
	Thorium-230	2.1×10^{-4}
	Radium-226	2.8×10^{-4}
	Lead-210	6.9×10^{-4}
	Polonium-210	1.2×10^{-3}
Natural thorium series	Thorium-232	2.3×10^{-4}
	Radium-228	6.9×10^{-4}
	Thorium-228	7.2×10^{-5}
Fission products	Caesium-134	1.9×10^{-5}
	Caesium-137	1.3×10^{-5}
	Strontium-90	2.8×10^{-5}
	Iodine-131	2.2×10^{-5}
Other radionuclides	Tritium	1.8×10^{-8}
	Carbon-14	5.8×10^{-7}
	Plutonium-239	2.5×10^{-4}
	Americium-241	2.0×10^{-4}
	Potassium-40	6.2×10^{-6}

7.5.3 AVERAGE HUMAN DOSE OF RADIATION

The dose of radiation received varies significantly between individuals and communities, and depends on locality, lifestyle, diet and type of dwelling. The global average for the individual dose of radiation from natural sources has been estimated to be 2.4 mSv per year (UNSCEAR 2000). Of this annual dose, less than 10 per cent comes from ingestion of food and drinking water containing radium and other radionuclides of the natural uranium and thorium series. Australian data suggest that the average annual dose in this country may be slightly lower, at approximately 2 mSv per year (Webb ***et al.*** 1999).

7.6 Approach for derivation of guideline values for radionuclides

The *Australian Drinking Water Guidelines* (ADWG) provide:

- a single guideline value for the annual exposure to radioactivity in drinking water;
- a method to assess the radiological quality of water;
- a simple screening method to assure compliance with the guideline;
- a method for assessing water if screening levels for gross radioactivity are exceeded.

This approach reduces the need for routine, costly and time-consuming analyses to identify individual radionuclides present in the water.

7.6.1 PRACTICES AND INTERVENTIONS

The ADWG are based on the recommendations of the ICRP (ICRP 1991, 2000) and the NHMRC (NHMRC 1995). Both organisations distinguish between 'practices' and 'interventions' as follows:

- A 'practice' is a situation where the dose of radiation received is increased by the activities under consideration; for example, the development of a uranium mine or nuclear power station. Radiation dose limits can be imposed on the operation so that compliance with these limits reduces risks from the 'practice' to levels considered 'acceptable'. If the facility cannot be designed or operated to comply with the radiological protection standards, then the facility can be forced to close.
- An 'intervention' may be required when the public are exposed to a radiation source that is already present and incidental to the situation under consideration. Such situations include exposure to natural sources of radiation, or exposure from abandoned radioactive waste from past operations. Frequently, these situations result in prolonged radiation exposures. Action to reduce the radiation dose to the exposed population may therefore be warranted and is called an 'intervention'.

Reducing the radiation exposure from radionuclides in drinking water requires an intervention. For example, the supply may be treated to reduce the levels of radioactive contaminants, an alternative supply may be substituted, or, in the extreme case, the population may be relocated to an area where better quality water is available.

The levels considered acceptable in practice provide a basis for setting levels that require an intervention.

7.6.2 ESTIMATION OF THE DOSE FROM RADIONUCLIDES IN WATER

To estimate the equivalent dose to members of the public from the ingestion of radionuclides in drinking water, the parameters required are the concentration of the radionuclides in water (measured in Bq/L), the daily consumption rate of water (L/day), and the dose conversion factor for the particular radionuclide.

The World Health Organization (WHO) has estimated that adults consume an average of 2 L of water per day, and this figure is believed to be an appropriate average figure for Australia, giving an annual consumption of 730 L for each adult Australian. Therefore, the amount of each radionuclide ingested per year from the water supply is the concentration of that radionuclide in the water (Bq/L) multiplied by 730.

The annual dose from an individual radionuclide consumed in water is calculated using the following equation:

Annual dose =	dose per unit intake	x	annual water consumption	x	radionuclide concentration
(mSv/year)	(mSv/Bq)		(litre/year)		(Bq/L)

Usually, a water supply contains more than one radionuclide; therefore, the doses arising from each individual radionuclide must be summed to give the total dose.

7.6.3 ESTIMATION OF RISK FROM LOW-LEVEL RADIATION

Lifetime

Because of the very low level of exposure resulting from consumption of drinking water containing radionuclides, and the radionuclides involved, it is not possible to distinguish a radiation-induced cancer incidence from the baseline level of cancers in the general population. Therefore, the health risks must be estimated by extrapolation from the effects at higher doses.

The ICRP (1991) estimates the lifetime risk of a fatal cancer resulting from exposure to radiation to be 5 x 10-2 per Sv of radiation dose, that is, five additional fatal cancers for every 100 people exposed per year. On the basis of this estimate, a dose of 1 mSv per year gives an annual risk of 5 x 10-5, that is, about five additional fatal cancers per 100,000 people exposed per year. (Additional fatal cancers are those that occur in addition to those that result from all other causes.)

Any increase in genetic disorders (including birth defects) is expected to be very much less than any increase in the cancer rate and therefore an acceptable dose level for cancer risk will also be protective for genetic risk.

Acceptable dose from drinking water

Both the ICRP (1991) and the NHMRC (1995) recommend that the need for, and the extent of, intervention to reduce radiation exposure should be determined on the basis of a generalised cost–benefit analysis, where the resulting public health benefit should be balanced against the overall costs of achieving a reduction in radiation exposure. The outcome of this type of analysis will almost always be specific to a particular situation because the costs of reducing exposure vary widely depending on the situation. It is thus not possible to set a completely generic level at which intervention must be undertaken to reduce the radiation dose from radionuclides in water supplies.

Guidance can, however, be gained from the recommendations of Lokan (1998) and the ICRP (2000) on the protection of the public in situations of prolonged exposure. The ICRP noted that, on radiological grounds alone, intervention may not be necessary for doses below 10 mSv per year. However, this applies to the total dose from all sources of exposure. The ICRP also recommended that, for commodities that are essential for normal living and are amenable to intervention, an individual dose of approximately 1 mSv per year is an acceptable intervention exemption level (ICRP 2000). This is consistent with the recommendation of the NHMRC (1995) of a public exposure limit for practices of 1 mSv per year from all sources.

Furthermore, Lokan (1998) concluded that a value of 1 mSv per year might be appropriate as a default action level above which some corrective action will be necessary.

7.6.4 GUIDELINE VALUE FOR DRINKING WATER

Based on the above, it is recommended that a guideline dose of 1 mSv per year should be applied for radioactivity in drinking water. When the existing or potential dose from the radionuclide content exceeds this guideline dose, a decision on the need for and the degree of remedial action (intervention) should be based on advice from the relevant state health authorities, and should include a cost–benefit analysis.

There may be some circumstances where there is no practical alternative but to accept a dose that exceeds the guideline dose of 1 mSv, together with a potential slight increase in the risk to health as a consequence. However, if doses from the use of a particular water supply will exceed 10 mSv per year, immediate action must be taken to reduce the existing or potential exposures.

7.6.5 APPLICATION OF GUIDELINE VALUES

This guideline deals only with situations where the radionuclide concentrations arise either from natural sources, or, more rarely, as the result of past practices (such as abandoned mining operations). It specifically does not apply to situations where the radionuclides arise from current practices under regulatory control, such as an operating uranium mine.

Therefore, the guideline should not be used to support an increase in the radionuclide concentrations of drinking water as a result of an operation, on the grounds that the overall dose levels remain below 1 mSv per year.

7.7 References and Bibliography

APHA/AWWA/WEF (American Public Health Association/American Water Works Association/Water Environment Federation) (1998). *Standard methods for the examination of water and wastewater.* 20th edition, Washington DC.

AS 2531 (1982). *Australian Standard for Waters — Determination of gross alpha and gross beta activities.* Standards Association of Australia, Sydney.

AS/NZS 5667.1 (1998a). *Water quality – Sampling – Guidance on the design of sampling programs, sampling techniques and the preservation and handling of samples.*

AS/NZS 5667.5 (1998). *Water quality – Sampling – Guidance on sampling of drinking water and water used for food and beverage processing.*

ICRP (International Commission on Radiological Protection) (1991). Recommendations of the ICRP. Annals of the ICRP 21(1.3). ICRP Publication 60, Pergamon Press, Oxford, United Kingdom.

ICRP (International Commission on Radiological Protection) (1996). Age-dependent doses to members of the public from intake of radionuclides: Part 5 Compilation of ingestion and inhalation dose coefficients. ICRP Publication 72, Pergamon Press, Oxford, United Kingdom.

ICRP (International Commission on Radiological Protection) (2000). Protection of the public in situations of prolonged radiation exposure. ICRP Publication 82, Pergamon Press, Oxford, United Kingdom.

ISO 9695 (International Organization for Standardization) (1991). Water quality — measurement of gross beta activity in non-saline water — thick source method. International Standard 9695. International Organization for Standardization, Geneva, Switzerland.

ISO 9696 (International Organization for Standardization) (1991). Water quality — measurement of gross alpha activity in non-saline water — thick source method. International Standard 9696. International Organization for Standardization, Geneva, Switzerland.

Lokan KH (1998). Drinking water quality in areas dependent on groundwater. *Radiation Protection in Australasia* (1998) 15(1):11–14.

NHMRC (National Health and Medical Research Council) (1995). Recommendations for limiting exposure to ionising radiation (1995). Radiation Health Series No. 39, Government Publishing Service, Canberra.

UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (2000). Sources, effects and risks of ionising radiation. Report to the General Assembly. ISBN 92-1-142238-8, New York.

Webb DV, Solomon SB, Thompson JEM (1999). Background radiation levels and medical exposure levels in Australia. *Radiation Protection in Australasia*, 16(2):25.

CHAPTER 8 Drinking Water Treatment Chemicals (Revised 2006)

Chapter 8 Drinking Water Treatment Chemicals (Revised 2006)

8.1 Introduction

The production of safe reticulated drinking water is vital for society. In recent decades, there have been numerous examples throughout the world of poor water quality having an adverse impact on human health. Such episodes are rare in Australia, but the dire consequences of compromised disinfection and blooms of cyanobacteria serve to remind us of the need for drinking water treatment.

Addition of chemicals to make water safe for consumption is widely practised by the water industry and has generally been accepted by the community. However, safeguards must be sufficient to ensure that any residual amount of these chemicals, by-products of their reactivity or minor contaminants in their formulations, do not pose an unacceptable health risk.

Treatment chemicals are added to drinking water mainly to reduce or eliminate the incidence of waterborne disease, for other public health measures, and to improve the aesthetic quality of the water. Any chemical used in, on, or near drinking water sources, or used during the treatment of drinking water should:

- be effective for the desired outcome;
- not present a public health concern;
- not result in the chemical, its by-products or any contaminants exceeding *Australian Drinking Water Guidelines* (ADWG) values.

This chapter provides guidance on chemicals used during the storage, treatment, and distribution of drinking water and quality assurance procedures.

8.2 Scope and limit of application of this chapter

Chemicals used near water for purposes other than direct improvement of water quality are not considered as drinking water treatment chemicals. Such chemicals include fertilisers and other agricultural chemicals used in properties adjacent to water storages, herbicides used to reduce vegetation along waterways, and pesticides used to control mosquitoes and other disease vectors in water storages. Use of these chemicals near raw water sources should be carefully considered, and the risks associated with their use should be minimised to ensure that water quality and public health are not jeopardised. Further information on these chemicals is given in Section 6.3.3 and in the *Australian and New Zealand Guidelines for Fresh and Marine Water Quality* (NWQMS 2000).

This chapter does not cover the specialised chemicals used in water treatment for non-potable uses (e.g. chemicals used in industrial boilers and air conditioning cooling towers), nor does it cover the impact on water quality of materials in direct contact with water. Information on these chemicals and impacts is given in Australian Standards AS/NZS 3666.1:2002 *Air handling and water systems of buildings – Microbial control – design, installation and commissioning*; AS/NZS 5667.7:1998 *Water quality – Guidance on sampling of water and steam in boiler plants*; and AS/NZS 4020:2002 *Testing of products for use in contact with drinking water* respectively.

Information on occupational exposure to drinking water treatment chemicals resulting from their manufacture, transportation or use should be obtained from the manufacturer and Material Safety Data Sheets (MSDS), or from the appropriate state or territory occupational health and safety authority (see Section 8.9).

8.3 Overview of chemical treatment processes

In the production of drinking water, a number of different chemicals may be added to the water. The types and quantities of chemicals can vary widely and will depend on a range of factors including raw water quality, treatment processes employed and treated water quality objectives. Chemical treatment processes are used to:

- control algae;
- remove turbidity and colour;
- remove microorganisms;
- remove algal metabolites and synthetic pollutants;
- reduce organic matter;
- reduce the concentration of iron, manganese and other elements;
- reduce pesticides and herbicides;
- control taste and odour;
- soften;
- buffer or modify the pH;
- disinfect;
- control corrosion in distribution systems.

Chemical treatments may also be used for other public health measures, including fluoridation (to prevent dental caries).

The following sections outline common processes employed in water treatment to achieve these objectives.

8.3.1 CONTROL OF ALGAE

Algicides are used to reduce toxic or odorous algal blooms in water reservoirs. The chemical commonly used in the management of algal growth is copper sulfate. Before an algicide is used, the possible effects on aquatic biota, the accumulation of copper in sediments, the potential impacts on downstream treatment processes, and final treated water quality should be considered.

The use of copper as an algicide is controlled in some states. Information on the use of algicides should be obtained from the appropriate state or territory authority (see Section 8.9).

8.3.2 COAGULATION AND FLOCCULATION

The primary use of coagulant and flocculant chemicals is in the removal of suspended and colloidal solids such as clays. Coagulation is particularly important in the treatment of surface waters. Removal of the solids is achieved by aggregating fine suspended matter into larger flocs. Coagulant and flocculant chemicals will also remove some natural organic matter, colour and microorganisms (e.g. bacteria, viruses and algae). The size and strength of the floc can be controlled and modified, depending on the treatment process in use, and the floc can be removed by sedimentation and filtration.

8.3.3 ADSORPTION

Adsorption is primarily used to improve water quality through the accumulation of substances at the interface between two phases, such as a liquid and a solid, due to chemical and physicochemical interactions. The solid on which adsorption occurs is called the adsorbent. Activated carbon is an excellent adsorbent.

Adsorption is commonly used to remove organic contaminants such as herbicides, pesticides, algal toxins and metabolites; it is also used to remove compounds that may have an adverse impact on the taste and odour of water.

8.3.4 SOFTENING

Softening is undertaken as part of water treatment to remove calcium and magnesium salts, particularly carbonates and bicarbonates, which cause water hardness. Hard water can cause scale build-up on water heating elements, and problems with the use of soaps and detergents. Softening very hard waters can also lead to high concentrations of sodium in water. While this may possibly give the water a salty taste, it is unlikely to present a health concern. Water that is too soft can be corrosive, which may occur when reverse osmosis is being used for water treatment, in which case it may be necessary to restore some hardness to prevent corrosion.

8.3.5 OXIDATION

Various oxidants may be added to water to oxidise problem compounds. For example, chlorine or potassium permanganate may be added to control iron and manganese. The oxidised forms of iron and manganese are readily removed by coagulation, flocculation and filtration. Oxidants may also be used to oxidise compounds that have an adverse impact on the taste and odour of water, and organic contaminants such as pesticides.

Ozone, and possibly hydrogen peroxide, may be added to oxidise organic compounds, and thus reduce the amount of coagulant required. Adding these chemicals also helps to reduce the length of long-chain organic molecules, which are then more effectively removed by granular activated carbon.

8.3.6 DISINFECTION

Disinfection of water is generally used either alone or as the final step in water treatment, after clarification or filtration. Disinfection is widely used to prevent the passage of bacteria, viruses and some protozoa into the distribution system. Typical chemicals used for disinfection of drinking water supplies are strong oxidants, such as chlorine (and its derivatives, chlorine dioxide and chloramine), ozone and hydrogen peroxide.

The efficiency of disinfection depends greatly on the quality of the source or treated water, and can also be strongly affected by conditions such as chemical contact time, the pH and turbidity of the water, and organic content of the water.

The aim of treatment processes used before disinfection should be to produce water with the lowest possible turbidity and organic content. Excessive particulate matter in the water can protect microorganisms from the action of disinfection chemicals. Also, excess organic matter and other oxidisable compounds in water can react with disinfection chemicals intended to inactivate microorganisms and can result in an increase in the formation of disinfection by-products (see Section 6.3.2 for general information on disinfection by-products, and the fact sheets in Section V for information on specific by-products). Best practice operation of a conventional water treatment plant should be able to produce treated water with a turbidity of less than 0.1 nephelometric turbidity units (NTU).

8.3.7 ADJUSTMENT OF PH

Adjustment of pH is important in drinking water treatment processes such as coagulation (particularly for the removal of natural organic matter), corrosion control and softening.

Control of pH is also important for effective disinfection and to minimise the formation of disinfection by-products. The efficiency of certain disinfectants is strongly dependent on pH.

8.3.8 ADDITION OF BUFFERING CAPACITY

Soft waters can be subject to pH change as they travel through the distribution system. The rate of change depends on a number of factors including the water hardness, the pipe materials and internal coating

used (e.g. cement lined pipe), the contact time, and temperature. Increasing the buffering capacity of the water can help control the rate of change of pH through the distribution system.

8.3.9 CORROSION INHIBITION

The mechanisms of corrosion in a water distribution system are complex, and involve an interrelated combination of physical, chemical and biological processes. These depend greatly on the materials used within the distribution system and the chemical properties of the water, particularly its buffering capacity. Water corrosivity can be minimised by adjusting pH and increasing calcium carbonate hardness (resulting in a positive Langelier index). Corrosion can also be reduced by maintaining disinfection residual throughout the distribution system.

Corrosion inhibition chemicals (such as sequestering agents) are used to reduce corrosion of pipes and household services. They also control the build-up of scale deposits from the dissolved mineral content of drinking water. This is achieved through the addition of chemicals that form a protective film on the surface of pipes. While corrosion inhibitors reduce corrosion, limit metal solubility or convert one form of corrosion to another (e.g. alleviating tuberculation and replacing it with more uniform corrosion), they do not totally prevent corrosion.

8.4 Public health measures

8.4.1 FLUORIDATION

Fluoridation of drinking water is not a water treatment process, but has been and continues to be effective in reducing the incidence of dental caries. It has many advantages over alternative methods for fluoridation, due to its cost-effectiveness, consistency of exposure, equal distribution to all socioeconomic groups, and safety. In some areas, fluoride occurs naturally in drinking water.

In areas where the drinking water supply is artificially fluoridated (at the instigation of the relevant state or territory health authorities), the process is generally undertaken after clarification and chlorination of the water, because fluoride ions may adsorb onto the surface of suspended matter in the water and be subsequently removed through these processes. Fluoridation is generally achieved by adding either a slurry of sodium fluorosilicate, a solution of hydrofluorosilicic acid or (less commonly) a saturated solution of sodium fluoride, added as a metered dose for a given rate of water flow. Correction of pH may be needed out after fluoride addition.

Use of fluoride is controlled by state and territory legislation and regulations, and local regulations. Some of these are outlined in Table 8.1 (see also Section 8.9).

Table 8.1 *State and Territory fluoride legislation and regulations*

Australian Capital Territory	• *Electricity and Water (amendment) Act (no 2) 1989*. No 13 of 1989—Section 13
New South Wales	• Fluoridation of Public Water Supplies Regulation 2002. <www.legislation.nsw.gov.au> • *Fluoridation of Public Water Supplies Act 1957*
Northern Territory	• *Dental Act Schedule 3 1999*
Queensland	• Fluoridation of Public Water Supplies Regulation 1998. Reprinted as in force on 4 January 1999 • *Fluoridation of Public Water Supplies Act 1963*. Reprinted as in force on 21 December 1998
South Australia	• There is no fluoride legislation in South Australia
Tasmania	• *Fluoridation Act 1968*
Victoria	• *Health (Fluoridation) Act 1973*
Western Australia	• *Fluoridation of Public Water Supplies Act 1966*

8.5 Assessment of Chemicals acceptable for use in drinking water treatment (revised 2016)

8.5.1 CHEMICALS ASSESSED PRIOR TO 2004

The addition of drinking water treatment chemicals to the ADWG as listed in Table 8.2 occurred in 2004. This list was based on the NHMRC Drinking Water Treatment Chemicals Committee's consideration of NHMRC's *Chemicals used for treatment of drinking water supplies* (1989). The acceptability of the chemical was dependent upon: the practical application of the chemical (e.g., to clarify dirty water, or destroy or inactivate harmful microorganisms); whether it achieved its purpose; and it being non-toxic when ingested at concentrations present in treated water.

As a result, the ADWG considers the chemicals in Table 8.2 suitable for use in water supplies where:

- standard operating procedures are applied;
- risk control measures to ensure effectiveness of a particular chemical are applied (e.g., controls to revent over- or under-dosing);
- it is ensured that residuals and contaminants from the addition of multiple treatment chemicals will not exceed recommended health-related guideline values at the consumer's tap, taking into account combined contributions from all treatment chemicals added and source water; and
- the potential for a chemical to interact with any other added chemical or other compounds present in the water has been considered.

The fact sheets in Part V provide detailed information on chemicals listed in Table 8.2. .

Table 8.2 *Chemicals recommended for use in the treatment of drinking water (2004)*

Treatment chemical	***Formula***	***Original date of approval by NHMRC***	***Uses***
Aluminium chlorohydrates	$AlCl(OH)_5$	2005	Coagulation
Aluminium sulfate (alum)	$Al_2(SO_4)_3$	1983	Coagulation
Ammonia	$NH_{3\ aq}$	1983	Generation of chloramines for disinfection
Ammonium sulfate	$(NH_4)_2SO_4$	1983	Generation of chloramines for disinfection
Calcium hydroxide (hydrated lime)	$Ca(OH)_2$	1983	pH correction Softening Corrosion control
Calcium hypochlorite	$Ca(OCl)_2$	1983	Disinfection/oxidation
Calcium oxide (quick lime)	CaO	1983	Coagulation aid pH correction Softening Corrosion control
Carbon, powdered activated/ granulated activated (PAC/GAC)	C	1983	Adsorption
Chlorine	Cl_2	1983	Disinfection/oxidation
Chlorine dioxide	ClO_2	2005	Disinfection/oxidation
Copper sulfate	$CuSO_4$	1983	Algicide
Ferric chloride	$FeCl_3$	1983	Coagulation
Ferric sulfates	$Fe_2(SO_4)_3$	1983	Coagulation
Hydrochloric acid	HCl	2005	pH correction
Hydrofluorosilicic acid (fluorosilicic acid)	H_2SiF_6	1983	Fluoridation

Treatment chemical	***Formula***	***Original date of approval by NHMRC***	***Uses***
Hydrogen peroxide	H_2O_2	1983	Disinfection Oxidation
Hydroxylated ferric sulfate		2005	Coagulation
Ozone	O_3	2005	Disinfection/oxidation
Polyacrylamides	$(C_3H_5NO)_n$	1977	Coagulation aid Flocculation aid Filter aid
Polyaluminium chlorides	$Al_n(OH)_mCL_{(3n-m)}$	1979	Coagulation
Poly aluminium silica sulfates	$Na_{12}(AlO_2)$ $(SiO_2)_{12}.xH_2O$	2005	Coagulation
Polydiallyldimethylammonium chlorides (polyDADMACs)		1982	Coagulation and coagulation aid
Potassium permanganate	$KMnO_4$	1983	Disinfection/oxidation
Sodium aluminates	$NaAlO_2$	1983	Coagulation
Sodium bicarbonate	$NaHCO_3$	1983	pH correction Softening Corrosion control
Sodium carbonate (soda ash)	Na_2CO_3	1983	pH correction Softening Corrosion control
Sodium fluoride	NaF	1983	Fluoridation
Sodium fluorosilicate	Na_2SiF_6	1983	Fluoridation
Sodium hexametaphosphate	$(NaPO_3)_x$	1983	Corrosion control
Sodium hydroxide (caustic soda)	NaOH	1983	pH correction Softening Corrosion control
Sodium hypochlorite	NaClO	1983	Disinfection/oxidation
Sodium silicate	Na_2SiO_3	1983	Coagulation aid Flocculation aid pH correction Corrosion control
Sodium tripolyphosphate	$Na_5P_3O_{10}$	2005	Corrosion control Softening
Sulfuric acid	H_2SO_4	1983	pH correction
Zinc orthophosphate	$Zn_3(PO_4)_2$	1987	Corrosion control

8.5.2 NEW WATER TREATMENT CHEMICALS

NHMRC does not hold the mandate to approve or recommend new drinking water treatment chemicals in addition to those listed in Table 8.2. However, the Framework in the ADWG provides guidance on the risk management principles that should be considered to demonstrate the safety (Section 3.4.5) and efficacy (Section 3.9) of chemicals to be used for the treatment of drinking water. The Framework also reinforces the need to consider the local factors such as source water quality and other treatment barriers in use when considering the utility of drinking water treatment chemicals. Health-based guideline values for specific chemicals are provided in the fact sheets in Part V.

As discussed in Section 2.4–2.5, application of the Framework will vary depending on the arrangements for water supply within each jurisdiction; for example, in some states, water supply is managed by one agency, whereas in other states it is managed locally by numerous water suppliers. Although the guidelines are not intended to be applied as standards, it is recognised that some jurisdictions may choose to regulate the guidelines through legislation or operating licences.

Chemical manufacturers, importers and water utilities wishing to use water treatment chemicals not listed in Table 8.2 are required to consult the relevant state or territory legislation, and liaise with the appropriate agencies. These agencies may include health departments, water resource departments, natural resource and environment departments, agriculture departments, local governments, planning authorities, and catchment water management boards. These agencies will take into consideration health, environmental, efficacy, and occupational health and safety issues.

A number of Commonwealth schemes have a role in chemical regulation. The National Industrial Chemicals Notification and Assessment Scheme (NICNAS), administered by the Department of Health, assesses new industrial chemicals prior to their manufacture or import into Australia and (on a priority basis) chemicals already in use in Australia. NICNAS also maintains the Australian Inventory of Chemical Substances (AICS). Industrial chemicals are chemicals that are not: therapeutic goods that are subject to regulation by the Therapeutic Goods Administration; food additives or chemicals in food for which standards are established by Food Standards Australia New Zealand; or pesticides and veterinary medicines that are subject to safety and efficacy assessment and registration by the Australian Pesticides and Veterinary Medicines Authority.

New industrial chemicals (those not listed on AICS), or chemicals listed on AICS that are proposed to be used other than in accordance with conditions of use (if any) annotated on AICS, must be assessed by NICNAS.

8.6 Quality assurance for drinking water treatment chemicals

8.6.1 RISKS ASSOCIATED WITH DRINKING WATER CHEMICALS

A cornerstone of the management of drinking water quality (see Chapters 2 and 3) is the analysis of hazards and the management of risk.

The intentional addition of chemicals to water intended for drinking purposes carries with it a potential risk. This may result from any of the following:

- the toxicological properties of the chemical itself;
- underdosing or overdosing of the chemical;
- contaminants in the chemical arising from the manufacturing process or the raw materials used;
- contaminants in the chemical arising during transport, storage and use on site;
- by-products formed through the use of the chemical.

Contamination of chemicals can be minimised by the use of good manufacturing practice, which uses quality control and quality assurance programs to maximise product purity. The purity of chemicals

used in Australia for the treatment of drinking water supplies will vary depending on the manufacturing process. Contaminants that may occur in specific treatment chemicals are outlined in the fact sheets (see Section V). The information in the fact sheets is based on the best data available at the time of publication. However, research and industry experience may lead to changes in manufacturing processes or better understanding of the properties of the chemicals, which in turn may lead to changes in procedures for how water treatment chemicals should be handled, stored and used.

8.6.2 MANAGING RISKS

A complete water quality management program needs to recognise any potential risks from use of drinking water treatment chemicals and include strategies to manage them appropriately. These risks should be minimised by the implementation of a quality assurance system for the management of production, supply, delivery and use of water treatment chemicals.

The first step in managing the risk associated with the use of drinking water treatment chemicals is to ensure that the chemicals supplied meet a minimum standard, as established by the relevant state or territory regulatory agency. For example, water authorities may formally specify the strength of active ingredient and acceptable contaminant levels in each drinking water treatment chemical (see Section 8.6.3). However, this in itself will not adequately control the risk. The contractual requirement should be supported by batch-testing data provided by the supplier from an independent NATA (National Association of Testing Authorities) accredited laboratory, and random testing carried out by the water authority itself. Chemicals should not be accepted for delivery unless a batch analysis certificate has been obtained and checked by the water authority.

Formal accreditation of the manufacturing facility by an independent accreditation agency (e.g. the International Organization for Standardization (ISO) or NSF International) provides a further level of risk management. Such accreditation should include random site visits to the manufacturing facilities by the relevant regulatory agency and, if warranted, the water authority.

Chemical suppliers should be evaluated and selected on their ability to supply products in accordance with required specifications. Documented procedures for the control of chemicals, including purchasing, verification, handling, storage and maintenance, should be established to assure the quality of the chemical at the point of application (see Section 3.10.1). Responsibilities for testing and quality assurance of chemicals (supplier, purchaser or both) should be clearly defined in purchase contracts.

An important step in a quality assurance system for chemical addition to drinking water is to ensure that the required chemical is of the specified quality, and specified strength, and is delivered into the correct storage vessel, at the correct site at the correct time. This is necessary to:

- ensure that the correct chemical at the required concentration is used in drinking water treatment;
- ensure that cross-contamination of storages does not occur;
- ensure inappropriate and unsafe mixing of chemicals does not occur;
- help to ensure the health and wellbeing of staff and contractors during the delivery and dosing process.

Broadly, the objective of the water treatment chemical quality assurance system is to manage all the factors associated with the specification, contract management, supply, storage, use and handling of water treatment chemicals that could have an adverse impact on the health and wellbeing of staff, contractors and consumers. Box 8.1 outlines the components that make up an effective quality assurance system for drinking water treatment chemicals.

Box 8.1 *Desirable components of a quality assurance system*

A quality assurance system for chemicals used in the production of drinking water might include:

- selection of chemical suppliers based on their ability to meet specified requirements for supply and delivery, monitoring and analytical testing of contaminants;
- selection of suppliers who have a quality management system that is certified by an independent accreditation agency;
- an appropriate monitoring program to ensure that chemicals comply with specifications;
- an audit process for the supplier's manufacturing, storage and delivery processes;
- a formal checklist for the dispatch and delivery process;
- a delivery driver induction system for each site, with each driver inducted onto each site, together with appropriate record-keeping procedures;
- the provision of details of the delivery site (site photographs may be useful);
- an identity check directly linking the delivery driver to the chemical company;
- the clear identification and labelling of chemical storage vessels, filling points and delivery pipe work at all sites (locks on filling points are desirable);
- a requirement that chemicals should only be delivered when an appropriate water authority staff member is present to check documentation, including batch analysis certification, and to ensure unloading to the correct storage vessel;
- a standard operating procedure for the delivery and receipt of chemicals at each delivery site, including a documented acceptance criteria system to assist site operations staff in assessing whether to accept or reject the delivery of a chemical;
- a gross visual check of the chemical and, where appropriate, simple physical testing by the water authority representative at the delivery site before unloading;
- a check by both parties that the delivery vessel is being connected to the correct storage vessel;
- a check that appropriate personal protective equipment is being worn, and that relevant health and safety requirements are being addressed;
- appropriate recording and storage of relevant documentation;
- a system to ensure that any spillage associated with the delivery process is contained and does not escape to the environment;
- an emergency procedure in the event of possible systems failure or human error.

The combination of a chemical quality assurance system and a delivery and storage quality assurance system such as those outlined in Box 8.1 can significantly reduce risks to all stakeholders. The combined system should include formal quality audits (see Section 3.11).

8.6.3 SPECIFICATIONS FOR THE SUPPLY OF DRINKING WATER TREATMENT CHEMICALS

Preparing specifications for a chemical supply contract can be a time-consuming and difficult task. Documents should be prepared in conjunction with a risk assessment and controls recommended in Sections 8.5.1 and 8.5.2.

To simplify the process for water authority staff preparing their own specifications, an example specification for the supply and delivery of liquid aluminium sulfate (Al_2SO_4) to a water authority is provided in Box 8.2.

The specification includes details on the required content of aluminium, which is often, but not always, expressed as equivalent aluminium oxide (Al_2O_3); product clarity; solids content; and pH, as well as specific impurity limits. The specification also details some delivery and acceptance criteria. Product strengths and basic characteristics of the chemicals can be obtained from the relevant fact sheets in Section V. The water authority may customise these specifications to suit their particular situations and risks.

The specification should also clearly define the arrangements and responsibilities for ensuring the treatment chemical is not contaminated during transport or storage prior to transport.

Box 8.2 *Example specification for the supply and delivery of liquid alum to a water authority*

Aluminium Sulfate (ALUM)– Specification reference

This specification is for the supply and delivery of liquid aluminium sulfate ($Al_2(SO_4)_3.14H_2O$) to [*Name of water authority*] sites. This specification is based on the NHMRC *Australian Drinking Water Guidelines* (2010), the American Water Works Association Standard for Aluminium sulfate – liquid, ground or lump (ANSI/AWWA B403-93) and the Water Chemicals Codex (NRC 1982).

Liquid aluminium sulfate is not currently listed as Dangerous Goods.

REQUIREMENTS

Material Safety Data Sheets (MSDS)

The successful tenderer must supply a current MSDS with a review date not exceeding five (5) years. The MSDS must, as a minimum, comply with the requirements of the National Occupational Health and Safety Council (NOHSC) MSDS Guidelines. Whilst the NOHSC-MSDS format is preferred, alternative formats exceeding the level of information required by NOHSC-MSDS Guidelines are acceptable.

Liquid aluminium sulfate clarity

Liquid aluminium sulfate shall be of such clarity as to permit the reading of flow measuring devices without difficulty.

Content of aluminium

The water soluble aluminium content of liquid aluminium sulfate is expected to be greater than or equal to 4.23% of Al, or to fall within the range of 7.5 to 8.0 % as $(Al_2(SO_4)_3$.

Suspended solids

In liquid aluminium sulfate, it is expected that the level of suspended solids is below 0.2%.

pH

The pH of liquid aluminium sulfate is expected to fall within the range of 2.3 to 2.8 pH units.

Specific impurity limits

It is expected that the total water-soluble iron (expressed as Fe_2O_3) content of liquid aluminium sulfate shall be no more than 0.35%.

The level of contamination of the liquid aluminium sulfate shall be such that compliance with the recommended maximum impurity content (RMIC) values from Table 8.4 in the NHMRC *Australian Drinking Water Guidelines* is achieved. The RMICs, in mg/kg, for $Al_2(SO_4)_3$ are:

Impurity	Dose: 20 mg/L	Dose: 60 mg/L	Dose: 120 mg/L
Arsenic	16.5	5.5	2.7
Cadmium	4.7	1.6	0.8
Chromium	117.5	39.2	19.6
Lead	23.5	7.8	3.9
Mercury	2.4	0.8	0.4
Selenium	23.5	7.8	3.9
Silver	235	78	39

VERIFICATION

Quality assurance

The supplier is expected to possess a Quality System that facilitates the tracking of product from raw material to delivery. [*Name of water authority*] may audit this Quality System to verify the correctness of information relating to the purchased product. In addition, [*name of water authority*] may sample the purchased product at the point of destination to verify the quality of the supplied product.

Liquid alum samples

If [*name of water authority*] elects to sample the product at the point of destination, the sampling procedure outlined in the American Water Works Association Standard for Aluminium Sulfate – Liquid, Ground, or Lump (ANSI/AWWA B403-93) will apply.

Nonconforming product

If [*name of water authority*] discovers that the aluminium sulfate delivered does not meet the requirements of this specification, a notice of nonconformance will be issued to the supplier through the [*name of water authority*] 's Quality System, within ten working days of the receipt of the goods.

A nonconformance will also be issued if deficiencies are detected during any audit of the supplier's Quality System.

DELIVERY

Liquid

Marking, packaging and shipping of aluminium sulfate shall comply with AS 3780:1994 *The Storage and handling of corrosive substances*, and current federal, state, territory, and local regulations.

The carrying vessel shall be in a suitable condition for hauling liquid aluminium sulfate and shall not contain any substances that might affect the use or usefulness of the liquid aluminium sulfate in treating potable water or in treating wastewater.

Contamination

Bulk or semi-bulk containers shall be carefully inspected prior to loading of the chemical by the supplier to ensure no contaminating material exists.

The supplier must have a system in place to ensure that liquid aluminium sulfate is not contaminated by any other product. This may involve implementing a specific cleaning regime between loads or the dedication of tankers or containers to only one type of product.

Certificate of weight

[*Name of water authority*] may require that weight certificates accompany bulk shipments from a certified weigher or [*name of water authority*] may check the weights on delivery.

Affidavit of compliance

[*Name of water authority*] requires an affidavit from the manufacturer or supplier that the aluminium sulfate furnished according to [*name of water authority*]'s order complies with all applicable requirements of this specification. [*name of water authority*] also requires that the supplier provide a certified analysis of the aluminium sulfate. [*name of water authority*] may also elect to use in-house analytical equipment to analyse the product to ensure compliance with this specification.

Documentation

A copy of the order, the delivery docket, and the affidavit of compliance and/or the record of certified analysis will accompany the delivery of aluminium sulfate. This documentation shall be left in an appropriate location at the delivery point.

Further, a copy of the delivery docket is to accompany the invoice (with references to the delivery docket number), and forwarded to [*name of water authority*]'s Accounts Department to facilitate timely payment of accounts.

8.7 Monitoring and analytical requirements

A quality-controlled system for management of drinking water treatment chemicals should be supported by appropriate testing and monitoring.

All chemicals used in water treatment should be tested, to check both the concentration of the active ingredients and the presence of contaminants relative to a specification. This is to ensure that the effectiveness of the treatment process, the quality of the water and the integrity of the assets are not compromised.

Requirements for testing by the manufacturer should be clearly defined in the specification, including testing methods. The amount, type of testing and whether NATA-certified results from an external laboratory are required may need to be negotiated to achieve a solution that is both effective and affordable. Clear statements as to the testing methods should be included in the specification. The specification should require test results to be available before the chemical delivery is unloaded at the water authority's plant, to allow operational staff on site to reject delivery if specified requirements are not met.

Various physical characteristics can also be examined as part of the quality assurance program. Table 8.3 lists simple suggested acceptance criteria for some water treatment chemicals that could be applied by operational staff on site at the treatment plant. These criteria rely on human senses or simple equipment.

Table 8.3 *Acceptance criteria for some water treatment chemicals*

Chemical	***Tests***	***Acceptance criteria***
Aluminium chlorohydrates	*Visual*	Clear, colourless liquid
	Specific gravity	1.32–1.35 at 25°C
	pH	3.5–4.5
Aluminium sulfate (alum)	*Visual*	Clear colourless to pale brown (free of solids)
	Specific gravity	1.28–1.34 at 20°C
	pH	2.3–2.8
Ammonia	*Visual*	Colourless gas or liquid
	Specific gravity	0.8 as a liquid
Ammonium sulfate	*Visual*	Off-white crystal
	Specific gravity	1.77 at 20°C
Calcium hydroxide (hydrate lime)	*Visual*	Soft, white crystalline powder
	Solubility	0.165g/100g of saturated solution at 20°C
	Bulk density	450–560 kg/m^3
Calcium hypochlorite	*Visual*	White crystalline solid, practically clear in water solution
	Specific gravity	2.35 in liquid
Calcium oxide (quick lime)	*Visual*	Grey-white solid (sometimes yellowish to brown)
	Specific gravity	3.2 – 3.4 as calcium hydroxide
	Bulk density	1030 kg/m^3 (pebble); 1050 kg/m^3 (powder)
Carbon, powder activated, granular activated *(PAC/GAC)*	*Visual*	Black solid (PAC 20-50 µm; GAC 0.7 – 1.2 mm)
	Density	250–600 kg/m^3
Copper sulfate	*Visual*	Blue crystal, crystalline granule or powder
Ferric chloride	*Visual*	Brownish-yellow or orange crystalline form
	Specific gravity	42% solution: 1.45 at 20°C
	pH	42% solution: 1–2
Ferric sulfates	*Visual*	Yellow crystal or greyish-white powder, or a red-brown liquid solution.
	Specific gravity	Liquid solution: 1.5–1.6
Hydrochloric acid	*Visual*	Clear colourless to clear yellow (free of solids)
	Specific gravity	28% solution: 1.14 at 20°C
Hydrofluorosilicic acid (fluorosilicic acid)	*Visual*	Colourless to pale yellow liquid
	Specific gravity	22% solution: 1.18 at 20°C
Hydrogen peroxid	*Visual*	Colourless syrupy liquid (concentrations from 20% to 60%)
	Specific gravity	1.07–1.24 at 20°C
	pH	1–4
Hydroxylated ferric sulfate	*Visual*	Translucent, dark red (free of solids)
	Specific gravity	1.45–1.6 at 25°C
	pH	< 2

Table 8.3 *Acceptance criteria for some water treatment chemicals*

Chemical	***Tests***	***Acceptance criteria***
Polyacrylamides	*Visual*	White crystalline solid, supplied as a powder or aqueous solution, dispersed in light mineral oil
Polyaluminium chlorides (10%)	*Visual*	Pale yellow, slightly cloudy liquid
	Specific gravity	1.18–1.22 at 20°C
	pH	10% solution: 2.2–2.8
Polyaluminium silica sulfates	*Visual*	Slightly cloudy liquid, clear to yellow (free of solids)
	Specific gravity	1.32–1.36 at 25°C
	pH	2.8–3.6
Potassium permanganate	*Visual*	Odourless, dark purple crystal with blue metallic sheen
Sodium aluminates	*Visual*	White powder, or clear colourless to pale amber liquid
	Specific gravity	Liquid solution: 1.4–1.6
	pH	Liquid solution: 14
Sodium bicarbonate	*Visual*	White powder or crystalline lumps, soluble in water (60 g/L at 20°C)
	Specific gravity	2.159 at 20°C
	Solubility	96 g/L at 20°C
	Bulk density	1000 kg/m3
	pH	10 g/L solution: 8.4
Sodium carbonate (soda ash)	*Visual*	Greyish-white powder
	Bulk density	1000 kg/m^3 (dense); 500 kg/m3 (light)
Sodium fluoride	*Visual*	White, odourless powder (or crystal), easily soluble in water
	Specific gravity	2.78 at 20°C
	Bulk density	1040 – 1440 kg/m^3
	pH	1% solution – 6.5 4% solution – 7.6
Sodium fluorosilicate	*Visual*	White or yellowish white, odourless, crystalline powder
	Bulk density	880 – 1150 kg/m^3
Sodium hexametaphosphate	*Visual*	White granular powder
	Bulk density	800–1500 kg/m^3
Sodium hydroxide (caustic soda)	*Visual*	White, deliquescent solid)
	Specific gravity	30% solution: 1.33 46% solution: 1.48
Sodium hypochlorite	*Visual*	Pale yellow green
Sodium silicate	*Visual*	Lumps of greenish glass, white powders of varying degrees of solubility, or cloudy or clear liquids of varying viscosity
Sodium tripolyphosphate	*Visual*	White powder or granular solid
	pH	9.8 (aqueous solution) to 10.5 (slurry)
Sulfuric acid	*Visual*	Dense, oily, colourless to dark brown liquid.
	Specific gravity	1.2–1.85 at 20°C
Zinc orthophosphate	*Visual*	Clear odourless liquid

8.8 Contaminants in drinking water treatment chemicals

All chemicals used in the treatment of drinking water should be evaluated for potential contaminants and limits should be included in the specification. The fact sheets for the individual treatment chemicals (see Part V) identify potential contaminants for each chemical. Additional information may also be available from suppliers' specifications or from certification analyses that have been performed for overseas accreditation systems.

The determination of contaminants in drinking water treatment chemicals should be carried out by an independent laboratory accredited to undertake the necessary assays. An appropriate NATA-approved laboratory should be identified, in consultation with the relevant state or territory regulatory authority. A list of NATA-approved laboratories is available online[4].

In developing appropriate specification limits for contaminants, a more detailed systematic assessment of potential contaminants using a Recommended Maximum Impurity Concentration (RMIC) approach is recommended. The initial approach uses the principle that no contaminant in a particular chemical should add more than 10% of that allowable by the ADWG health value. For each contaminant, this involves:

- calculating from the health guideline value the maximum concentration allowable in the treated water as a result of being dosed with the bulk chemical. In some situations a stricter value than the health guideline may be warranted if the contaminant is known to cause aesthetic problems or the water authority wishes to carry a lower risk level;
- based on the expected maximum dose of chemical and its strength, calculate the RMIC for each contaminant (mg/kg of solution).

A sample calculation for determining the RMIC of lead in Alum is provided in Box 8.3.

Box 8.3 *Sample calculation for determining the lead recommended maximum impurity concentration in Alum*

The following is a sample calculation for the derivation of a Recommended Maximum Impurity Concentration (RMIC) for lead in Alum and is based on the NHMRC guideline value for lead in drinking water of 0.01 mg/L. The maximum amount of lead (in mg/L) that may be added to drinking water through the use of alum is determined through the following three steps:

Derivation of the maximum amount of lead that can be added to drinking water through Alum:

$$\frac{0.01}{10} = 0.001 \ mg/L$$

Where:

- 0.01 mg is the NHMRC guideline value for lead; and
- 10 is the percentage of the guideline value considered an acceptable source of contamination in the drinking water (a safety factor of 10 is considered a reasonable contribution by a given impurity in a water treatment chemical).

(1) Derivation of the amount of Alum that will contain 0.001 mg lead:

$$\frac{80 \text{ m/L}}{0.43} = 186 \ mg$$

In the case of the maximum Alum dose of 80 mg/L(1), with a solution strength of 43 % w/w [$Al_2(SO_4)_3.14H^2O$];

Where:

- 80 mg/L is the dose of the drinking water treatment chemical (e.g. Alum); and
- 0.43 is the solution strength of the drinking water treatment chemical (e.g. Alum – 43%)

(2) Derivation of the RMIC for Alum at the plant:

$$\frac{1 \times 10exp6}{186 \text{ mg}} \times 0.001 \text{ mg/L} = 5.4 \ mg.lead/kg \ of \ Alum \ solution$$

Where:

- 1×10^6 is the number of milligrams in a kilogram;
- 186 mg is the amount of Alum solution that will contain 0.001 mg of lead
- 0.001mg/L is the maximum amount of lead per litre that can be added through the Alum dose

Footnote

(1) The dose of 80 mg/L alum is based on the water treatment plant being designed to regularly treat dirty water events under an enhanced coagulation mode. If the plant was designed to treat low turbidity water for particle removal only, the maximum alum dose may be as low as 10 mg/L which would give an RMIC of 43.2 mg/kg for lead at this plant.

4 http://www.nata.asn.au/go/facilities-andamp-labs

In some cases RMICs may need to be reduced based on concentrations of the impurity in source water and where multiple treatment chemicals are used to ensure that recommended health-related guideline values are not exceeded at consumer taps. RMICs calculated by the water authority should be used as the minimum basis for chemical specifications. Water authorities are encouraged to use tighter specification values where these can be easily achieved cost-effectively. These calculated RMICs should never be seen as a license to degrade the purity of the drinking water treatment chemical.

To assist water authorities in this process, Table 8.4 contains RMICs for selected contaminants that have NHMRC health guideline values. RMICs have been calculated for some of the more common treatment chemicals, typical maximum dose rates and chemical bulk concentrations. RMICs have not been determined for contaminants that are not identified in the fact sheet for an individual treatment chemical. Aluminium sulfate has been used to illustrate the principle of applying different maximum doses to determine RMIC.

Some treatment chemicals may also contain known contaminants for which there are only aesthetic NHMRC guideline values. RMICs approach can also be used to calculate these contaminants where appropriate.

Where there is no ADWG health value for an identified contaminant, water authorities may be able to determine a RMIC based on a review of overseas drinking water guidelines (e.g. WHO, USEPA, EEC, the Chemical CODEX). If no RMIC can be calculated from a recognised drinking water guideline value, then the principle of due diligence would encourage a water authority to maintain concentrations as low as practicable.

Where suppliers are unable to meet the RMIC, then the water authority should examine what levels of the contaminant are reaching consumers to determine if a higher concentration can be tolerated in the treatment chemical without significantly changing the risk of not meeting the ADWG value. This analysis should attempt to identify other significant sources of the contaminant, its variability over time, and all expected operational conditions. If a higher contaminant level in the bulk chemical is acceptable (i.e. contributes more than 10% of the guideline value), then water authorities should consider whether there is a need for additional controls specifically for that contaminant in the chemical specification, contractual procurement arrangements, treatment plant operations, and monitoring through to consumers' taps.

Table 8.4 *Recommended maximum impurity concentrations for selected drinking water treatment chemicals*

IMPURITY			Antimony	Arsenic	Barium	Cadmium	Chromium	Copper	Cyanide	Fluoride	Lead	Mercury	Nickel	Selenium	Silver
		NHMRC Health Guideline Value (mg/L)	0.003	0.01	0.7	0.002	0.05	2	0.08	1.5	0.01	0.001	0.02	0.01	0.1
Treatment Chemical*	Chemical Strength (%)	Example doses (mg/L)													
Aluminium chlorohydrate	23	100 (as Al_2O_3)	0.7	2.3	161	0.5	11.5	460		345	2.3	0.2	4.6	2.3	23
Aluminium sulfate (Alum)	47	20 (as $Al_2(SO_4)_3$)	7.1	23.6	1645	4.7	117.5	4700		3525	23.5	2.4	47	23.5	235
Aluminium sulfate (Alum)	47	60 (as $Al_2(SO_4)_3$)	2.4	7.9	548	1.6	39.2	1567		1175	7.8	0.8	15.7	7.8	78
Aluminium sulfate (Alum)	47	120 (as $Al_2(SO_4)_3$)	1.2	3.9	274	0.8	19.6	783		588	3.9	0.4	7.8	3.9	39
Calcium hydroxide	99	30 (as $Ca(OH)_2$)		33.0	2310	6.6	165			4950	33	3.3	66	33	330
Calcium hypochlorite	65	3 (as Cl_2)		217.0	15167	43.3	1083.3			32500	216.7	21.7	433.3	216.7	2167
Calcium oxide	10	500 (as CaO)		0.1	14	0.04	1			30	0.2	0.02	0.4	0.2	2
Chlorine	100	3 (as Cl_2)		333.0							333.3	33.3			
Copper sulfate	25.5	1 (as $CuSO_4.5H_2O$)		255.0							255		510		
Ferric chloride	42	120 (as $FeCl_3$)	1.1	3.6		0.7	17.5	700	28		3.5	0.4	7	3.5	35
Ferric sulfate	20	100 (as $Fe_2(SO4)_3$)	0.6	2.0		0.4	1	400	16		2	0.2	4	2	20
Hydrochloric acid	33	5 (as HCl)	19.8			13.2	330				66		132		
Hydrofluorosilicic acid	16	1.5 (as F)		107.0		21.3					106.7				
Hydroxylated ferric sulfate	12.5	100	0.4	1.3		0.3	6.3	250	10		1.3	0.1	2.5	1.3	13
Polyaluminium chloride	10	100 (as Al_2O_3)	0.3	1.0	70	0.2	5	200		150	1	0.1	2.0	1	10
Potassium permanganate	99	1 (as $KMnO_4$)				198	4950					99			
Sodium fluoride	45	1.5 (as F)	90			60					300				
Sodium Fluorosilicate	60	1.5 (as F)	120			80									
Sodium hydroxide	50	10 (as NaOH)	15			10	250				50	5	100		
Sodium hypochlorite	12	3 (as Cl_2)				8						4	80		
Sulfuric acid	98	5 (as $H2SO_4$)	58.8	196.0	13720	39.2	980	39200		29400	196	19.6		196	

* Table includes recommended maximum impurity concentrations (RMIC) for selected drinking water chemicals. Further information on determining RMIC can be found in Box 8.3.

8.9 Useful contacts

Australian Government

National Health and Medical Research Council
GPO Box 1421
Canberra ACT 2601

Tel: (02) 6289 9000 or 13 000 64672
E-mail: nhmrc@nhmrc.gov.au
Internet: www.nhmrc.gov.au

Safe Work Australia
GPO Box 641
Canberra ACT 2601

Tel: (02) 6121 5317
E-mail: info@swa.gov.au
Internet: http://safeworkaustralia.gov.au/

National Industrial Chemicals Notification and Assessment Scheme (NICNAS)
GPO Box 58
Sydney NSW 2001

Tel: (02) 8577 8800
E-mail: info@nicnas.gov.au
Internet: http://www.nicnas.gov.au

Office of Chemical Safety and Environmental Health
Office of Health Protection
Department of Health and Ageing
GPO Box 9848
Canberra ACT 2601

Tel: 1800 020 103 (freecall) or 02 6289 1555
Internet: http://www.health.gov.au/internet/main/publishing.nsf/Content/health-central.ht

Australian Capital Territory

Health Protection Services
Locked Bag 5005
Weston Creek ACT 2611

Tel: (02) 6205 1700, 13 2281
Internet: http://www.health.act.gov.au

Department of the Environment, Climate Change, Energy and Water
GPO Box 158
Canberra City ACT 26010

Tel: 13 22 81
E-mail: environment@act.gov.au
Internet: http://www.environment.act.gov.au/

ACT Workcover
GPO Box 158
Canberra City, ACT 2601

Tel: (02) 6207 3000
E-mail: workcover@act.gov.au
Internet: http://www.workcover.act.gov.au/

New South Wales

NSW Office of Water
Department of Primary Industries
Level 10, Macquarie Tower
Locked Bag 5123
Parramatta, NSW 2124

Tel: 1800 353 104
E-mail: water.enquiries@dpi.nsw.gov.au
Internet: http://www.water.nsw.gov.au/

Department of Environment and Heritage
PO Box A290
Sydney South NSW 1232

Tel: (02) 9995 5000
Email: info@environment.nsw.gov.au
Internet: http://www.environment.nsw.gov.au/index.htm

Workcover NSW
Locked Bag 2906,
Lisarow NSW 2252

Tel: 02 4321 5000
E-mail: contact@workcover.nsw.gov.au
Internet: http://www.workcover.nsw.gov.au/Pages/default.aspx

NSW Health Locked Mail Bag 961 North Sydney NSW 2059	Tel : (02) 9391 9939 E-mail: waterqual@doh.health.nsw.gov.au Internet: http://www.health.nsw.gov.au/

Northern Territory

Environmental Health, Department of Health PO Box 40596 Casuarina NT 0811	Tel: (08) 8922 7152 Internet: http://www.health.nt.gov.au/Environmental_Health/Water_Quality/index.aspx
NT Department of Land Resource Management PO Box 496 Palmerston NT 0831	Tel: (08) 8999 5511 Internet: http://www.lrm.nt.gov.au/
NT Worksafe GPO Box 3200 Darwin NT 0801	Tel: 1800 019 115 E-mail: ntworksafe@nt.gov.au Internet: http://www.worksafe.nt.gov.au/

Queensland

Environmental Health Branch Queensland Health Water Quality Unit PO Box 2368 Fortitude Valley BC QLD 4006	Tel: 07 3239 3931 E-mail: ehu@health.qld.gov.au Internet: http://www.health.qld.gov.au/ph/ehu/
Department of Environment and Heritage Protection GPO Box 2454 Brisbane QLD 4001	Tel: 13 74 68 E-mail: info@ehp.qld.gov.au Internet: http://www.ehp.qld.gov.au/water/guidelines/index.html
Workplace Health and Safety Queensland Office of Fair and Safe Work Queensland GPO Box 69 Brisbane QLD 4001	Tel: 1300 362 128 Internet: https://www.worksafe.qld.gov.au/

South Australia

Water Quality Unit SA Health PO Box 287 Rundle Mall ADELAIDE SA 5000	Tel: (08) 8226 7100 E-mail: waterquality@health.sa.gov.au Internet: http://www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/protecting+public+health/water+quality/providing+safe+drinking+water
Environment Protection Authority (SA) GPO Box 2607 Adelaide SA 5001	Tel: (08) 8204 2000 E-mail: epainfo@epa.sa.gov.au Internet: http://www.epa.sa.gov.au/
WorkCover SA GPO Box 2668 Adelaide SA 5001	Tel: 13 18 55 E-mail: info@workcover.com Internet: http://www.workcover.com/

Tasmania

Environmental Health Unit Department of Health and Human Services GPO Box 125 Hobart TAS 7001	Tel: 1800 671 738 E-mail: public.health@dhhs.tas.gov.au Internet: http://www.dhhs.tas.gov.au/peh/water
Department of Primary Industries, Parks, Water and Environment GPO Box 44 Hobart TAS 7001	Tel: 1300 368 550 E-mail: EnvironmentEnquiries@dpiwe.tas.gov.au Internet: http://www.dpiwe.tas.gov.au/
WorkSafe Tasmania PO Box 56 Rosny Park TAS 7018	Tel: (03) 6166 4600 or 1300 366 322 E-mail: wstinfo@justice.tas.gov.au Internet: http://worksafe.tas.gov.au/home

Victoria

Environmental Health Department of Human Services GPO Box 4057 Melbourne VIC 3001	Tel:1300 761 874 E-mail: water@health.vic.gov.au Internet: http://www.health.vic.gov.au/environment
Environment Protection Authority GPO Box 4395 Melbourne VIC 3001	Tel: 1300 372 842 E-mail: contact@epa.vic.gov.au Internet: http://www.epa.vic.gov.au/
WorkSafe Victoria Ground Floor 222 Exhibition Street Melbourne VIC 3000	Tel: 1800 136 089 E-mail: info@workcover.vic.gov.au Internet: http://www.vwa.vic.gov.au/home

Western Australia

Public Health and Clinical Services Division Department of Health PO Box 8172 Perth Business Centre Perth WA 6849	Tel: (08) 9222 4222 E-mail: webmaster@health.wa.gov.au Internet: http://www.public.health.wa.gov.au

National organisations

Australian Water Association (AWA) PO Box 222 St Leonards NSW 1590	Tel: (02) 9436 0055 or 1300 361 426 E-mail: info@awa.asn.au Internet: http://www.awa.asn.au
Water Research Australia PO Box 1751 Adelaide SA 5001	Tel: (08) 7424 2445 Internet: http://www.waterra.com.au/
National Association of Testing Authorities, Australia (NATA) 7 Leeds Street Rhodes NSW 2138	Tel: 1800 621 666 Email: corpcomm@nata.asn.au Internet: http://www.nata.asn.au/

Standards Australia Limited GPO Box 476 Sydney NSW 2001	Tel: (02) 9237 6000 E-mail: mail@standards.org.au Internet: http://www.standards.com.au/
Water Services Association Australia (WSAA) PO Box 13172 Law Courts Post Office Melbourne VIC 8010	Tel: (03) 8605 7666 E-mail: info@wsaa.asn.au Internet: http://www.wsaa.asn.au

International organisations

American Water Works Association (AWWA) 6666 W. Quincy Ave Denver, CO 80235 USA	Internet: http://www.awwa.org/
Codex Alimentarius Commission Viale delle Terme di Caracalla 00153 Rome, Italy	E-mail: Codex@fao.org Internet: www.codexalimentarius.net/
International Organization for Standardization (ISO) 1, ch. de la Voie-Creuse, Case postale 56 CH-1211 Geneva 20 Switzerland	E-mail: central@iso.org Internet: http://www.iso.org/iso/en/ISOOnline.frontpage
NSF International PO Box 130140 789 N. Dixboro Road Ann Arbor, MI 48113-0140, USA	Tel: (+ 1) 734-769-8010 E-mail: info@nsf.org Internet: www.nsf.org
World Health Organization Water, Sanitation and Health Programme Avenue Appia 20 1211 Geneva 27 Switzerland	Tel: (+ 41 22) 791 21 11 Internet: http://www.who.int/water_sanitation_health/en/

8.10 References and Bibliography

AWWA (American Water Works Association) and ASCE (American Association of Civil Engineers) (1997). *Water Treatment Plant Design*, 3rd edition. McGraw-Hill Professional, USA.

Clesceri LS, Greenberg AE, Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC

Faust SD, Aly OM (1998). *Chemistry of Water Treatment*, 2nd edition. Ann Arbor Press, Michigan.

IARC (International Agency for Research on Cancer) (1984). *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*. World Health Organization, Geneva.

IPCS (International Programme on Chemical Safety) (2000). *Environmental Health Criteria No 216. Disinfectants and Disinfectant By-products*. World Health Organization, ICPS, Geneva, pp 1–26, 370–380.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Rome, Italy.

KIWA (1994). *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01, The Netherlands.

Letterman RD (ed) (1999). *Water Quality and Treatment, A Handbook of Community Water Supplies*, American Water Works Association, 5th edition. McGraw-Hill Professional, New York.

National Registration Authority for Agricultural and Veterinary Chemicals (1996). *The Requirements Manual for Agricultural Chemicals*. National Registration Authority, Canberra.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC.

NWQMS (National Water Quality Management Strategy) (2000). *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*, NWQMS, Australian and New Zealand Environment and Conservation Council, Agriculture and Resource Management Council of Australia and New Zealand, Canberra.

PART III MONITORING

CHAPTER 9 Overview of monitoring (Revised 2011)

Chapter 9 Overview of monitoring (Revised 2011)

9.1 Introduction

The ***Framework for Drinking Water Quality Management*** (the Framework), outlined in Chapters 2-4, is based on a preventive strategy that encompasses total system management from catchment to consumer to assure safe drinking water.

A central aspect of this approach is the use of monitoring to confirm the effectiveness of the preventive measures and barriers to contamination, and to enhance understanding of system performance.

This is achieved through the collection of data that increase understanding of the entire water supply system, including the hazards and risks that are present, the performance of treatment barriers, and the integrity of the distribution system.

9.2 Monitoring overview

The Framework encourages a considered, overall strategy for monitoring that includes:

- operational monitoring in the source/catchment, through the treatment process, and in the distribution system, to ensure that processes and activities are functioning optimally to achieve safe drinking water;
- verification of drinking water quality, which consists of:
  - drinking water quality monitoring in the distribution system to verify the quality of treated water as supplied to the consumer; and
  - consumer satisfaction monitoring to assess consumer comments and complaints;
- ***investigative studies and research monitoring*** (including baseline monitoring where new water sources are going to be used to supply drinking water) to identify and characterise hazards, and increase understanding of a water supply system;
- ***validation monitoring*** of new operational processes and barriers, to assure effective operation and control; and
- ***incident and emergency response monitoring***, undertaken in response to incidents or emergencies.

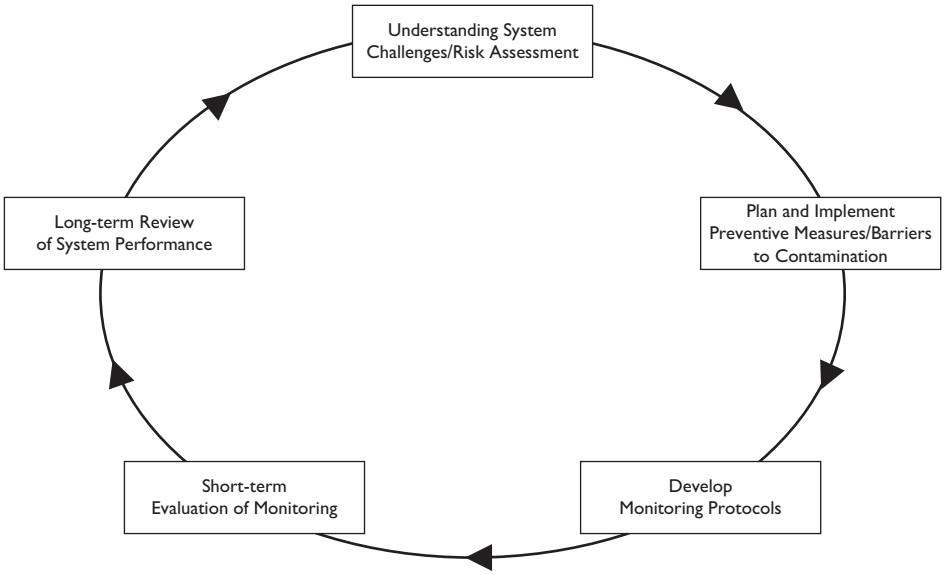
Each type of monitoring supports the others in the overall understanding and management of a water supply system, and in interpreting the monitoring data that are generated.

The overall goal of monitoring is to provide a high level of public health protection by minimising the risk of supplying contaminated drinking water. Water suppliers therefore need to ensure that monitoring attention and resources are directed to those aspects that provide the greatest assurance of drinking water quality.

Monitoring programs that focus primarily on the quality of treated drinking water do not effectively guarantee the supply of safe drinking water. Event-driven, intermittent contamination or system failures (characteristic of failures in the developed world) are very difficult to recognise and control through the monitoring of treated drinking water quality alone. In addition, the results of such monitoring do not become available until after the drinking water has been supplied to customers.

Developing a monitoring program is not a static activity, but part of an ongoing, iterative process of system management that seeks to understand the challenges and risks, plan and implement measures to prevent contamination (appropriate to the level of risk), monitor and assess the effectiveness of these barriers, plan improvements, and adjust preventive measures and monitoring programs as required. This approach is summarised in Figure 9.1.

Figure 9.1 *Monitoring as part of an ongoing process of system management*



The following sections of this chapter describe important principles and elements of a strategic monitoring system for assuring drinking water safety.

9.2.1 MONITORING PRIORITIES

While the potential contaminants of drinking water supplies are many, there is evidence of human health impacts via drinking water supplies for only a limited number of these. The most significant contaminants are waterborne microbial pathogens: they represent the clearest and most acute risk to drinking water safety, and they can cause outbreaks of illness that affect a high proportion of the community and, in extreme cases, result in death (see Box 3.3, Section 3.4). The single most important monitoring activity is therefore to ensure that microbial contamination does not cross barriers and enter the drinking water supply.

Chemical or radiological contamination does occur and, in some specific cases, serious health effects via drinking water have been documented. However, illness from such contamination typically arises from specific natural local conditions or from site-specific contamination by humans (distribution cross-connections, inadvertent chemical addition or sabotage). Priority chemicals (arising primarily from natural contamination) include arsenic, fluoride (above concentrations applied for dental protection), selenium, nitrate, lead and uranium. Iron and manganese are also mentioned as frequent sources of aesthetic water quality problems, and these may lead consumers to use alternative water supplies that may not be safe with respect to microbial pathogens (WHO 2008).

Chemicals used in water treatment may pose a risk because of the potential for inadvertent contamination, and they should be monitored accordingly. By-products of disinfection should also be monitored, because of the possible adverse health effects from chronic exposure to these chemicals. However, it remains uncertain whether exposure to disinfection by-products at the levels typically found in drinking water causes human disease, and given the established health risks associated with waterborne microbial pathogens, disinfection should never be compromised.

Most other chemicals, including pesticides and other trace organics, do not warrant the same level of monitoring attention as microbial pathogens or the chemicals of main concern, unless there is evidence or reasonable inference of their potential presence, as determined through site-specific investigation and analysis of the water supply system.

Box 9.1 summarises monitoring priorities based on health risk.

Box 9.1 *Monitoring priorities based on health risk*

Key characteristics related to health include:

- microbial indicator organisms and disinfectant residuals;
- any known characteristics that can be reasonably expected to exceed the guideline value, even if occasionally;
- any chemicals used in treatment processes and any by-products that may result from their use;
- any potential contaminants identified through the water supply system analysis (see Section 3.2.1) and hazard identification (see Section 3.2.3), even if undetected.

Some characteristics not related to health, such as those with significant aesthetic impacts, should also be monitored. Where aesthetic characteristics (e.g. taste and odour) are frequently unacceptable, further investigation may be needed to determine whether there are problems with significance for health.

Monitoring can be direct, where the characteristic of concern is monitored directly; or indirect, where surrogates or indicators are monitored.

Surrogates are typically quantifiable characteristics that can serve to measure the effectiveness of processes in controlling specific hazards or groups of hazards.

Indicators are physical, chemical or microbial characteristics that are representative of a broader group of related characteristics. Indicators provide an alternative to monitoring for the possible presence of other hazardous substances that are more difficult to monitor.

Effective surrogates and indicators:

- directly measure process performance characteristics that are related to the effectiveness of the process in preventing or eliminating hazards;
- are amenable to the setting of trigger levels, guideline values and/or target criteria, so that results can be responded to;
- provide warning of process performance failures early enough to allow corrective action to be taken before unsafe water is supplied to consumers; and
- are low cost and reliable to monitor, and where required, are amenable to on-line monitoring.

Some examples of surrogates and indicators are:

- electrical conductivity (EC), which is widely used as a surrogate for total dissolved solids;
- turbidity, a widely used surrogate for the performance of media filtration systems;
- trihalomethanes (THMs), which, because they are the most common disinfection by-products and occur in the highest concentrations, can be used as indicators for the possible presence of a range of related chlorine-derived disinfection by-products;
- faecal indicator bacteria, which are numerous in faeces and serve as indicators for the possible presence of faecal contamination and, by inference, enteric pathogens.

9.2.2 PRINCIPLES OF MONITORING FREQUENCY

The frequency of monitoring for each water quality characteristic differs for each characteristic, and depends on the hazard characteristics and risk profile as identified through analysis of the water supply system. In general, characteristics that pose a high level of risk require more monitoring, while those posing a low risk require less monitoring. Typically, the most frequent monitoring is for microbial safety, followed by known or identified high priority and site-specific contaminants, with less frequent monitoring for any contaminants that are not likely to present a risk.

Operational monitoring of preventive measures and barriers throughout the water supply system should be carried out with sufficient frequency to reveal any challenges or failures promptly, so that corrective actions can be taken. Online and continuous monitoring should be used wherever possible, particularly for essential processes identified as critical control points, such as disinfection and filtration (see Sections 3.3.2 and 9.4.3).

Frequency of source water monitoring is determined by the variability and understanding of the challenges present. Monitoring should be more frequent where water quality is more variable or less understood. Depending on the historical data available and the present understanding of source water characteristics, a baseline investigation of contaminants in a water supply may be required to assess hazards and their risk levels.

Frequency of monitoring downstream of treatment depends on how effectively treatment barriers are controlled and the level of understanding of the distribution system. Identified problem areas may dictate increased monitoring frequencies (e.g. where there is difficulty in maintaining chlorine residuals).

Disease outbreaks associated with drinking water supplies are often linked to unusual events. Such events should therefore be recognised as potential triggers for increased challenges and potential sub-optimal performance, and should alert water managers to the potential for problems and the need for increased monitoring of performance throughout the system (Box 9.2). Unusual events include any sudden or extreme change in weather, flow or water quality, as well as power outages, new assets, treatment variations, and maintenance and repairs. The increased monitoring frequency should be maintained until there is confidence that water quality is back within specification.

More detailed discussion on specific sampling frequencies within the water supply system is provided in Section 9.4.5.

***Box 9.2** Contamination events*

Waterborne disease outbreaks associated with drinking water supplies are almost invariably linked to a significant change in conditions that provides a sudden challenge to a water system; for example, heavy rainfall or run-off from heavy snow melt (Craun *et al*. 2003, Curriero *et al*. 2001, Hrudey and Hrudey 2004). Most water treatment processes function best under steady-state conditions, and performance can seriously deteriorate with major fluctuations in water quality or flow. Run-off from heavy rainfall or snow melt can dramatically increase flow and turbidity as well as the concentration of natural organic matter (Logsdon *et al*. 2004, Hunter 2003). Run-off can also potentially alter the pH and alkalinity of source waters (Logsdon *et al*. 2004). These fluctuations in flow or quality cause additional stress on water treatment systems and can interfere with the effectiveness of water treatment. The failure of treatment processes to cope with the impacts of heavy rainfall and run-off events is a common theme to many waterborne disease outbreaks.

In addition to changing environmental conditions and their impacts on source water, any changes from the normal operation of a water treatment system can also pose a significant risk of waterborne disease outbreaks. Reviews of outbreaks related to drinking water supplies have frequently identified increased contamination during periods of maintenance of the water treatment plant and storage facilities, plant upgrades, and changes to water treatment processes, as well as during construction or repair of water mains (Hrudey and Hrudey 2004 pp 83-94, Logsdon *et al*. 1996, Kramer *et al*. 1996, Craun *et al*. 2003, Nygard *et al*. 2007).

Furthermore, reported waterborne disease outbreaks frequently reveal that systems are at most risk when a combination of risk factors coincide; for example, heavy rainfall during plant maintenance and repairs, increased demand for water, and inadequate treatment performance, coupled with old facilities. There are a number of instances where outbreaks have occurred after problems had been revealed but no corrective action had been taken.

The risk of contamination of water supplies with microbial pathogens is always present. While safeguards and multiple barriers may be in place, the historical absence of waterborne outbreaks in a water system is no guarantee that one will not occur in the future unless the effectiveness of the barriers is continuously maintained and verified. Constant vigilance and effective monitoring programs that support understanding of a water supply system – its challenges and capabilities – are of paramount importance in assuring the safety of drinking water.

9.2.3 CATCHMENT-TO-CONSUMER MONITORING

Assuring the safety of drinking water requires an effective monitoring strategy that is preventive rather than reactive, and that aims to promote an understanding of the entire water supply system. An integrated approach to monitoring incorporates all aspects of the water supply system, including catchment and source water, treatment processes, the distribution system and consumers, to provide key information on system management and operation.

Source water

Effective system management requires knowledge of the source water (be it surface, ground or sea water) and the characteristics of the associated catchment area. Source water monitoring assists a water supplier in understanding what hazards are possible and the contamination challenge (i.e. the level of risk) they present.

Source water monitoring can also be preventive, in that it can be developed to provide an opportunity for real-time process control and the prediction of potential contamination; for example, concentration spikes of microbial pathogens associated with storm or heavy rain events. Early warning monitoring should be established to capture any changes from normal baseline levels, which in turn would trigger an appropriate response; for example, closing intakes or using alternative sources if available, changing treatment practices, increasing vigilance of system operation within specifications, and increased monitoring throughout the system (Wu 2004, Gullick ***et al***. 2003).

Where a new drinking water source is to be brought on line (either adding to an existing water supply system, or as part of a new one), a range of monitoring and other background investigations are needed to inform hazard identification and risk assessment for the supply system. Monitoring requirements will be influenced by the characteristics of the water source and catchment. Types of monitoring to be considered include:

- microbiological monitoring based on potential sources of faecal contamination (e.g. sewage and septic waste, livestock);
- microbiological and chemical monitoring to assess intermittent or seasonal pollution patterns;
- chemical monitoring based on identified agricultural, mining, industrial and urban pollution sources;
- chemical monitoring based on geological features (particularly for groundwater sources);
- identification of existing land uses and planned developments.

Further information on the monitoring considerations for source waters can be found in the Appendix to this document; in ***Australian Guidelines for Water Quality Monitoring and Reporting*** (ANZECC and AMRCANZ 2000); in the Cooperative Research Centre for Water Quality and Treatment (CRCWQT) reports 11, ***A Guide to Hazard Identification and Risk Assessment for Drinking Water Supplies*** (2004), and 37, ***Strategic Water Quality Monitoring for Drinking Water Safety*** (2007); and in the CRCWQT and Water Quality Research Australia report 78, ***Risk Assessment for Drinking Water Sources*** (2009).

Water treatment plant

Monitoring of treatment processes and barriers is fundamental to a preventive strategy for drinking water safety. The advantage of monitoring treatment performance is that, if set up correctly, ineffective treatment processes (e.g. inadequate disinfection, shortening of filter runs, degraded filtered water quality) can be identified and acted upon in close to real-time, to prevent potentially contaminated water from reaching consumers. To help ensure that unsafe water is not delivered to consumers, monitoring results need to be promptly evaluated and reported and, where appropriate, corrective actions need to be implemented immediately.

Box 9.3 provides an example of a situation where failure to respond to changes in treatment plant performance contributed to an outbreak of waterborne disease.

If the source water challenge and water treatment capabilities are understood, if attention is focused on understanding treatment performance, and if performance is monitored continuously, this provides a high level of assurance of drinking water safety.

***Box 9.3** Importance of monitoring treatment performance*

In North Battleford, Canada, in 2001, poor treatment performance was observed for a prolonged period after maintenance work on the solids contact unit (SCU). When the SCU was brought back on line, it was achieving minimal clarification due to difficulties in re-establishing an effective floc blanket. This, along with filtration that was not optimised, resulted in treated water turbidities being much higher than usual. Given the poor treatment performance and the known vulnerability of the river water source to contamination by *Cryptosporidium parvum*, operational staff should have recognised the seriousness of the risk and informed public health officials. Although regulatory requirements were not exceeded, if the normal range of raw water turbidity and the implications of poor turbidity removal been properly understood, appropriate actions could have been taken. As it was, this poor performance continued for several weeks and a breakthrough of *C. parvum* occurred, ultimately affecting between 5800 and 7100 people (Laing 2002).

Distribution system

Good design, management and integrity of distribution systems are essential for maintaining water quality. Monitoring programs should consider the potential for stagnation and ingress of contamination through faults in the distribution system. Stagnation and growth of biofilms can occur in poorly designed and operated distribution systems, while ingress of contamination can occur through tanks, reservoirs and pipes, cross-connections to the pipe network, and poor control of repairs or installation of new mains. Monitoring the integrity of the distribution system, and the quality of water supplied to consumers, is necessary to confirm that drinking water quality is maintained.

Consumers

Monitoring consumer satisfaction is another important surveillance mechanism. Consumers are located throughout distribution systems and their feedback can be directly related to the quality of drinking water supplied. They can provide timely information on potential problems, particularly within the distribution system, that may otherwise go unidentified.

9.3 Developing a monitoring program

Monitoring is an integral component of risk management. Because it is not possible to monitor for all things at all times, the monitoring program for a particular water supply system must be structured so that it enhances system knowledge and feeds into decision-making processes. This requires a considered approach to designing the program, the data it will generate, and how these data will be used.

Effective water quality monitoring requires the systematic collection of physical, chemical, biological, and observational information, and the analysis, interpretation and reporting of those data, all according to a pre-planned design (ANZECC and ARMCANZ 2000).

The monitoring program should be designed by personnel who understand the water supply system, the assessment of water quality, and the preventive management approach detailed in the Guidelines. The program may be developed in consultation with water supply system operators, planners and health regulators, or authorities responsible for auditing the performance of the drinking water supply system.

A monitoring program develops out of, and is based upon, system analysis and the risk assessment process. Once the hazards and key characteristics are identified and the preventive measures and barriers assessed, monitoring can be designed to provide the information needed for the effective management and operation of the drinking water system.

The monitoring program should address four broad questions:

- What are the hazards and risks of concern, what are the sources and what data exists? (i.e. investigative studies and research, including baseline monitoring)
- Are the barriers sufficient to manage the hazards and risks? (i.e. validation monitoring)
- Are the preventive strategies working now? (i.e. operational monitoring)
- Did the preventive strategies work? (i.e. verification of drinking water quality)

Once the objective and purpose of a monitoring activity is defined, the following questions could be used to determine the specifics:

- What data can be collected to provide the needed information?
- Is this the most effective way to generate this information? What alternatives are available for achieving the desired objective?
- How will the data be collected?
- Where will the data be collected?
- When will the data be collected?
- What will be done with the information? How will the data be used?
- How will the data be interpreted and evaluated?
- How will the data be responded to, and who should be notified?

Analysis of this type will help to generate data that are meaningful and useful. Each monitoring activity is done for a purpose, with every piece of monitoring datum a precursor for action. Monitoring activities relate not only to the collection of samples for laboratory analysis, but also to observations, field measurements, and monitoring using on-line instrumentation. All monitoring activities, and their bases, need to be documented into a comprehensive monitoring program that supports an integrated and comprehensive understanding of the water supply system, including the rationale for the monitoring decisions.

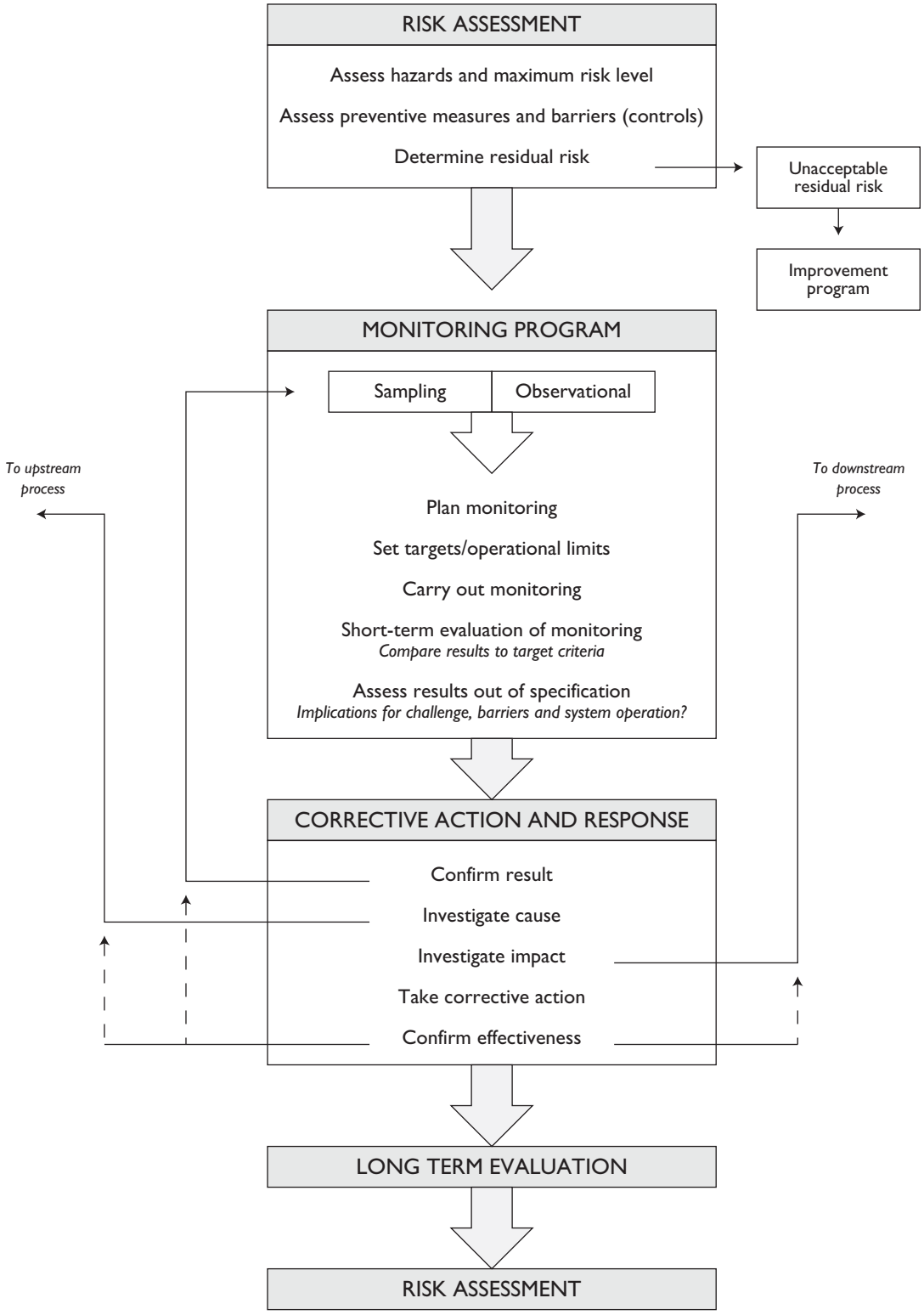
Figure 9.2 illustrates the development of a monitoring program that is based on a system-specific water supply analysis and risk assessment process to identify the hazards or characteristics, and the associated preventive barriers that require most attention and focus.

All monitoring data should be subject to short-term evaluation. In addition, monitoring data collected over the long-term should also be reviewed periodically and linked back into the system analysis and risk assessment. The aim is to assess whether there have been any significant changes to key characteristics or levels of challenge that warrant changes to system management, including the monitoring strategy.

Further information on developing monitoring programs is covered elsewhere in the National Water Quality Management Strategy. The ***Australian Guidelines for Water Quality Monitoring and Reporting*** (ANZECC and AMRCANZ, 2000) provide a comprehensive framework for monitoring and reporting, and include guidance on establishing water quality monitoring programs, including setting program objectives, study design, field sampling, analyses, data analysis, and reporting and information dissemination.

Additional guidance on water quality monitoring can be found in the Cooperative Research Centre for Water Quality and Treatment (CRCWQT) reports 11, ***A Guide to Hazard Identification & Risk Assessment for Drinking Water Supplies*** (2004) and 37, ***Strategic Water Quality Monitoring for Drinking Water Safety*** (2007), and the CRCWQT and Water Quality Research Australia (WQRA) report 78, ***Risk Assessment for Drinking Water Sources*** (2009).

Figure 9.2 *Generic process for monitoring activities*



9.4 Operational monitoring

Preventive measures and barriers to contamination should be applied from catchment to consumer in accordance with the multiple barrier approach, and these measures and barriers should be regularly monitored to assure their ongoing effectiveness.

Operational monitoring includes a planned sequence of measurements and observation throughout the water supply system to ensure and confirm performance of preventive measures and barriers to contamination. **The importance of operational monitoring to the effective maintenance of preventive barriers to contamination cannot be overstated**. To be effective, operational monitoring is needed at those points within the water supply system, including critical control points (see Box 9.4), such that if an adverse result is obtained, corrective action can be triggered to ensure that unsafe water does not reach the consumer.

Developing a protocol for monitoring operational performance of a water system requires the following steps:

- Identify preventive measures (see Element 3 of the Framework, Section 3.3).
- Select operational characteristics and associated operational criteria (objectives) to be used to assess the operational process or activity.
- Establish corrective actions to address any excursions in operational characteristics from defined criteria/objectives.
- Include frequent, routine monitoring of operational characteristics and ongoing analysis of monitoring results.

***Box 9.4** Critical control points*

A critical control point (CCP) is defined as an activity, procedure or process at which control can be applied, and that is essential to prevent a hazard or reduce it to an acceptable level.

For each CCP, a critical limit for operational performance needs to be established that represents a complete loss of control of the process and the existence of an unacceptable health risk. Failure to meet a critical limit should result in immediate notification of the health authority.

To be effective, and to provide assurance that unsafe water is not delivered to consumers:

- Those characteristics that are monitored at CCPs should be monitored continuously, using on-line analysers wherever possible.
- The monitoring should be alarmed so that operational staff are alerted promptly of adverse results.

Wherever possible, data should be sent to a supervisory control and data acquisition (SCADA) system (or some equivalent data capture system), so that trends over time can be evaluated and acted upon.

9.4.1 OPERATIONAL CHARACTERISTICS

The characteristics selected for operational monitoring should provide useful information concerning operational activities and performance. It is common, particularly in monitoring the operation of treatment processes, to use surrogates or indicators for water quality characteristics when direct testing is difficult, time-consuming or expensive.

Table 9.1 provides examples of characteristics commonly used for operational monitoring, by location from catchment to consumer.

Table 9.1 *Examples of operational monitoring characteristics*

Operational monitoring characteristics#	Stages of Water Supply and Treatment Processes					
	Source water	**Coagulation / Flocculation**	**Clarification (Sedimentation/ Flotation)**	**Filtration (Media/Membrane)**	**Disinfection (Chlorine/UV) Post Chemical Dosing**	**Distribution system**
pH	✓	✓			✓	✓
Turbidity (or particle count)	✓		✓	✓	✓	✓
Temperature#	✓		✓	✓		✓
Dissolved oxygen	✓					
Stream or river flow#	✓					
Rainfall#	✓	✓	✓		✓	
Escherichia coli (E. coli)	✓				*	*
Alternative microbial faecal indicators	✓					
Total coliforms					✓	✓
Heterotrophic plate count (HPC)					✓	✓
Colour	✓		✓		✓	
Conductivity (total dissolved solids)	✓					
Alkalinity#	✓	✓			✓	
Organic carbon#	✓					
Algae, algal toxins and metabolites	✓				✓	
Chemical dosage#		✓			✓	
Flow rate#		✓			✓	
Net charge#		✓				
Streaming current value#		✓				
Headloss#				✓		
C.*t*^					✓	
UV calculated dose#					✓	
UV intensity#					✓	
UV transmissivity#					✓	
UV lamp age#					✓	
Disinfectant residual#					✓	✓
Flux#				✓	✓	
Transmembrane pressure (TMP)#				✓		
Membrane integrity test (pressure based test)#				✓		
Hydraulic pressure#						✓
Tank integrity#						✓

Many of the characteristics list in this Table do not have Fact Sheets, or guideline values, but relate directly to the operation of water treatment processes which are beyond the scope of the current Guidelines to describe. Many of these characteristics will be supply-system specific, and not amenable to the setting of guideline values. Further information on these characteristics should be sought from water treatment experts

^ C.*t* = a measure of free chlorine residual concentration (C) and contact time (*t*)

* Monitoring for *E. coli* is typically undertaken at these locations as part of a drinking water quality monitoring program, but may be used to inform operational decisions.

In addition to field measurements, grab sampling for laboratory analysis, and online instrumentation, observational monitoring can also provide information on system challenge and barrier performance. Routine observational monitoring should be in place from catchment to consumer to identify and confirm, for example:

- the general level of activity in the catchment and/or reservoir, any illegal activities and sources of contamination, and the effectiveness of preventive measures such as gates, fences and signs;
- the security of the water treatment plant and chlorination facilities;
- that the chemicals used in water treatment are appropriate (see Chapter 8);
- the integrity of dosing and ancillary equipment;
- the performance of treatment processes such as effective floc formation, bubbling in granular filters, membrane integrity;
- the integrity of service tanks or reservoirs and the pipe network; and
- that routine preventive maintenance is undertaken throughout the system.

9.4.2 TARGET CRITERIA

Target criteria (operational objectives) should be set for each operational characteristic included in the monitoring program. The target criteria, which reflect the effectiveness of each process or activity, can be quantitative (numerical), for example, having a turbidity target for post-filter water, or qualitative (descriptive), as in setting acceptable levels of human activity in a catchment or reservoir.

Operational monitoring results are compared with these target criteria to assess if anything unusual is occurring within the water supply system. Any deviation from established targets should be regarded as a trend towards a potential loss of control over the system, and should result in appropriate investigations or corrective actions to ensure control is maintained and/or mitigate potential problems.

For some operational monitoring, particularly that undertaken upstream of treatment, target criteria and objectives for characteristics are often based on a more subjective assessment of what is considered normal or acceptable in the light of the water system analysis / risk assessment process (e.g. ***E. coli*** numbers in feeder streams and raw water). Monitoring of these characteristics provides an indication of changes from background levels that may influence the reliability of the original estimated challenge and consequently, the adequacy of subsequent downstream processes. The long-term review of monitoring results provides an opportunity to validate these criteria, and adjust preventive measures and associated criteria as required.

9.4.3 CRITICAL LIMITS AT CRITICAL CONTROL POINTS

For preventive measures identified as the critical control points for the water supply system, critical limits must also be defined and validated. A critical limit is a prescribed tolerance that distinguishes acceptable performance from unacceptable performance at a critical control point in terms of hazard removal or attenuation. Critical limits often incorporate both a numerical limit and a delay period, to ensure that alarms are not activated inappropriately (Mosse 2009). The delay period should be appropriate to the system: if the delay period is too short, repeated false alarms could lead to alarms being ignored or inactivated; if it is too long, a significant quantity of potentially unsafe water could be supplied to consumers before an alarm is activated.

Critical limits and the delay period are system-specific and determined through balancing the capability of the instrumentation and equipment used in the alarm system with the risk characteristics of the water supply system (e.g. whether treated water is supplied direct to consumers, or a storage exists before consumers receive the water).

Breaching a critical limit represents loss of control of the process and the existence of a health risk, either directly through the supply of unsafe water, or indirectly, where multiple critical control points exist, by exceeding the capacity of subsequent processes. Such events should result in immediate corrective actions to re-establish operations within specification, and notification of the health regulator.

Setting target criteria that are more stringent than the critical limits at critical control points will enable corrective actions to be instituted before an unacceptable health risk occurs. Exceeding a target criterion at a critical control point would generally not require that the health regulator be notified, providing corrective action successfully prevents deviation from a critical limit.

Box 9.5 provides an example of setting operational requirements for filtration as a critical control point.

***Box 9.5** Target criteria and critical limits for filtration*

Where drinking water is sourced from multi-use surface water with risk of contamination by ***Cryptosporidium***, filtration is often the primary barrier to these chlorine-resistant protozoan pathogens. It is critical that filter performance be optimised and continuously monitored to ensure that the required pathogen removal is achieved and safe drinking water provided at all times.

Whilst not a perfect measure of performance, continuous monitoring of filtered water turbidity is currently the best practical surrogate for assessing filter performance. It is strongly recommended that continuous on-line turbidity meters be installed on the outlet of ***each*** individual filter, as monitoring only at the combined filter outlet may fail to detect poor performance of an individual filter (Mosse & Murray 2008).

With filtration defined as a critical control point, a **critical limit** is set to define unacceptable performance contributing to a significant health risk (e.g. 0.5 NTU). Measured turbidities above this limit indicate loss of control of the process and compromised pathogen removal. To ensure critical limits are not breached, **target criteria** should also be established. A target criterion for filtration may be to achieve <0.2 NTU. To avoid unnecessary alarms, target criteria should incorporate a delay period in which the criteria are continuously breached before an alarm is generated (Mosse 2009).

9.4.4 CORRECTIVE ACTION

Where monitoring results indicate a deviation from target criteria or critical limits, appropriate corrective actions and process adjustments should be instituted to maintain water quality. Examples of corrective actions are:

- repairing fences;
- removing dead animals from catchment areas;
- increasing catchment controls;
- inspecting the water supply system for faults;
- altering the flow rate of the water treatment plant to reduce loading;
- manual backwashing of filters;
- adjusting process control;
- inspecting and calibrating monitoring equipment;
- engaging back-up equipment;
- adjusting pH;
- selecting an alternative raw water source, if available;
- increasing disinfection dose;
- booster disinfection;
- flushing of mains; or
- increasing monitoring and observation.

Additional monitoring will be required throughout the system to verify the effectiveness of any corrective actions.

9.4.5 OPERATIONAL MONITORING FREQUENCY

Operational characteristics throughout the system should be monitored often enough to reveal any failures and trigger a response within a timeframe that is appropriate to how critical the monitored activity or process is. This applies to both measurements and observational monitoring. Online and continuous monitoring should be used wherever possible, particularly at critical control points. For operational characteristics that are deemed less critical or are more stable, grab samples or regular inspections may be sufficient.

Frequency of observations may be increased at times of increased risk; for example, inspections of reservoirs for algal blooms may be more frequent during summer, or floc blanket observations during the coagulation process may be increased when there are higher flow rates through the treatment plant.

Operational monitoring requirements and frequency of monitoring will vary for each water supply, depending on the key characteristics identified through analysis of the water supply system and risk assessment. Table 9.2 provides an example of how selected operational characteristics can be used within a catchment-to-consumer operational monitoring program.

Table 9.2 *Example of an operational monitoring program (characteristics and frequencies)*

Location	Characteristic	Monitoring frequency	Rationale
Catchment			
General catchment	*Rainfall*	Daily	Understand impact of rainfall on water quality – to help predict challenge under range of rainfall intensity.
	Inspection	Monthly → Daily Frequency depends on level of access and use permitted in catchment.	Detect human and animal activities that could cause contamination; confirm that fences and signs are effective.
Feeder streams in catchment	*Turbidity, colour, E. coli*	Monthly plus events	Early warning of changes to raw water quality to allow timely changes to treatment processes. Detect local contamination and disturbances.
	Cryptosporidium	Risk-based	Assess if treatment barriers are needed to effectively remove or inactivate *cryptosporidium*.
Source Water			
Storage dam or raw water reservoir	*Temperature Profile* *Dissolved oxygen profile*	Monthly → Weekly	Information for management of water quality in storage with existing or new management systems.
	General water quality profile	Weekly → Event based	Allow best quality water to be selected for supply.
	Inspection	Weekly	Detect human and animal activities that could cause contamination.
	Algal cell counts	Monthly → Daily Risk-based	Early warning to activate management actions to prevent algal blooms in storage and forewarning of need for additional monitoring, observational surveillance. Information for changes to water treatment processes in order to maintain effective removal of algae and algal metabolites.
	Cryptosporidium	Risk-based	Information for changes to water treatment processes in order to maintain optimal *Cryptosporidium* removal.

Location	Characteristic	Monitoring frequency	Rationale
	Turbidity *Colour*	Continuous Weekly → Event based	Information for changes to water treatment processes in order to maintain optimal turbidity and colour removal.
River intake	*Rainfall*	Daily	Understand impact of rainfall on water quality to help predict challenge under range of rainfall intensity.
	Inspection	Weekly → Daily	Detect sources of contamination and activities that could cause contamination.
	Turbidity *Colour*	Continuous Weekly → Event based	Inform changes to water treatment processes in order to maintain optimal turbidity and colour removal.
	Iron, Manganese	Weekly (risk-based)	Inform changes to water treatment processes in order to maintain optimal iron and manganese removal; forewarn of water quality that may cause customer complaints.
	Algal cell counts	Monthly → Weekly (risk-based)	Forewarn of possible algal risk and need for additional monitoring, observational surveillance.
	Cryptosporidium	Risk-based	Inform changes to water treatment processes in order to maintain optimal *Cryptosporidium* removal.
	Turbidity, *E. coli* *Cryptosporidium* Pesticides Colour	(Rainfall-related monitoring) Risk-based	Understand rainfall effects Identify high challenge periods and forewarn downstream processes; identify local point source of contamination. Intervene in catchment before reservoir affected. Feedback to industry and source of contamination
Treatment Processes			
Coagulation *(inlet to flocculation tank)*	pH	Daily → Continuous	Optimise pH for effective coagulation of selected coagulants when raw water quality changes. Provide alarm if pH is outside set limits.
Flocculation *(last compartment)*	Floc characteristics	Daily → Event based	Optimise floc characteristics for effective clarification or filtration when changes occur to raw water quality or operating conditions.
Clarifier *(clarified water outlet)*	Turbidity Colour Visual observation of floc or floc blankets	Daily → Continuous Daily → Event based Daily → Event based	Confirm coagulant dose, pH correction, flocculation and clarifier operations are optimised when changes occur to raw water quality or operating conditions. Provide alarm if turbidity is above set limit. Assess if adjustment needed to process to improve stability of clarification process.
Filtration *(Individual or combined filtered water)*	Turbidity Filter Headloss	Continuous Continuous	Provide alarm if filtrate turbidity is above set maximum. Trigger for initiating filter cleaning. Trigger for initiating filter cleaning to avoid turbidity breakthrough.

Location	Characteristic	Monitoring frequency	Rationale
Filtration *(Combined filtered water post pH correction)*	pH	Continuous	Confirm target pH range is maintained. Provide alarm if pH is outside target limits for effective disinfection and corrosion control.
	Aluminium (If aluminium-based coagulant used)	Weekly	Assess inadvertent carry-over of aluminium from sub-optimal flocculation pH.
Chlorine Disinfection *(Chlorine >30 minutes after chlorine injection)*	Free chlorine residual	Continuous	Provide alarm if chlorine residual is outside set limits for maintaining integrity of water quality during reticulation and for reticulation hygiene.
UV Disinfection	UV dose rate	Continuous	Confirm UV system is operating satisfactorily. Provide alarm if below minimum set dose.
Fluoridation *(Downstream of fluoride dosing)*	Fluoride	Daily Continuous (if provided)	Confirm target dose is maintained. Provide alarm if fluoride is outside set limits.
Distribution System			
Disinfection *(At various locations in the reticulation system selected by careful monitoring design)*	Chlorine residual	Continuous → Daily	Confirm total chlorine target or free chlorine residual target range are achieved.
Service Reservoirs and tank	Integrity from contamination	1 to 5 yearly	Confirm roof/hatches are effective against ingress of contaminants.
Consumers	Customer complaints	Ongoing	Clusters of complaints of turbidity, objectionable taste and odour, illness allow investigation to identify cause(s) of water quality problems.

9.4.6 CHLORINATION AS A CRITICAL CONTROL POINT: AN EXAMPLE

Chlorination is the most commonly used process for disinfection and is highly amenable as a critical control point. Table 9.3 outlines, as an example, the operational requirements relevant to the chlorination process as a critical control point. The requirements include monitoring the key operational characteristics of residual chlorine concentration, flow rate (contact time) and chlorine dose; and establishing critical limits and target criteria for effective operation. Turbidity, temperature and pH also require monitoring, as they influence chlorination effectiveness and the validity of C.t calculations. Corrective actions to address deviations in operating limits must be identified, and chlorination performance should be regularly verified.

Appendix A1.8 provides additional information on chlorination as a critical control point.

Table 9.3 *Example of chlorination as a critical control point*

Hazards

Enteric bacteria, viruses and Giardia

Process controls

- Chlorine dosing system
- Plant flow rate/operation of clear well storage
- pH adjustment
- Chlorine cylinder changeover
- Backup power/duplicate facilities

Operational monitoring

Characteristic	*Target criteria*	*Critical limits*	*Monitoring methods*
Chlorine residual	> 0.5 mg/L	Specific low chlorine residual set to achieve a minimum C.*t* requirement based on maximum flow and minimum storage times. Time is an important factor in determining the critical limit. e.g. if there is a filtered water storage prior to supply to customers, an interruption to chlorination of up to several hours may not result in the C.*t* value falling below the minimum limit.	On-line, continuous chlorine residual analyser, flow and pH. 24-hour monitored alarms on residual monitoring, pH and chlorine dosing equipment. Regular turbidity and temperature monitoring, and chlorine demand calculations. Increase frequency on changing water quality Appropriate electronic or hard copy monitoring records.
pH	pH 6.5–7.5		
Flow rate	Set to achieve minimum contact time		
Chlorine dose	Set points ± x%		
Turbidity	< 1.0 NTU		
Temperature			

Corrective action

Any breach in critical limits or target criteria should result in any of the following operating procedures as necessary:

- inspect and calibrate equipment
- adjust flow rate
- adjust chlorine dose or feed point
- carry out additional monitoring, increase sampling and testing
- recalculate C.*t* values
- implement unplanned maintenance procedure
- secondary or booster disinfection
- use alternative supply or divert water
- engage backup equipment
- plant automatic shutdown
- implement emergency response
- record actions to be taken and report (internally or externally as required).

Verification

- Calibration and maintenance of equipment
- Drinking water quality monitoring
- Consumer satisfaction
- Evaluation and audit

9.5 Verification of drinking water quality

Verification of drinking water quality provides an assessment of the quality of drinking water being supplied to consumers. It incorporates monitoring drinking water quality in the distribution system and assessing consumer satisfaction.

Verification of drinking water quality provides an important link back to the operation of the water supply system and additional assurance that the preventive measures and treatment barriers in the water supply system have worked, and are working, to supply safe drinking water. This information helps in assessing long-term system performance and identifying any trends or problems within the water supply system that may have gone unrecognised, and it provides confidence to consumers and regulators regarding the quality of water supplied.

9.5.1 MONITORING CONSUMER SATISFACTION

Monitoring consumer satisfaction can provide valuable and timely information on potential problems that may go unidentified by performance monitoring. Changes from the norm are particularly noticeable to consumers, who are often the first to identify something unusual about the water delivered to their tap. For example, there is evidence from waterborne disease outbreaks that consumer comments and complaints have drawn attention to changes in water quality or quantity that ultimately led to the outbreak (Box 9.6).

In addition, because consumers are located throughout distribution systems, they offer a wide-ranging source of information on potential contamination, compared to limited monitoring in the distribution system.

An effective consumer complaint and response system that is operated by trained personnel and closely linked to the operation of the water supply system is an important component of any preventive strategy for drinking water safety. The types of complaints that could signal potential contamination include off taste, off odour, turbidity, unusual colour, reduced water pressure, water supply interruption, suspicious activity, or illness (see Box 9.7). All complaints need to be investigated and documented, including the associated responses. Complaints of illness warrant particular attention and should be reported to the health authority for joint investigation.

Clearly, water suppliers would like to operate in a manner such that consumers will never need to complain. Nevertheless, to maximise the ability to detect contaminated water and respond to problems effectively, a water supplier should ensure that consumers are educated on what to expect in relation to the quality of their water (what is normal) and are encouraged to inform the supplier of any water-related concerns, including symptoms of illness.

***Box 9.6** Responding to customer complaints*

n 1993, Milwaukee, USA, experienced an outbreak of cryptosporidiosis. Difficulties in treatment operation resulted in the turbidity of treated water reaching levels much higher than normal. Not only did operators fail to respond to the turbidity spike, they also failed to recognise the significance of the accompanying dramatic increase in consumer complaints, which reached nearly 50 one day around the time of the turbidity spikes, against a background of less than 5. This suggested that the quality of water was substantially impaired, but the failure to recognise the problem meant that the chance of effective response and immediate corrective action was lost (Hrudey and Hrudey 2004).

A number of other documented outbreaks have involved some level of consumer detection of problems (Hrudey and Hrudey 2004).

9.5.2 DRINKING WATER QUALITY MONITORING

Drinking water quality monitoring is used to provide assurance that the quality of drinking water in the distribution system, as supplied to the consumer, is meeting guideline values, agreed levels of service, and/or any regulatory requirements. It can provide an additional means of detecting any unrecognised problems that may be occurring upstream or within the distribution system, and can trigger the necessary corrective actions.

Drinking water quality monitoring cannot prevent unsafe water being supplied to consumers, as results are typically not available for days to weeks after collecting the sample, so that any corrective actions occur *after* the water has been supplied. **Drinking water quality monitoring should not, therefore, be used in place of or as a substitute for operational monitoring.**

As it is neither physically nor economically feasible to test for all drinking water quality characteristics equally, monitoring effort and resources should be directed at significant or 'key' characteristics – that is, those characteristics identified in the system-specific hazard identification and risk assessment process as likely to be present. These key characteristics require more frequent monitoring. Characteristics that the risk assessment shows are unlikely to be present, or pose a low risk, are monitored very infrequently, or may not need to be included in the drinking water quality monitoring program.

Generally, sampling and analysis are required most frequently to assure microbial safety and less often for chemical and radiological compounds. This is because of the acute and almost universal health risk posed by waterborne microbial pathogens, whereas the guideline values for most (but not all) chemical characteristics are based on lifetime exposure. In the absence of a specific event (e.g. spills, chemical overdosing at a treatment plant), episodes of chemical contamination that would constitute an acute health concern are rarer.

Sampling locations for drinking water quality monitoring

Drinking water quality monitoring confirms the final quality of water that is supplied to consumers. As such, it needs to be undertaken throughout the distribution system at points representative of the quality of water supplied to consumers' properties (e.g. at or close to water meters).

The location and number of sampling points within a distribution system are determined by the complexity of the system. For purposes of management, monitoring and reporting, large and complex distribution systems should be divided into discrete water quality monitoring zones. These zones are typically:

- supplied from a single source, and/or
- hydraulically separated from other zones (single or multiple sources).

As the priority for monitoring drinking water quality is to confirm microbial safety, the design of the microbiological sampling program often dictates the location of sampling points. Sampling points are normally placed well into the distribution system to be representative of what most consumers have received. They should also be spread geographically to give coverage across the system or zone.

Circumstances where microbial quality has the potential to change within a distribution system need to be considered. This is most likely where the system is depressurised, increasing the chance for ingress (e.g. at a service tank). Sample points should, therefore, be included downstream of any tanks (often called a subzone) even though the source water may be unchanged.

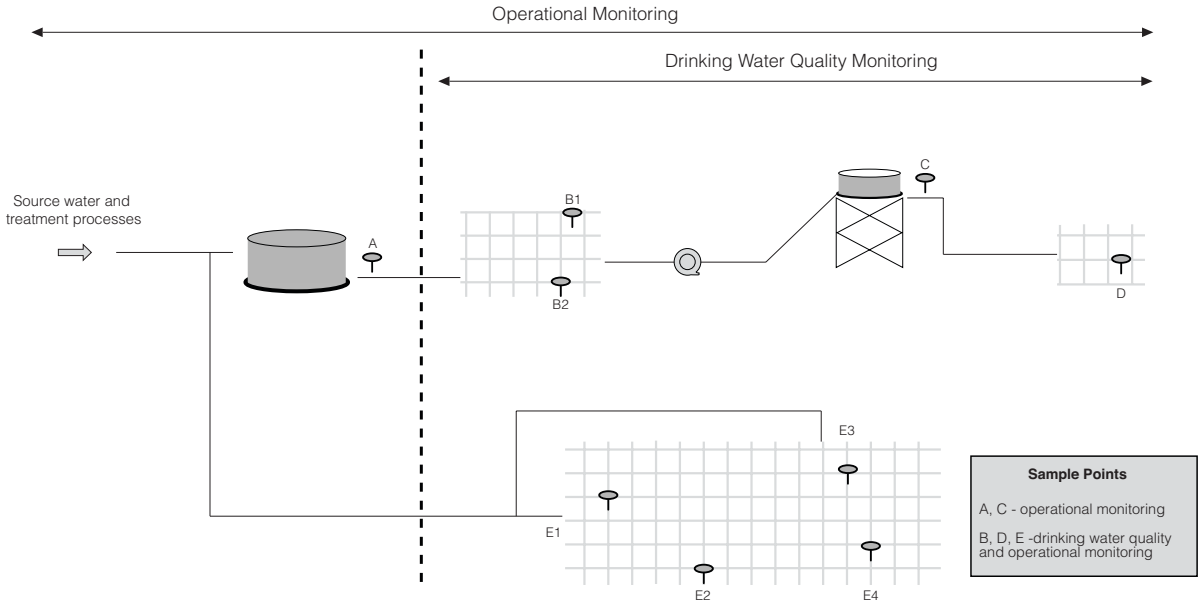
Samples for physical and chemical quality monitoring can usually be taken from the sample points used for microbiological monitoring. Since physical and chemical quality monitoring requires many fewer samples in a given period, a decision must be made on whether to rotate sampling around all the sample points within a zone (providing an indication of performance across the zone) or to use only one or two fixed sample points (providing an opportunity to plot trends).

For chemical characteristics that are more stable and unaffected by the distribution system, sampling can occur at the entry point to the distribution system. For some characteristics that change across the distribution system (e.g. THMs), additional monitoring from specific areas may be required to ensure the data collected are representative of all water supplied.

Operational monitoring such as chlorine residual monitoring is typically also carried out concurrently at these sample points, as well as at other strategic locations within the distribution system, such as entry points (e.g. outlets of service reservoirs/tanks), trunk mains, and dead ends.

Figure 9.3 provides an illustration of sampling locations within a typical distribution system and shows how operational monitoring and drinking water quality monitoring are integrated.

Figure 9.3 *Example of sample locations in a typical distribution system*



Microbial quality – sampling frequency

Routine monitoring for specific microbial pathogens is not recommended as it is usually complex, expensive and time-consuming, and may fail to detect their presence. Rather, the recommendation is to monitor for the microbial indicator bacterium ***E. coli*** as a marker for the presence of faecal contamination and the possible presence of microbial pathogens (see fact sheets on microbial indicators). Whilst there are limitations to the use of ***E. coli*** as an indicator of faecal contamination of water supplies (e.g. ***Cryptosporidium*** oocysts may survive chlorine disinfection and may be present in the absence of ***E. coli***), it is currently the best verification indicator available for faecally related microbial quality.

The recommended minimum monitoring frequency for ***E. coli,*** based on World Health Organization recommendations (WHO 2008), is detailed in Table 9.4. Samples should be collected at points within the distribution system that are representative of the quality of water supplied to consumers.

Table 9.4 *Recommended minimum frequency of* E. coli *monitoring*

Samples that are representative of the quality of water supplied to consumers should be collected and analysed for *E. coli* at the following minimum frequency:

Population in monitoring zone	**Minimum number of samples**
>100,000	Six samples per week per monitoring zone, plus one additional sample per month per monitoring zone for each 10,000 above 100,000
5,000–100,000	One sample per week per monitoring zone plus one additional sample per month per monitoring zone for each 5,000 above 5,000
1,000–5,000	One sample per week per monitoring zone (52 samples per year)
<1000	One sample per week per monitoring zone (52 samples per year). Where the water supply in this category is remote, the recommended sampling frequency needs to be balanced against the logistics of collecting the samples, the risk profile for the supply, and the risk mitigation processes that are operating on the supply. With remote water supply systems, regular physical inspections and operational monitoring are more beneficial to ensuring water quality than infrequent *E. coli* sampling.

Sampling frequency should be increased at times of flooding or emergency operations and following repair work or interruptions to supply.

The frequency of sampling should be increased during any significant environmental events or emergency operations, or following interruptions of supply or repair work. More frequent sampling should also occur at sample points where previous results have indicated potential problems. Operational monitoring such as disinfectant residuals, temperature and turbidity are often taken concurrently with *E.coli* to provide complementary evidence of system status and enhance interpretation of data.

The results of the *E. coli* monitoring program will not prevent unsafe water being supplied to consumers, and drinking water quality monitoring should not be substituted for or used in place of a well-constructed operational monitoring program. For systems serving small communities, regular physical inspections of the water supply system, and the monitoring of critical processes and activities, such as chlorination, yield more information than infrequent sampling (see Chapter 4).

Drinking water quality (non-microbial) – sampling frequency

Monitoring requirements for non-microbial characteristics will vary for each water supply system, depending on the key characteristics identified through water supply system analysis and risk assessment. In general, characteristics that pose a high level of risk require more frequent monitoring, while those posing a low risk require less monitoring. The closer the mean value of a characteristic is to the guideline value, and/or the greater its variability, the more frequent the monitoring needs to be. Those characteristics that are deemed, on the basis of a thorough analysis of the catchment and water supply system, unlikely to be present will typically require very infrequent monitoring, or no monitoring at all.

Table 9.5 provides a generic guide to monitoring frequency for drinking water quality characteristics. Monitoring frequencies and characteristics for individual systems should be adjusted as needed, based on the ongoing review of the water supply system and risk assessment.

Table 9.5 *Generic frequencies for monitoring non-microbial drinking water quality as supplied to the consumer*

	Frequency of sampling				
	Weekly	***Monthly***	***Quarterly***	***Annually+***	***Comments***
Physical characteristics	pH Temperature Total dissolved solids[1]	Colour Turbidity Dissolved oxygen Hardness[2]		Taste and odour	[1] If reverse osmosis used, or there are known salinity issues, otherwise quarterly [2] If water is treated for hardness
Water treatment related chemicals (if used)	Fluoride[1] Aluminium[2] Chlorine Copper (seasonal)		Any related organic contaminants, e.g. acrylamide, carbon tetrachloride, epichlorohydrin		[1] Will not require frequent monitoring if fluoridation is not carried out or if naturally occurring fluoride is either absent or at a level below the level for optimal fluoridation. [2] Aluminium not likely to be present if no alum-based coagulant is used.
Disinfection byproducts (DBPs)		THMs[1,2] Ammonia, nitrite, nitrate[2] Bromate, formaldehyde[3] Chlorite[4]			[1] Where chlorine or chloramine are used. [2] Where chloramine is used. [3] Where ozone is used. [4] Where chlorine dioxide or liquid chlorine is used. If detected at elevated concentrations, close to, or above guideline values, additional related DBPs should also be analysed.
Inorganics	Iron Manganese		Arsenic, nitrate, fluoride, selenium, lead, mercury[1] Ammonia, cadmium, chromium, nickel, zinc, copper, hydrogen sulfide	Tin, silver beryllium, uranium, iodide, molybdenum, boron, barium	[1] Priority contaminants: quarterly sampling for groundwater sources, more frequent monitoring when detected at elevated concentrations; otherwise sampling reduced to annually, seasonally or event-related (e.g. storm events, reservoir turnover events).
Pesticides and organic toxicants		If detected or potential presence		If not detected	Monthly or quarterly sampling for pesticides/organic toxicants previously (or potentially) detected; seasonally annually, or event-related (e.g. storm events, spills) for other pesticides/organic toxicants.
Radiological				Radionuclides	New supplies should be assessed quarterly for one year, then every 2 years (groundwater) or 5 years (surface water). Increase frequency to quarterly if guideline screening levels exceeded.

9.6 Water quality issues beyond the point of supply

Under most jurisdictional legislation and arrangements within Australia, the responsibility of water suppliers ends at the point of supply to the customer, typically the water meter. The primary responsibility for ensuring that water supplied beyond the water meter remains safe and aesthetically acceptable rests with various stakeholders including:

- building and site owners or managers;
- plumbing and building regulators;
- plumbers;
- plumbing material suppliers;
- private individuals.

Under the catchment-to-consumer tap preventive management framework promoted by these Guidelines, however, water quality should be managed up to the point of consumption, usually the customer tap, to account for water quality changes that may arise as a result of the internal plumbing arrangements on customer properties. This management may be achieved by liaison between the water supplier and the stakeholders listed above.

Both microbial and chemical quality can deteriorate within buildings due to poor design and management of internal plumbing systems. While internal plumbing systems are largely outside of the control of water suppliers, incompatibility between the chemistry of drinking water as supplied and the quality and nature of internal plumbing and fittings can cause system-specific impacts, and it is reasonable to expect that water suppliers be aware of these issues. The two most common issues are:

- plumbosolvency – that is, mobilisation of lead into solution from lead pipes and brass fittings (which may contain traces of lead), and the solder used to join pipes, as a result of the supply of plumbosolvent water. The issue of plumbosolvency is rare in Australia. Similar issues can arise with the corrosion of pipes and fittings containing copper (cupprosolvency), leading to "blue" water;
- hardness – scaling of pipes, and of water elements in kettles and hot water services, resulting from the supply of very hard water.

Other possible impacts include the following:

- The supply of unbuffered desalinated water into areas not traditionally supplied with water of reduced salinity may exacerbate corrosion, particularly in hot water systems.
- Microbial and chemical contamination can be associated with distribution systems in large buildings. This risk arises particularly where large volumes of water are stored for extended periods in on-site header tanks, or ingress of untreated water occurs through faults in the pipe network, or there are cross-connections with non-drinking water supplies.
- Drinking water that sits unused in pipe networks for extended periods of time may have elevated levels of metals. This is seen particularly in schools after lengthy holiday breaks, where water to drinking fountains has remained stagnant in pipes, with the result that children have consumed water with elevated levels of copper (Scholz ***et al.*** 1995, Walker 1999, Brodlo ***et al.*** 2005).

Role of building and site owners and managers and plumbing oversight agencies

The ***Trade Practices Act 1974*** requires plumbing and fittings to be fit for purpose, and that purpose includes being fit for the safe conveyance, storage and use of water of a chemistry as supplied within a particular area. Building and site owners, and managers and plumbing oversight agencies, are responsible for ensuring that the plumbing systems and fittings used within their areas of responsibility are fit to convey drinking water without leading to exceedances of water quality guidelines. In addition, these stakeholders should liaise with standards-setting bodies and water suppliers to ensure that the procedures for approving plumbing materials, fittings and systems are adequate, and that any products that are used comply with the requirements of ***AS/NZS 4020:2005: Testing of Products for Use in Contact with Drinking Water.***

Role of water suppliers

Although Australian water suppliers are not responsible for the actions related to water quality management beyond the point of supply, they should be aware that the drinking water that they supply may interact with internal plumbing and cause unintended water quality issues (either aesthetic or health-related). The ***Trade Practices Act 1974*** requires water supplied by water suppliers to be fit for purpose, including the conveyance, storage and use of that water within approved plumbing assets, fittings and plumbed-in systems available in water supply areas. In effect, this means that water suppliers have obligations if they are aware of potential negative impacts of mains water on correctly designed and installed plumbing systems.

Some recommended actions that water suppliers can take to minimise the risks associated with interaction of internal plumbing and supplied drinking water are:

- Liaise with relevant state-based plumbing authorities to ensure that plumbers use only materials that meet the requirements of ***AS/NZS 4020:2005: Testing of Products for Use in Contact with Drinking Water.***
- Liaise with standards-setting bodies and plumbing regulators to ensure that the procedures for approving plumbing materials, fittings and systems are adequate to manage any short-, medium- and long-term risks associated with those materials, fittings and systems when carrying the water supplied in any particular supply area.
- Prepare information for customers on water quality issues that may have an adverse impact on their internal plumbing.
- Provide advice to customers with large reticulated networks on water quality issues that may arise from having stagnant water within their pipe networks.
- Develop and disseminate information to schools, highlighting, in particular, issues related to stagnant water, and suggesting that drinking fountains and other water-using devices be flushed before school returns after holiday periods.
- Ensure, wherever practicable, that each property is separately metered so that areas of low flow can be identified.
- In liaison with building and site owners and managers and plumbing oversight agencies, consider undertaking investigative monitoring studies to examine the interactions of water as supplied with the plumbing and fittings used in the water supply area.

Useful additional references on this issue include Rajaratnam ***et al.*** (2002), WHO and World Plumbing Council (2006), and WHO (2010).

9.7 Investigative studies and research monitoring

Investigative studies and research monitoring can be used to increase understanding of a water supply system, identify and characterise potential hazards, fill gaps in knowledge, and inform targeted capital expenditure, system augmentation and operational improvements. By improving understanding of the factors affecting water quality characteristics, such monitoring allows suppliers to anticipate periods of poor water quality and respond to them more effectively.

Investigative studies and research monitoring can often also be used to provide further information for the risk assessment process and reduce uncertainty. Examples include:

- baseline monitoring of characteristics or contaminants in potential new water sources, to identify water quality problems (Box 9.7);
- source water monitoring, to understand the temporal and spatial variability of water quality characteristics;

- event-based monitoring in source water and catchment areas, to determine the magnitude of impacts (duration and maximum concentrations);
- developing early warning systems, to improve the management of poor water quality;
- examining mixing effects within a water storage;
- evaluating characteristics of an aquifer through pumping tests and analyses;
- studying the movement of water within reservoirs, to determine short-circuiting effects;
- examining backwash return water and its effect in increasing microorganism load;
- examining the effects of natural events that affect drinking water quality, such as bushfires or floods (Box 9.8).

***Box 9.7** Baseline monitoring of new drinking water sources*

Baseline monitoring of raw water quality should be carried out for all new water supplies being considered, as well as any poorly characterised existing systems.

Baseline monitoring informs the hazard identification and risk assessment process, and the development of effective ongoing monitoring regimes, by identifying major water quality problems and the key characteristics that should be routinely measured. This characterisation of the water supply also establishes a base for assessing long-term trends and changes in water quality over time, and provides information to compare and select source waters for future supply.

The extent of sampling and the timeframe required for a baseline assessment will depend on land use in the catchment, levels of pollution found, and variability or trends in water quality. A land-use survey of the catchment should be carried out to identify any important features likely to affect water quality. Where catchments and supplies are beyond the water supplier's jurisdiction, exchange of information and collaborative assessment of the quality of source waters is strongly recommended.

The baseline water quality and potential levels of risk should be periodically assessed to identify any significant changes in water quality arising from changed land-use practices or the impacts of water abstraction (particularly from unconfined aquifer systems), as well as longer-term natural variability in water quality that may not have been evident from initial baseline monitoring.

Detailed advice on what characteristics to consider in a baseline monitoring program can be found in CRCWQT report 11 – *A Guide to Hazard Identification & Risk Assessment for Drinking Water Supplies* (2004).

***Box 9.8** Investigative studies on the effect of floods on water quality*

Following many years of drought, a record dry year in 2006 yielded the lowest streamflows on record for Melbourne's protected water supply catchments. The dry conditions continued until a storm in late June 2007. While not enough to break the drought, the resulting run-off produced very turbid inflows to Upper Yarra Reservoir and ultimately high turbidity into some of Melbourne's unfiltered water supply.

As turbidity increased in the water supplied to customers, Melbourne Water carried out monitoring to determine whether:

- the stormwater run-off from the forested catchments represented an increased risk of protozoan or bacterial pathogens in the raw water; and
- the existing ultraviolet (UV) and chlorination processes could adequately inactivate bacterial pathogens at higher turbidities.

Inspection and monitoring of the streamflows in the Upper Yarra catchment in the days following the storm event indicated the potential for a major water quality incident in the days following. The turbidity of streamflows in the Yarra River and water transferred from the Thomson River (via a tunnel to the Yarra River) were in the range of 50 to 100 NTU two days after the storm.

A range of investigations and sampling were undertaken within and downstream of Upper Yarra Reservoir in response to the turbidity incident (Hellier and Stevens 2009). This included genotyping of *Cryptosporidium* isolates.

A beneficial outcome of the incident has been the increased knowledge of water quality risks associated with the catchments and improved disinfection validation information. In particular, Melbourne Water has established a specific public health limit of 15 NTU for turbidity for its unfiltered, protected catchment sources, this being the point at which the UV dose was reduced to near critical limits.

The experience with pathogen monitoring highlighted the importance of rigorous risk assessment, underpinned by previous catchment research, to support water supply management decisions (e.g. whether boil water advisories are warranted).

An important lesson was the value of event sampling of streamflows into reservoirs for future events to characterise the turbidity and microbial load – first bacterial indicators, then pathogens if warranted. This was not carried out in the 2007 incident and would have assisted in an earlier determination of pathogen risks (Hellier and Stevens 2009).

9.8 Validation of barrier performance

Typically, validation monitoring is required where new treatment processes or significant operational changes are being implemented. In particular, validation monitoring provides assurance that, where health-related assumptions are made (e.g. that the barrier will adequately remove ***Cryptosporidium***), that these assumptions are justifiable (see Section 3.9.2).

Validation monitoring involves identifying the operational requirements that should be used to ensure that processes reduce risk to an acceptable level on an ongoing basis. In some cases, validation can be completed entirely using desktop assessment based on existing evidence; in other cases, objective empirical evidence from monitoring is needed. Validation monitoring may form part of the validation evidence base, but the precise nature of the evidence required depends on the nature of the process being validated.

One of the most common applications of ***in situ*** validation monitoring is during or just after commissioning of new unit processes. Once the process is considered to be operating as intended, but before it is brought on line to supply water to consumers, microbial and/or chemical characteristics should be assessed in samples taken before, during or after the unit process to confirm that it can reduce the concentration of substances to the extent required.

Many drinking water treatment plant manufacturers, or suppliers of treatment processes, will undertake such tests on modular units and then market those units as being pre-validated. This is commonly true for membrane and ultraviolet treatment systems. Where a pre-validated unit or system is used, then separate validation of the unit is not considered necessary, providing the validation is appropriate for the characteristics of the water to be treated.

Some examples of where validation monitoring should be undertaken include:

- monitoring cyanotoxin concentrations pre and post a powdered activated carbon dosing system, to check the toxin reduction capability of the batch supplied;
- monitoring microbial indicator and particle count concentrations pre and post a media-based filtration plant, to check its pathogen-reduction capability;
- monitoring arsenic concentrations pre and post an arsenic treatment plant, to check its arsenic-reduction capability.

As part of the validation process, validation should also be undertaken on control systems, such as alarm systems or systems that instigate a plant shutdown, to ensure that they are operating correctly and respond to exceedances of target criteria or control limits.

Once a unit process has been validated, ongoing monitoring of the unit is needed to ensure that it is operating correctly. This ongoing monitoring will form part of the operational monitoring program for the water supply system.

Table 9.6 gives examples of typical validation monitoring programs for a range of commonly used unit processes, as well as providing advice on the ongoing proof-of-performance testing that is part of the operational monitoring program.

Table 9.6 *Examples of validation monitoring and proof-of-performance testing*

Process step to be validated	**Validation monitoring**	**Characteristics that will subsequently be used ascertain ongoing proof of performance**
Media filtration plant[a]	Establish optimal filter run times and associated operational envelope. Establish optimal ripening periods and associated operational envelope. Inlet and outlet microbial indicator concentrations:[b] • Monitoring should at the very least include plate count and *E. coli*; it would ideally include coliphage and clostridial spores; and it may include some pathogens.	• Turbidity upstream and downstream of system • Pressure loss across each filter bed • Particle counts on outlet • pH and temperature • Coagulant dosage rate • Streaming current
Membrane plant (microfiltration or ultrafiltration)[c]	Establish operational envelope with respect to factors such as transmembrane pressure, flux and temperature. Inlet and outlet microbial surrogate concentrations:[b] • Refer to the USEPA *Membrane Filtration Guidance Manual* (2005)	• Filtrate turbidity • Filtrate particle counts • Membrane integrity testing • Transmembrane pressure • Flux
Membrane plant (reverse osmosis)	Inlet and outlet microbial surrogate concentrations:[a] • Refer to the USEPA Membrane Filtration Guidance Manual (2005)	• Electrical conductivity and possibly total organic carbon • Oxidation-reduction potential • Flux (recovery)
Ultraviolet plant[c]	Establish operational envelope with respect to factors such as flow, UV transmissivity and turbidity. Inlet and outlet microbial indicator concentrations:[b] • Refer to the USEPA *UV Disinfection Guidance Manual* (2006)	• Turbidity upstream of disinfection system • UV transmissivity • UV intensity and/or calculated dose • Flow rate to enable calculation of retention times • Ballast functionality, lamp power and lamp status • Lamp age • Lamp fouling
Chlorination plant	Validation of C.*t**.	• Turbidity upstream of disinfection system • Free chlorine, temperature and pH at downstream monitoring point that represents the total required contact time • Flow rate to enable calculation of contact time (C.*t*)
Backflow controls	Check pressure at lowest pressure parts of system on peak flow days to ensure no negative pressure events	• Pressure measured at pump stations and tank levels at service reservoirs

a Validation testing of media filtration systems is a highly specialised and complex task.

b If inlet microbial indicator concentrations are too low to enable validation of the expected microbial reduction performance, seeding of challenge microorganisms should be required.

c The USEPA provides definitive guidance on the validation of these types of unit processes.

* C*t* = a measure of free chlorine residual concentration (C) and contact time (*t*)

9.9 Incident and emergency response monitoring

General aspects of incident and emergency response are discussed in Section 3.6. Any emergency or incident is likely to trigger an increase in monitoring frequency. The increase in testing frequency for grab samples should reflect the risk that the incident poses to consumers and the characteristics being monitored. The increase in testing frequency should continue until water quality is confirmed as being back within specification.

Emergency incident plans need to take into consideration the capability and availability of operational and laboratory personnel. Experience shows that overwhelming laboratories with samples during incident conditions can cause major problems for laboratory quality control and can lead to adverse outcomes. It is important to maintain the quality of laboratory analysis regardless of the urgency of testing. It may be necessary to have contracts in place with back-up laboratories that can provide support during incident or emergency response conditions.

9.10 Reliability of monitoring data

9.10.1 SAMPLE INTEGRITY

If the data collected as part of a monitoring program are to be meaningful, the samples need to be collected from appropriate locations, by trained personnel, working to a predetermined plan, and the procedures employed in the collection, preservation and transport of samples to the laboratory should be chosen with regard to the characteristics being measured.

Procedures for sampling and sample preservation are provided in Information Sheet 2.1, Sampling Information – Handling Requirements and Preservation.

9.10.2 METHODS

It is important that the results obtained in analyses are valid. If analysis of water supplies is to be useful, methods must yield consistent results; however, different methods of analysis can in some cases give different results on the same water sample. To ensure consistency, at least one standard method of analysis is suggested in the fact sheet for each characteristic listed in the Guidelines. Wherever possible, the recommended method should be used, as it is well documented, has undergone extensive evaluation (preferably through comprehensive inter-laboratory comparison programs), is readily available and is widely used by larger water suppliers. If other methods are to be used, it is important that they meet these criteria, and give results that are consistent with the standard methods. Even minor variations to documented methods can lead to inaccurate measurements.

Whatever analytical technique is used, it must give a result that can be compared to the listed health and aesthetic guideline values. This is especially true in relation to the limit of detection for the method. Wherever possible, the method used should have a limit of detection that is less than the guideline value. The limit of detection for each suggested method has therefore been provided in the relevant fact sheet.

The whole analytical process, from sampling through to presentation of results, needs to be managed in accordance with sound quality assurance principles and should include quality control checks as part of the quality assurance process. Quality assurance principles are set out in documents such as ISO 9001, and supported by programs such as the National Association of Testing Authorities (NATA) schemes. Wherever possible, analyses should be undertaken at NATA-accredited laboratories.

As part of the testing procedure, certified reference materials should be used so that results can be benchmarked against a specified reference. This will help ensure that measurement results from different methods and laboratories are comparable.

9.10.3 DETECTION LIMITS

Ideally, the detection limit of the analytical method used should be lower than the level at which the characteristic might become a health concern. A health-based guideline value usually provides a benchmark against which the detection limit of the analytical methodology can be measured. If the methodology cannot achieve such a detection limit, there will inevitably be some difficulty or imprecision in assessing risk, and efforts should be made to find a more sensitive analytical method.

Some fact sheets mention limit of detection (LOD) whilst others refer to a limit of quantitation (LOQ). The LOD is normally used to indicate the lowest level that can be reliably detected, while the minimum level than can be reliably quantified (LOQ) is often somewhat higher than the LOD.

The advice of an analyst should be sought when selecting the analytical method to be used.

9.10.4 MEASUREMENT UNCERTAINTY

There is an inherent level of uncertainty associated with the measurement of water quality characteristics, in addition to the uncertainty arising from sampling. This inherent uncertainty arises from a number of sources, but it primarily relates to the accuracy of the laboratory equipment used to produce a result, and various measurement errors that may be introduced through the analytical process.

In some cases, the level of uncertainty will be insignificant relative to the quoted result; in other cases, however, it can be quite significant. Under the ISO Standard used by NATA for accreditation, ***AS ISO/ IEC 17025—2005 – General Requirements for the Competence of Testing and Calibration Laboratories,*** laboratories are required to estimate the uncertainty associated with the results they produce (known as the measurement uncertainty).

Upon request, NATA-accredited laboratories are required to provide the measurement uncertainty (MU) associated with a particular analytical result (x), which should be expressed by the laboratory as either x ± MU, or as a percentage of x, or in any other manner accepted by the relevant accreditation body.

Organisations performing water quality testing are encouraged to request that their laboratories provide MU data as part of their analytical results reporting. This will promote an appreciation of the variability in the analytical data being received.

9.10.5 FIELD TESTING

Field testing can be used for operational monitoring of drinking water supplies, and its use is encouraged, particularly for small and remote systems where access to laboratory-based testing is difficult.

Some tests, including those for temperature, free and total chlorine and monochloramine, are always undertaken in the field. Sample storage times and conditions affect results such that unless analysis can be undertaken within a short time of sampling, field testing is the only method of deriving representative results.

Beyond those tests which must be done in the field, it is possible to acquire, at reasonable cost, basic chemical test kits for common physical and chemical characteristics, including hydrogen sulfide (H_2S), pH, dissolved oxygen, electrical conductivity, colour, iron, manganese, turbidity, chlorine and fluoride. These test procedures are well within the capabilities of trained treatment plant operators and system caretakers. The test results should generally, however, be regarded as indicative only, and should complement, but not replace, more reliable laboratory tests.

Recent advances in field tests for indicator microorganisms, such as total coliforms and ***E. coli***, are making such tests feasible as part of drinking water quality monitoring in small and remote locations where it may not be possible to get samples to laboratories within the timeframe required for accurate analysis, or the costs of doing so are prohibitive.

The test results of field testing will not have the standing of those produced by NATA-accredited laboratories, but they do permit regular and frequent monitoring. What the field tests sometimes lack in precision and reliability needs to be balanced against the benefits of the increased frequency of monitoring that is possible. Furthermore, such kits enable many tests to be performed in the field, thus avoiding the need to preserve and transport samples to a laboratory.

In all cases where field testing is undertaken, it is essential that those doing the testing are appropriately trained, that analysers are calibrated as per the manufacturers' specifications, and that an audited quality assurance program, ideally including proficiency testing, is in place to monitor testing performance. The extent to which a monitoring program relies on the results of field kits should be discussed with the relevant health regulator.

9.11 Monitoring advice for small, remote or community-managed water supplies

While small, remote water supplies are typically managed by a community group or a small private operator, some are managed by water utilities.

The same general principles apply for such supplies as for any other, with decisions on monitoring informed by risk assessment, and operational monitoring taking a higher priority. For example, tests of microbial quality of drinking water are a valuable adjunct to, but not a substitute for, assessing source water protection, treatment, and the integrity of treatment barriers through to the consumer's tap. Given the limitations in the ability of indicators to predict health risk accurately, it is essential to maintain effective barriers to faecal contamination.

Operational monitoring of small supplies will typically include a greater focus on observational monitoring, including regular inspections (preferably weekly) of:

- local source water catchment or recharge areas and source water reservoirs;
- boreheads, to ensure that they are sealed and secure;
- fences and enclosures around bores, tanks and other infrastructure;
- tank roofs and above-ground pipes and valves, to ensure that integrity is maintained, roofs are intact and there are no breaks or leaks; and
- drainage at bore sites, air valve pits and scour valve pits.

Contamination events are often associated with extreme events, and observational monitoring should be undertaken to assess impacts of heavy rainfall, flooding and storms on infrastructure.

The most common form of treatment in small supplies is disinfection. Where applied, it will always be a critical control point. Ideally disinfection should be monitored continuously using automatic systems with alarms. If this is not available, it should be replaced by frequent manual checks as detailed in Table 9.7.

Table 9.7 *Recommended operational monitoring at disinfection points in small, remote or community-managed water supplies*

Operational monitoring – disinfection	
Chlorination	**Ultraviolet**
Chlorine residual continuously monitored with alarms for non-compliance with critical limits (residual below minimum concentration) Chlorine residual checked manually downstream of chlorinator several times per day Observations of chlorinator performance such as: • correct rotameter setting • hypochlorite dosing pump operating correctly • the amount of gas or liquid hypochlorite being used by the system • sufficient quantities of disinfectant available to ensure chlorination is not interrupted	UV light intensity continuously monitored with alarms for non-compliance with critical limits UV light power available and UV running checked several times per day. Observations of UV light performance such as heat of sleeves, fuses operational etc.

If other treatment processes are applied, then appropriate operational monitoring will be needed. This should follow similar principles to the monitoring of disinfection, with on-line monitoring of disinfection preferred. If on-line monitoring is not available, it should be replaced by frequent manual testing.

In general, drinking water quality monitoring of small water supplies should be based on the principle that it is much more effective to test for a narrow range of key characteristics as frequently as possible, than to conduct comprehensive but lengthy (and possibly largely irrelevant) range of analyses less often.

Key characteristics to be monitored as part of drinking water quality monitoring should include those with the potential to present significant risks and for which reliable verification of safety is required. As discussed in Section 9.2.1, priority chemicals particularly for groundwater supplies include arsenic, fluoride (at concentrations above those applied for dental protection), selenium, nitrate, lead and uranium. Iron, manganese, total dissolved solids and hardness can be sources of aesthetic water quality problems.

If data are not available, testing should be undertaken to determine background concentrations of key health-related hazards. This should be informed by a risk assessment, taking account of local geology, potential sources of chemicals (e.g. existing or abandoned mining sites) and known problems in the area identified by testing of nearby water supplies. Initially, quarterly monitoring is recommended, to include consideration of any seasonal influences.

Further testing should be based on mean concentrations and variability. In general, the closer the mean value of a characteristic is to the guideline value, or the greater its variability, the more frequent the monitoring needs to be (for these characteristics, the suggested frequency is annual). Those characteristics that, based on risk assessment, are not likely to be present or have been shown to be present in concentrations well below guideline values typically require monitoring very infrequently (e.g. every two years) or not at all.

Observational monitoring should be used to supplement the chemical testing program; for example, checking for chemical spillage and appropriate application, uses and storage of fertilisers and pesticides.

The monitoring program (and the available results) should be discussed with the regulator who has responsibility for the oversight of drinking water quality, to determine an appropriate sampling frequency.

Frequent testing for aesthetic characteristics is generally not justified once concentrations are established unless variability is expected or specific controls are introduced (e.g. desalination, pH correction, filtration of surface waters).

While weekly ***E. coli*** testing is recommended in Table 9.4, there can be practical difficulties in performing microbial testing for small remote communities. The recommendation to collect weekly samples needs to be balanced against the logistics of collecting, preserving and transporting the samples, the risk profile for the supply, and the risk mitigation processes that are operating on the supply. Alternatives can include less frequent testing or the use of field kits.

The following resources have been developed to assist operators of small remote water supplies:

- the NHMRC ***Community Water Planner – A tool for small communities to develop drinking water management plans,*** which focuses on microbiological water quality (http://www.nhmrc.gov.au/publications/synopses/eh39.htm);
- a field guide also developed by the National Water Commission to assist the implementation of the Community Water Planner in remote Indigenous communities (http://www.wqra.com.au/cwplanner/CWPlanner.htm).

All of these tools provide guidance on monitoring requirements.

9.12 References

ANZECC (Australia and New Zealand Environment and Conservation Council) and ARMCANZ (Agriculture and Resource Management Council of Australia and New Zealand) (2000). *Australian Guidelines for Monitoring and Reporting*. National Water Quality Management Strategy Paper No. 7, ANZECC and ARMCANZ, Canberra.

Brodlo L, Loxley J, Byleveld P (2005). Illness associated with consumption of drinking water contaminated by copper corrosion. Presented at Australian Water Association Specialty Conference "Contaminants of Concern in Water", Canberra 22-23 June. Australian Water Association, Canberra.

CRCWQT (Cooperative Research Centre for Water Quality and Treatment) (2004). Report 11 – *A Guide to Hazard Identification & Risk Assessment for Drinking Water Supplies.*

CRCWQT (Cooperative Research Centre for Water Quality and Treatment) (2007). Report 37 – *Strategic Water Quality Monitoring for Drinking Water Safety.*

CRCWQT (Cooperative Research Centre for Water Quality and Treatment), WQRA (Water Quality Research Australia) (2009). Report 78 – *Risk Assessment for Drinking Water Sources.*

Craun GF, Calderon RL, Nwachuku N (2003). Causes of waterborne disease outbreaks reported in the United States, 1991-1998. *Drinking Water and Infectious Disease: Establishing the Link*, ed. Hunter PR, Waite M, Ronchi E. Boca Raton: CRC Press pp 105-117.

Curriero FC, Patz JA, Rose JB, Lele S (2001). The association between extreme precipitation and waterborne disease outbreaks in the United States, 1948-1994. *American Journal of Public Health,* 91(8):1194-1199.

Gullick RW, Grayman WM, Deininger RA, Males RM (2003). Design of early warning monitoring systems for source waters. *Journal of the American Water Works Association,* 95(11):58-72.

Hellier K, Stevens M (2009). The 2007 storm impacts on Melbourne's unfiltered water supply. Presented at Australian Water Association Ozwater 2009 Conference, Melbourne 16-18 March, Australian Water Association, Sydney.

Hrudey SE, Hrudey EJ (2004). *Safe Drinking Water: Lessons from Recent Outbreaks in Affluent Nations*. International Water Association Publishing, London, UK.

Hunter PR (2003). Climate change and waterborne and vector-borne disease. *Journal of Applied Microbiology,* 94(S):37S-46S.

Logsdon GS, Schneider OD, Budd GC (2004). Hindsight is 20/20: Using history to avoid waterborne disease outbreaks. *Journal of the American Water Works Association,* 96(7):66-74.

Laing RD (2002). *Report of the Commission of Inquiry into Matters Relating to the Safety of the Public Drinking Water in the City of North Battleford Saskatchewan*. Department of Justice, Government of Saskatchewan: 372 pp. Website [accessed May 14 2007]: http://www.northbattlefordwaterinquiry.ca

NHMRC (National Health and Medical Research Council) (2005). *Drinking Water Guidelines: Community Water Planner – A tool for small communities to develop drinking water management plans.* www.nhmrc.gov.au/publications/synopses/eh39.htm

National Water Commission (undated). *Community Water Planner – Physical, Chemical and Radiological.* http://www.communitywaterplanner.net.au/

Nygard K, Wahl E, Krogh T, Tveit OA, Bøhleng E, Tverdal A, Aavitsland P (2007). Breaks and maintenance work in the water distribution systems and gastrointestinal illness: a cohort study. *International Journal of Epidemiology,* 36:873–880.

Logsdon GS, Budd GC, Long BW (1996). Waterborne outbreaks – can we control them? Proceedings of the 1996 AWWA Annual Conference, Toronto, Canada.

Kramer MH, Herwaldt BL, Craun GF, Calderon RL, Juranek DD (1996). Waterborne disease: 1993 and 1994. *Journal of the American Water Works Association,* 88(9):66-80.

Mosse P, Murray B (2008). *Practical Guide to the Operation and Optimisation of Media Filters.* Water Industry Operators Association, Shepparton.

Mosse P (2009). Alarming problems. *WaterWorks – Official Journal of the Water Industry Operators Association*, December 2009, Water Industry Operators Association, Shepparton, p 2-4.

Rajaratnam G, Winder C, An M (2002). Metals in drinking water from new housing estates in the Sydney area. *Environmental Research Section,* A89:165-170.

Scholz A, Cavagnino P (1995). Investigation of suspected copper intoxication in children from drinking water. *NSW Public Health Bulletin*, 6:1-3.

Standards Australia (2005). *AS ISO/IEC 17025—2005 – General Requirements for the Competence of Testing and Calibration Laboratories*

USEPA (United States Environmental Protection Agency) (2005). *Membrane Filtration Guidance Manual.* EPA 815-R-06-009.

USEPA (United States Environmental Protection Agency) (2006). *Ultraviolet Disinfection Guidance Manual for the Final Long Term 2 Enhanced Surface Water Treatment Rule.* EPA 815-R-06-007

Walker NI (1999). Copper toxicosis in an Australian child. *European Journal of Medical Research*, 4:249-251.

WHO (World Health Organization), WPC (World Plumbing Council) (2006). *Health Aspects of Plumbing.* WHO, Geneva.

WHO (World Health Organization) (2008). *Guidelines for drinking-water quality incorporating 1st and 2nd addenda, Vol.1, Recommendations* – 3rd edition. WHO, Geneva

WHO (World Health Organization) (2010). *Water Safety in Buildings.* WHO, Geneva.

Wu J, Greenlee J, Syage J (2004). Rapid screening for chemical weapons infiltration in drinking water. *Journal of the American Water Works Association*, 30(11):44-7.

CHAPTER 10 Monitoring for specific characteristics in drinking water (Revised 2011)

Chapter 10 Monitoring for specific characteristics in drinking water (Revised 2011)

10.1 Introduction

Monitoring data are generated from various sources, such as continuous operational monitoring, field measurements, grab samples, and observational activities. Results from both operational monitoring and verification of drinking water quality should be evaluated over both the short and long term. Results should be documented systematically, and a system of regular reporting of results to relevant staff, departments and external stakeholders (e.g. regulators) should be implemented.

In the short term, monitoring results should be reviewed promptly to assess performance against target criteria and critical limits, guideline values, or agreed levels of service. Where results indicate that established criteria, such as critical limits, have been exceeded or deviated from, or control of the process has been lost, immediate corrective action is required.

The objective of the long-term review of monitoring data is to look at overall system performance in order to enhance understanding of recognised problems, identify any emerging problems and trends, and evaluate the risk to public health and the need for water quality improvement projects. Long-term evaluation of monitoring data can provide confirmation of the hazard identification and risk assessment process, and it assists in corroborating or modifying the assumptions made in the previous risk assessment, as well as increasing system knowledge. It also serves an important due-diligence function with respect to protecting public health, and it contributes to consumer and stakeholder confidence.

For the purposes of this chapter, "short-term evaluation" refers to the routine review of a single monitoring result, or a time-limited (for example, 24 hours) review of on-line monitoring data. "Long-term evaluation" refers to the review of data and the assessment of performance over a time period, typically 12 months (but this could be shorter for critical control points).

10.2 Assessing safety: short-term evaluation of monitoring

Monitoring results should be reviewed promptly and assessed against specified operational criteria, guideline values, agreed levels of service, or previous results, to ensure that preventive measures are functioning effectively and the drinking water quality supplied to consumers is acceptable. Monitoring results that fail to meet established criteria indicate a potential break in process control, and corrective actions are required to resolve the issue and regain control.

Those responsible for interpreting monitoring results and activities should have a sound understanding of the assessment process and the necessary responses, and should be familiar with any communication protocols. A considered approach to responding to potential failures should be developed and documented in advance, and should include any instructions on system investigation, additional monitoring, required adjustments upstream or downstream, and process control changes. The objective of the response is to re-establish the system within operating specification as rapidly as possible.

Immediate response and notification of the relevant health authority is required if there is a significant system failure that could pose a health risk or seriously affect water quality (e.g. non-conformance with critical limits, positive detections of ***E. coli*** within the distribution system, health-related chemical detections above the relevant guideline value). Incident and emergency response plans should be developed to deal with these failures. These plans will be particularly important for times when normal corrective actions cannot re-establish operational performance quickly enough to prevent drinking water of unacceptable quality from reaching consumers (see Section 3.6.2).

A process should be established for documenting and evaluating an event or incident, in order to identify opportunities for improvement. As necessary, the incident should also trigger further investigation, including a long-term review of relevant characteristics, to identify the underlying nature of any problems.

The following sections provide guidance on evaluating and responding to monitoring results from critical control points and other operational monitoring, as well as microbial, chemical and physical monitoring of the quality of drinking water as supplied to consumers.

10.2.1 SHORT-TERM EVALUATION OF OPERATIONAL MONITORING

Operational monitoring is carried out throughout the water system, including source water and catchment, treatment processes, and the distribution system. All operational monitoring results should be promptly reviewed against any established criteria, objectives and previous results to assess whether the system is operating under normal conditions or whether there is an increase in the level of challenge, or the preventive measures and barriers are not performing effectively.

When target criteria and/or critical limits (for critical control points) have not been met, operational staff need to remain aware that the water supply system may not be functioning effectively, and assess the immediate or future risk of supplying unacceptable and possibly unsafe water to consumers.

Box 10.1 sets out priorities for attention when operational characteristics are found to deviate from established operational criteria.

Box 10.1 *Priorities for attention where operational criteria are not met*

The response to deviations from any established operational criteria is risk-based, and will vary depending on the operational characteristic in question and the impact of the deviation on the provision of safe drinking water. As a guide, however, priorities for attention include:

1. deviations that could have a significant impact on critical control point performance; for example:
    - clarifier turbidity deviating from operational target criteria, affecting downstream filtration and resulting in inadequate filter performance;
    - colour of water through a UV plant exceeding the design specification, resulting in ineffective disinfection;
2. deviations that could have a direct impact on the microbial safety of the water; for example, reduction in chlorine residual in the distribution system, especially for water supply systems where *Naegleria* is considered a high risk, or where there is evidence of recontamination or breakdown of distribution system integrity
3. deviations that could contribute to consumers receiving drinking water containing a chemical characteristic above a health-related guideline value;
4. deviations that could contribute to consumers receiving aesthetically poor drinking water.

Critical control points (CCPs)

Of all operational monitoring, monitoring at CCPs is the most critical for assuring drinking water safety. Monitoring at CCPs should occur frequently, preferably continuously, using online analysers, and these analysers should be alarmed at both the target criteria and the critical limits, so that operational staff are alerted promptly of adverse results and effective operational control can be maintained.

Where alarm systems exist, a protocol for alarm response should also be established which considers the length of time an alarm condition can exist before a field response is initiated (to avoid callouts based on background "noise") and the actions to be taken if a field response is initiated. This may be influenced by factors such as the quality of the source water (low risk / high risk), existence of other barriers to contamination, and at which specific barrier or process the alarm occurs.

Target criteria breach

Any breach of target criteria should be regarded as a warning or indication of a change in system status and possibly the start of a trend towards loss of control of the process, which may ultimately result in a breach of a critical limit. Investigation and appropriate corrective actions to resolve any potential problems should immediately be undertaken to ensure a critical limit is not breached.

Possible corrective actions for deviations from target criteria at CCPs include:

- inspection of the water supply system for faults;
- manual backwashing of filters;
- alteration of plant flow rate to reduce loading;
- use of an alternative raw water source;
- increasing disinfectant dose;
- adjusting process control;
- inspection and calibration of monitoring equipment;
- engagement of backup equipment;
- increased monitoring and observation.

Box 10.2 provides an example of the short-term evaluation of filtration performance and the corrective action that should be taken when the target criterion for turbidity has not been met.

***Box 10.2** Short-term evaluation of filtration performance*

For the filtration critical control point example detailed in Box 9.5, the target criterion for turbidity at each filter was set at <0.2 NTU and a critical limit for turbidity was set at 0.5 NTU.

If the turbidity from an individual filter has exceeded 0.2 NTU continuously for longer than the pre-determined delay period, an alarm should alert the operator that the target criterion has been breached and target filtration performance is not being achieved. The operator should promptly assess the filtration process and investigate the cause of the alarm. If the exceedance is during normal operation, immediate corrective actions should be implemented to achieve target performance. This may include:

- visual inspection of the filter to identify abnormalities;
- reviewing turbidity trends for all individual filters;
- confirming that upstream processes (e.g. coagulation) are operating normally;
- assessing raw water quality for unusual loadings;
- checking filter flow rates;
- manual backwashing of the filter; and
- reducing the hydraulic load on the filter.

If the exceedance of the target criteria is the result of a backwash event, the operator should keep the filter performance under close surveillance to confirm that plant operation returns to normal as expected, and ensure that the critical limit of 0.5 NTU is not breached. If an alarm indicates that the critical limit is exceeded, this should result in the filter being immediately taken off line until operation is satisfactorily back within specification.

After corrective action has been taken, its effectiveness needs to be verified. This usually entails additional monitoring. Secondary impacts of the corrective action, and the need for adjustments or additional action further along in the supply system, should also be considered.

Exceedance of, or deviation from, a target criterion at a critical control point would not generally require notification of the health regulator, provided the corrective action successfully prevents a breach of a critical limit.

Critical limit breach

Breaching of a critical limit indicates that control of a process has been lost, probably resulting in an unacceptable health risk. The health regulator should be notified without delay, corrective action should be taken immediately to resume control and normal operation of the process, and implementation of an emergency response plan should be considered. The emergency plan may include:

- plant shutdown;
- immediate collection and review of all relevant results (e.g. if filtered water turbidity exceeds limits, this should include source water quality operation of downstream disinfection plants);
- water diversion and/or reliance on an alternative supply;
- reduction in flow and the holding of unsafe water in pipelines for disposal;
- additional treatment elsewhere in the system (e.g. secondary disinfection, spot dose, booster disinfection);
- mains flushing, cleaning and localised disinfection;
- increased sampling and monitoring of relevant operational and drinking water quality characteristics downstream throughout the distribution system;
- implementation of a boil-water advisory in consultation with the relevant health regulator, if microbial contamination is suspected.

Critical operational processes with online, continuous monitoring of performance can be equipped with alarm systems set at critical limits which, when breached, trigger an automatic immediate shutdown of the treatment plant. This mitigates the risk of producing water with an unacceptable level of associated health risk (e.g. supply of undisinfected water) to consumers. Where possible, the water transfer system may also be shut down or diverted, to ensure that unsafe water is not supplied to consumers.

When any critical limit is breached, rapid response and investigation are essential to ensure that consumers' health is protected and supply is maintained. It may also be necessary to issue a public advisory, depending on available knowledge of the situation, the rapidity and effectiveness of the actions taken in response to the breach, and whether drinking water of unacceptable quality has been or will be supplied to consumers. This decision will be made in consultation with the relevant health regulator.

When the system is back under control, the root cause of the barrier breach should be investigated and improvements made, based on the outcome of the investigation (see Section 3.6.2).

Other operational monitoring – catchment to consumer

In addition to evaluating data at critical control points, results from other operational monitoring activities throughout the system should also be promptly reviewed against established target criteria and objectives, and previous results, to assess whether:

- the system is operating under expected normal conditions or there is an increase in the level of challenge;
- the preventive measures and barriers are performing effectively; and/or
- the monitoring results indicate a trend in performance that may be associated with:
  - poor maintenance;
  - insufficient backwashing;
  - clogging of filters;
  - increased chlorine demand; or
  - poor calibration of monitoring equipment.

Results from observational monitoring activities would also be assessed. Any reports of barrier breaches, such as damage to tank roofs, backflow or cross-connections, are significant and require immediate attention. Other observations of concern, such as increased human activity in a catchment, floc blanket in poor condition, "boiling" in filter beds when backwashing, reduction in chlorinator maintenance, or failure to meet targets for testing of backflow prevention devices, while they may not have an immediate impact on water quality, should nevertheless be addressed promptly to bring performance back to established requirements and target criteria.

The potential impact of poor performance or failure of an upstream barrier on the performance or integrity of downstream barriers should also be assessed.

10.2.2 SHORT-TERM EVALUATION OF DRINKING WATER QUALITY MONITORING

Water utilities should always aim to supply drinking water that complies with the health-related and aesthetic guideline values detailed in these Guidelines.

If the results of drinking water quality monitoring within the distribution system show that the water being supplied to consumers does not comply with the relevant health-related and/or aesthetic guideline values, then corrective action should be taken, as detailed in the following sections.

Evaluating short-term microbial quality

The short-term performance measure for microbial quality is detailed in Table 10.1.

Table 10.1 *Performance measure for* Escherichia coli *within the distribution system*

• *Escherichia coli (E. coli)* should not be detected in a minimum 100mL sample of drinking water.
• If detected, immediate corrective action must be taken

Water suppliers should take all reasonable actions to meet the guideline for *E. coli*, which is that *E. coli* should not be detected in a minimum 100 mL sample of drinking water. In practice, *E. coli* may occasionally be present in drinking water in the absence of any identifiable source of faecal contamination. Nevertheless, if samples taken are found to contain *E. coli*, the response to each detection should be rigorous:

- Action should be taken urgently to identify and rectify any barrier breaches, and ensure that all the barriers are working continually and the system is safe. This should include checking disinfectant residuals.
- Further samples should be collected to confirm the presence of ***E. coli*** and determine possible sources and distribution. This should include a repeat sample from the point where the non-conforming sample was collected and, as appropriate, an upstream sample (e.g. a service reservoir or system entry point) and a downstream or adjacent sample (e.g. a nearby sampling location).
- An investigation should be initiated immediately to identify the underlying cause(s) of any barrier breaches or unexplained results, and put in place corrective actions to prevent future faecal contamination and detection of ***E. coli.***
- Further sampling should be undertaken to verify that the corrective actions have been effective.
- All actions taken in relation to the detection should be documented.

If any of the repeat samples returns a positive result for *E. coli*, the response needs to be escalated. Depending on the circumstances, the escalation may involve:

- additional water treatment, including increased disinfection and spot dosing with chlorine;
- provision of an alternative water supply;

- issuing of a boil-water advisory (based on advice from, or done in consultation with, the relevant health regulator).

Additional, more widespread monitoring of the supply system should also be undertaken to determine the extent of contamination.

Procedures on reporting and responding to *E. coli* detections should be established with the relevant health regulator, and should be included in incident protocols. The procedures should include agreement on the requirements to be met before an incident is deemed to be closed.

Detection of *E. coli* in the drinking water system is a serious issue. At the conclusion of any incident, a debrief should be held to assess the problem and response, and agree to any short- and long-term actions needed to prevent a recurrence. Responsibility for undertaking those actions should be clearly established. If no identifiable source of contamination is determined, a long-term review of microbial system performance should be triggered to look for any emerging problems or trends.

Box 10.3 provides an example of a response protocol for *E. coli* detections.

***Box 10.3** Example response protocol for* E. coli *detections*

- Notification of the health regulator and immediate implementation of measures to render the water supply safe, as a priority. For chlorinated systems, establishing a free chlorine residual throughout the distribution system provides a high level of assurance that bacterial contamination will be inactivated. Actions that can be taken to increase residuals in the water supply system include increasing disinfection (e.g. chlorine dose rate), tank disinfectant dosing, mains flushing, and localised disinfection.
- Chlorine (or chloramine) residuals should be frequently monitored to provide assurance that this barrier to contamination is being continually maintained in the distribution system during the incident. This may be achieved by grab sampling or, preferably, installation of mobile chlorine monitors.
- A repeat sample for *E. coli* should be taken from the same sample point within 24 hours of the initial *E. coli* detection, to assess the effectiveness of remedial actions.
- Rapid investigation from catchment to tap to identify the contamination source or reason for the barrier breach. This includes gathering relevant information on water treatment performance and other operational data, including any consumer complaints, and initiating surveillance in the catchment/reservoirs, treatment plant and distribution system to assess any non-routine or unusual activities that may have occurred or are occurring.
- *E. coli* samples should also be concurrently taken from all other sample points within the supply zone, e.g. at the reservoir/tank outlet, downstream and adjacent points in the distribution. The purpose is either to confirm that the supply zone is free of contamination or to indicate the extent of any contamination.
- Outcomes of the repeat samples should be immediately reported to the health authority. If any repeat samples are positive, then further actions to protect public health will be determined.
- Two more sets of repeat samples from all sample points in the zone should be taken over the following week to provide assurance that the system has returned to operating within specification.
- Conduct a rapid investigation from catchment to tap to identify the contamination source or reason for the barrier breach. This includes gathering relevant information on water treatment performance and other operational data including any consumer complaints, and initiating surveillance in the catchment/reservoirs, treatment plant, and distribution system to assess for any past or present activities that are non-routine or unusual.
- Investigation should also include possibility of sample errors or sample contamination as a result of sampling conditions or transport, as well as laboratory quality assurance and possible analytical issues.
- Short-term corrective actions to eliminate any identified source of contamination or reasons for the positive result.

Close liaison should be maintained with the health regulator throughout the incident. The health regulator will determine the need to initiate emergency response plans, including issuing a public advisory, depending on the individual circumstances, the location of the sample and the investigation outcomes, and whether the sample represents a significant health threat to consumers.

Naegleria fowleri

An emerging issue in many jurisdictions is the detection of the free-living pathogenic amoeba *Naegleria fowleri*.

As free-living environmental organisms, *Naegleria* are not associated with faecal contamination and can be detected in the absence of *E. coli*. *N. fowleri* causes the waterborne disease primary amoebic meningoencephalitis (PAM). The route of infection is intra-nasal and PAM is associated with bathing rather than ingesting water.

Only *N. fowleri* has caused PAM. Other species of thermophilic *Naegleria*, however, may indicate the potential presence of *N. fowleri,* and detection of any *Naegleria* in drinking water should therefore be taken seriously, and corrective actions should be initiated while speciation is undertaken to determine if *N. fowleri* is present.

Naegleria are most likely to enter a water supply system at the source or at breaks in the sealed system such as open reservoirs and tanks. Under favourable conditions, they can proliferate in pipework and tanks. *Naegleria* can encyst and when in this state are more resistant to disinfection. Unless chlorine residual is continuous, the cysts are also able to survive in tank sediments and pipe biofilm.

Free chlorine or chloramine residual at 0.5 mg/L or higher will control *N. fowleri* provided the disinfectant residual persists throughout the water supply system at all times. A detection of thermophilic *Naegleria* in the treated water system should be taken seriously, as it indicates the potential presence of the pathogen *N. fowleri*, and that preventive measures and barriers have failed.

Box 10.4 provides an example of a response protocol to the detection of *Naegleria*.

Box 10.4 *Example response protocol for* Naegleria fowleri

A detection of thermophilic *Naegleria* in the treated water system should be taken seriously as it indicates the potential presence of the pathogen *N. fowleri*, and that preventive measures and barriers have failed.

Immediate response should include the following:

1. Notify the health authority and as a priority, take immediate action to render the water supply safe. To re-establish control of the water supply system, utilities should aim for free chlorine and chloramine residuals of at least 1 mg/L throughout the distribution system. Actions to increase residual in the water supply system include increasing disinfection (e.g. chlorine dose rate), tank disinfectant dosing, mains flushing, and localised dosing.
2. Check chlorine or chloramine residuals frequently to provide assurance that this barrier to contamination is being continuously maintained in the distribution system during the incident. This may be achieved by grab sampling or, where available, by installing mobile chlorine monitors.
3. Arrange speciation of the *Naegleria* if not provided with the original laboratory notification.
4. Take a repeat sample for *Naegleria* from the sample point to assess the effectiveness of remedial actions. *Naegleria* samples should also be taken concurrently from all other sample points within the supply zone (e.g. at the reservoir/tank outlet, downstream, and adjacent points in the distribution). The purpose is either to confirm that the supply zone is free of *Naegleria*, or to indicate the extent of the problem.
5. Investigate from catchment to tap to identify possible barrier breaches or contributing causes. This includes gathering relevant information on water treatment performance and other operational data such as consumer complaints of dirty water, burst mains, tank running empty, or flow reversals in water mains. Any disturbance of sediments increases the likelihood of *Naegleria* detection.
6. Communicate the results of the speciation immediately to the health authority. If *N. fowleri* is detected, consider further actions to protect public health, depending on the penetration and consistency of chlorine residuals in the distribution system.
7. Report outcomes of the repeat samples immediately to the health authority. If any of the repeat samples are positive, then further actions to protect public health will be determined.
8. Take another set of repeat samples from all sample points in the zone over the following week to provide assurance that the system has returned to operating within specification.
9. Take short-term corrective actions to eliminate any identified source of *Naegleria* or reasons for the positive result.

Close liaison should be maintained with the health authority throughout the incident. The health authority will determine the need to initiate emergency response plans, including issuing a public advisory.

At the conclusion of the incident, a debrief should be held to assess the problem and response and agree on any short and long-term actions needed to prevent a recurrence. Responsibility for undertaking those actions should be clearly established.

Actions that should be considered following a *Naegleria* detection include:

- Review operational limits for chlorine/chloramine residuals in the distribution system to ensure a minimum of 0.5 mg/L is maintained at all times;
- Review the frequency of cleaning to minimise sediment build-up in tanks and reservoirs;
- Introduce flushing and scouring programs to control sediment build-up in water mains;
- Install temporary chlorine boosters to ensure continuous residual above 0.5 mg/L;
- Modify system operation to reduce water age and stagnation to promote maintenance of chlorine residuals; and
- Take proactive measures to strengthen barriers when the challenge temporarily increases (e.g. increase chlorine residuals in the distribution system following a flow reversal event).

Evaluating short-term health-related chemical quality

The short-term performance measure for health-related chemical characteristics is detailed in Table 10.2.

Table 10.2 *Performance measure for health-related chemical characteristics within the distribution system*

- Chemical characteristics should not be detected in drinking water at concentrations above the relevant health-related guideline value.
- If a chemical characteristic is detected at a concentration above the relevant health-related guideline value, follow-up action must be taken.

Water suppliers should take all reasonable actions to ensure that drinking water does not contain any chemical characteristic in excess of a health-related guideline value, or an agreed level of service.

As described in Chapter 6, guideline values in the ADWG are generally rounded to a single significant figure. When interpreting monitoring results, water regulators and suppliers need to be aware that the guideline values are the results of rounding, and that the level of precision is one significant figure. When comparing a monitoring result with the guideline value the comparison should occur at the level of one significant figure. In determining compliance, state and territory water regulators can provide guidance.

Table 10.3 *Examples of comparing monitoring data to guideline values*

Guideline value	Monitoring data	Single significant figure of monitoring data	Consequence
2 mg/L	1.6 mg/L	2 mg/L	Does not exceed guideline.
1 mg/L	1.4 mg/L	1 mg/L	Does not exceed guideline.
1 mg/L	1.5 mg/L	2 mg/L	Exceeds guideline.
500 mg/L	547 mg/L	500 mg/L	Does not exceed guideline.
0.05 mg/L	0.0523 mg/L	0.05 mg/L	Does not exceed guideline.

With a few exceptions (e.g. nitrate, copper, sulfate, fluoride), all health-related guideline values relate to lifetime exposure, such that a single result above the guideline value is unlikely to present an immediate health risk. Nonetheless, each result above a health-related guideline value or an agreed level of service should be investigated to ensure that it does not pose any short-term acute effects or represent an emerging issue. Such results may at least indicate that a problem has occurred somewhere in the system with respect to barrier performance, and this should be investigated.

The recommended response to any detection of a chemical characteristic at concentrations above the relevant health-related guideline value is as follows:

- The detection is to be reported to the relevant health regulator, following established reporting protocols. Any health implications of the exceedance or non-conformity should be quickly assessed in relation to any short-term acute effects of the chemical in question, as this will influence the response.
- The water supply system should be inspected, and treatment records should be reviewed to ensure that if treatment barriers have been applied to manage the particular chemical characteristic (e.g. arsenic removal), they have not been compromised.
- Further sampling should be undertaken to verify the persistence and extent of the contamination. Sampling should also be undertaken to verify that corrective actions have been effective. The additional sampling should include a repeat sample from the point where the non-conforming sample was collected and, as appropriate, samples from source waters, upstream points (e.g. a service reservoir or system entry point) and a downstream or adjacent location (e.g. a nearby sampling point).
- All actions taken in relation to the detection should be documented.

A public advisory would not normally be required unless the concentrations found are so high that an acute health impact is possible. However, if any of the follow-up samples return a result above the relevant health-related guideline value, the issue needs to be discussed with the relevant health regulator. Depending on the circumstances, the discussions may result in:

- additional, more widespread monitoring of the supply system to improve understanding of the problem;
- operational changes to reduce the exposure;
- provision of an alternative water supply;
- longer-term improvements (e.g. additional treatment); and
- issuing of public advice.

Evaluating short-term aesthetic quality

The short-term performance measure for chemical and physical characteristics with aesthetic guideline values is detailed in Table 10.3.

Table 10.4 *Performance measure for aesthetic chemical and physical characteristics within the distribution system*

• Aesthetic chemical and physical characteristics should not be detected in drinking water at levels outside relevant aesthetic guideline values or agreed levels of service.
• If an aesthetic chemical or physical characteristic is detected at a level outside relevant aesthetic guideline values or agreed levels of service, follow-up action should be taken.

Whilst not presenting a health risk, the aesthetic guideline values ensure that drinking water is aesthetically pleasing and pleasant to drink. Many customers equate aesthetics with the safety of drinking water, so every effort should be made to meet the aesthetic guideline values.

The recommended response to any detection of a characteristic outside the relevant aesthetic guideline value is as follows:

- Inspect the water supply system and review treatment records to ensure that barriers have not been compromised.
- Undertake further sampling to verify the persistence and extent of the issue. Sampling should be from the point where the non-conforming sample was collected plus, as appropriate, an upstream sample (e.g. a service reservoir or system entry point) and a downstream or adjacent sample (e.g. a nearby sampling location).

It should be noted that some aesthetic characteristics, such as pH and turbidity, have an association with the safety of drinking water supplied as they affect treatment effectiveness.

Some water supply systems may consistently not meet an aesthetic guideline value because of the nature of the source water (e.g. high total dissolved solids). In these specific cases, investigating the reason for each elevated result is not recommended; rather, a normal operating limit or range should be established based on historical data. Any monitoring results outside these limits would then be assessed as unusual and would indicate a change in system operation that requires further investigation.

In many of the supplies where the nature of the source water results in ongoing non-compliance with an aesthetic guideline for a chemical or physical characteristic, an agreed level of service may be established with regulators and consumers. The agreed levels of service then become the values against which performance is measured.

10.3 Assessing performance: long-term evaluation of monitoring

The systematic review of monitoring results over an extended period of time (typically 12 months) is necessary to evaluate whether existing system management practices are effective in reducing risk, and to identify opportunities for improvement. The long-term evaluation of performance provides an essential feedback loop and comparison to the hazard identification and risk assessment process, and assists in corroborating or modifying assumptions and increasing system knowledge (see Figures 9.1 and 9.2).

The long-term evaluation of microbial, health-related chemical and aesthetic performance includes the assessment of all available monitoring information from catchment to consumer, including observational activities. The assessment should compare the monitoring information against objectives and criteria, such as guideline values or agreed levels of service.

Mechanisms should be developed for the long-term evaluation of microbial, health-related chemical and aesthetic performance. Useful tools to facilitate analysis of data sets include graphs, trend charts and, where appropriate, statistical evaluation.

The results from the long-term evaluation of performance should form an input to the senior management review (see Element 11 of the Framework, Section 3.11). Some aspects will be reported externally to consumers, stakeholders and regulatory authorities in accordance with requirements. It is an important due-diligence function that provides assurance that data are reviewed regularly and improvements are made in response to identified problems. Such assurance contributes to consumer and stakeholder confidence in the quality of the water being supplied and the actions being taken to manage the water supply system proactively.

10.3.1 LONG-TERM EVALUATION OF MICROBIAL PERFORMANCE

The long-term evaluation of microbial performance is a system-wide assessment that includes a performance evaluation of microbial monitoring data for the distribution system, including the point of supply to consumers, over a defined period, supplemented by all available operational monitoring data relevant to microbial system performance, from catchment to consumer. The purpose is to confirm the robustness of the system to deliver, reliably and continuously, drinking water that is free of microbial contamination.

Assessment of long-term microbial performance of the water supply system is undertaken to understand microbial challenges, and assess the effectiveness of preventive measures and barrier performance and whether they are being implemented appropriately. Any unacceptable increase in risk to consumers from changes in microbial challenge, barrier performance and/or system operation should be mitigated through short- and long-term improvements.

Information from any investigations, validation monitoring or incidents that may have occurred during the period of assessment should be included in the evaluation, for insights into problems and lessons learnt.

Where the long-term evaluation indicates deficiencies or opportunities to strengthen barriers to contamination, it is essential that short-term and long-term improvements are assessed, planned and implemented as a priority.

Review of microbial monitoring within the distribution system

Assessing the microbial quality of drinking water supplied to consumers over the long term is necessary to verify system performance and identify possible vulnerabilities in the water supply system or emerging trends of concern that may have gone unnoticed. This involves primarily an evaluation of *E. coli* monitoring data for the distribution system, including the point of supply to consumers.

As detailed in Section 10.2.3, the performance measure is that no *E. coli* should be detected in drinking water. Recognising the uncertainty attached to some *E. coli* results, the performance measure also

includes the requirement that corrective action be undertaken and documented in response to each detection, to prevent recurrences of potential faecal contamination.

E. coli results should be reported per water quality monitoring zone, detailing the number of samples collected, and the percentage of samples that were free of *E. coli* detections. The reporting statistics should not include results derived from repeat samples, or from emergency or investigative samples undertaken in response to detections.

In assessing and reporting on long-term microbial performance, the number of samples that contained *E. coli* should also be detailed. If *E. coli* was detected in any drinking water quality samples, the long-term review should report each investigation and the corrective actions taken.

Where a water quality monitoring zone records more than one sample positive for *E. coli* during the reporting period, a more detailed description of the actions taken to investigate and resolve the multiple detections should be prepared.

The rationale behind this reporting approach is that not all detections of *E. coli* can be directly linked to instances of faecal contamination, but all detections need to be investigated and appropriate remedial action taken.

A long-term target for *E. coli* may be determined in consultation with the relevant health regulator.

For water systems where drinking water temperatures in service tanks/reservoirs and the distribution system can consistently reach temperatures greater than 25ºC, the long-term evaluation of microbial performance should also include a similar review of *Naegleria* monitoring data.

For the long-term evaluation of microbial quality, it is important to assess concurrently *all* operational monitoring data relevant to microbial system performance, to provide assurance of the microbial data derived from water quality monitoring and confirm the robustness of the system to meet current and foreseeable challenges. Key aspects to include in this evaluation include performance of critical control points, source water, reservoir and catchment monitoring, and distribution system integrity monitoring.

Review of critical control point performance

The continuous operation of critical control points (CCPs), such as disinfection and filtration, within target criteria and critical limits is the single most important factor in ensuring the supply of water that is free of microbial pathogens.

Performance of CCPs over the long term should be assessed against the specified target criteria and critical limits by reviewing and reporting, for example:

- percentage of time, or volume of water supplied, where critical limit(s) were met;
- percentage of time, or volume of water supplied, where target criteria were met;
- number of alarms that occurred over the review period; and
- number of shut-downs to operation that occurred over the review period due to critical limit breaches.

Water suppliers should strive to supply water that has complied with critical limit criteria 100 per cent of the time. Where any performance objectives are not met, the review should try to understand the underlying nature of the problems. Any potential upstream and downstream factors that may be associated with objectives not being met should be considered. The review should confirm whether the apparent microbial challenge is within the design specification of the treatment process.

Any documented or anecdotal reports of malfunctions or incidents at CCPs will provide additional insights into problems and the actions required to improve performance. Review of CCP performance should also include the percentage of the planned routine preventive maintenance that has been undertaken, including instrument calibration.

Corrective actions to improve performance should then be planned, implemented, and monitored.

Box 10.5 provides an example of the long-term evaluation of filtration plant performance, which is an important part of the long-term evaluation of microbial performance of the system and the ongoing provision of safe drinking water.

Box 10.5 *Example of the long-term evaluation of filter performance*

Real-time turbidity monitoring is necessary to ensure that filtration is optimised and that the required pathogen removal is achieved at all times. In addition to routine review of filtration performance in the short-term to assess drinking water safety, the long-term review of filtration performance, over periods of months to years, is essential to demonstrate whether the barrier has operated effectively and continuously under a full range of challenges.

The assessment of filtration performance should include the percentage of time that target criteria and critical limits were met and whether any trends or problems can be identified. A cumulative frequency graph (Figure 10.5.1) or a frequency histogram (Figure 10.5.2) provide useful visual representations of filter performance over long time periods. Figure 10.5.1 indicates that for this particular filter, treated water met the turbidity critical limit of 0.5 NTU approximately 92% of the time, and the turbidity target criterion of 0.2 NTU approximately 65% of the time. A performance objective should be to achieve critical limits 100% of the time, while the target criterion may have some allowance for filter ripening after routine backwash events. In this example, however, the poor performance achieved from filtration, with the potential that unsafe water may have been supplied, would require that the operation of the filter be thoroughly examined and an improvement plan developed.

In conjunction with other microbial monitoring data from throughout the system, this filtration performance should be further evaluated to understand the causes of exceedances of established criteria and why corrective actions that were taken may not have been effective. Challenges from the source water, assessment of the design capabilities of the filtration plant, observational monitoring of the filters during backwash, operator training, instrument calibration, and preventive maintenance activities should also be reviewed to understand overall filtration performance, and identify improvements needed to meet current and foreseeable changes to challenges.

In Figure 10.5.2, turbidity data are compared to those from a previous year. The shift of the bars to the left in 2007 indicates improved operation and performance of the filter over time. The presentation of data in these formats provides a clear assessment of filtration performance over the long term; it allows filter problems to be identified early and provides or an early indication of deteriorating filter performance (Mosse and Murray 2008).

Figure 10.5.1 *Cumulative frequency graph of filtered water turbidity*

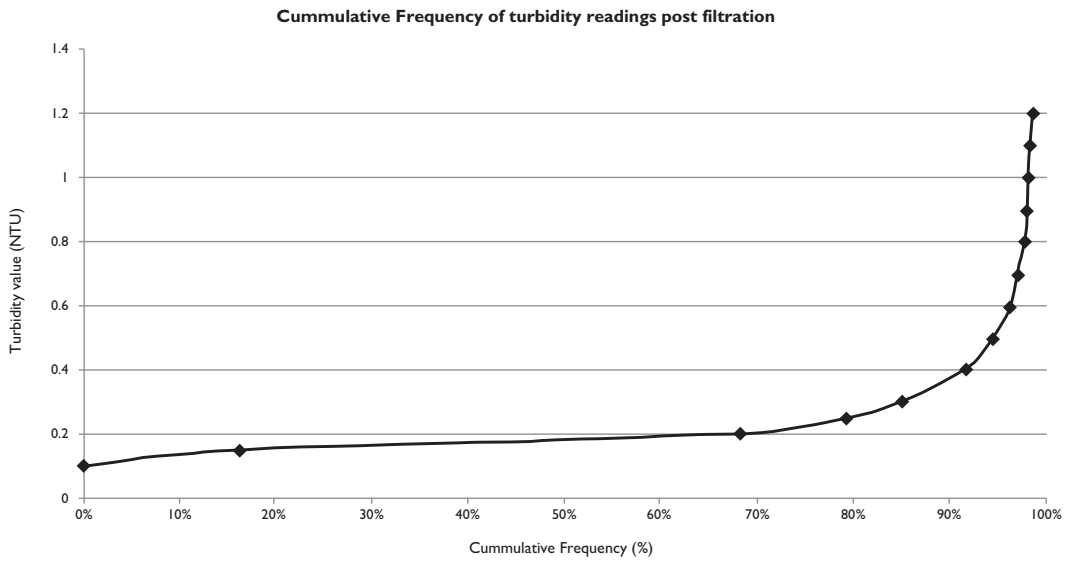
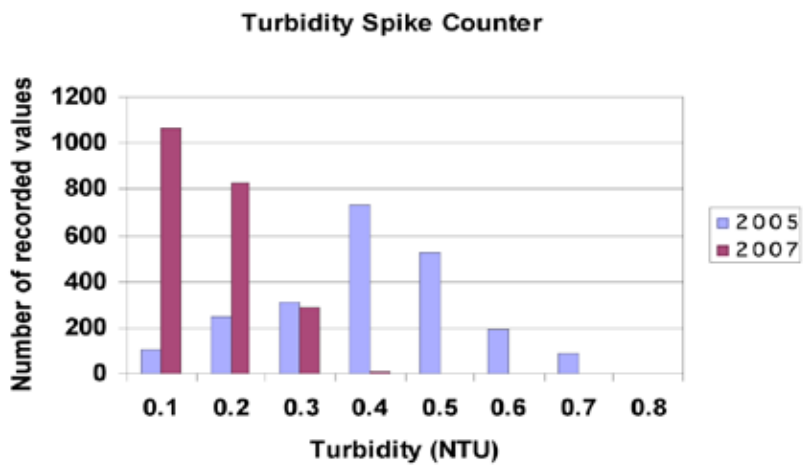


Figure 10.5.2 *Turbidity histogram*



Review of other operational monitoring – catchment to consumer

The long-term evaluation of microbial performance should also include a review of all other operational monitoring undertaken throughout the system. Source water (and reservoir) monitoring data should be reviewed to assess any changes in the microbial challenge to the water supply system and whether any trends of concern can be determined. Any significant hydrological or other events should be reviewed to assess any associations that may relevant to system management (e.g. the response of turbidity and *E. coli* to rainfall events; the response of algae to increases in reservoir water temperatures).

Observational and investigative monitoring results from the catchment area should be reviewed to provide insight into changes in the level of challenge/risk (e.g. from changes in land use, recreation, climatic and natural events). Review of incident reports can also highlight any new hazards and problems, and should be incorporated in the review.

The long-term evaluation of microbial performance is supported by reviewing monitoring activities in the distribution system. Chlorine residual performance in the distribution system provides useful information on potential problems and risks that may not have been revealed through microbial monitoring. Hence, chlorine residual penetration and consistency should be evaluated and any areas of concern identified. System integrity should be verified by reviewing preventive maintenance activities, as well as any monitoring and inspection records undertaken during the period.

Risk mitigation measures or system management improvements identified from the long-term evaluation of microbial performance should be planned and implemented as necessary.

10.3.2 LONG-TERM EVALUATION OF HEALTH-RELATED CHEMICAL PERFORMANCE

The long-term evaluation of health-related chemical performance is a system-wide assessment that includes evaluation, against guideline values, of chemical monitoring data for the distribution system, including the point of supply to consumers over a period, supplemented by all available operational, investigation and validation monitoring data from catchment to consumer.

The objective of the long-term evaluation is to understand system performance and confirm the robustness of the system to deliver drinking water reliably with concentrations of chemicals below the recommended maximum concentrations. The long-term evaluation of chemical performance should inform any short- and long-term actions required to improve the management of risk.

Review of health-related chemical monitoring within the distribution system

In assessing and reporting long-term chemical performance against health-related guideline values, the number of monitoring results available for the reporting period needs to be considered.

In most cases, monitoring of chemical characteristics is monthly, although in some cases it may only be quarterly. Therefore, there may only be between 4 and 12 data points available to assess system performance.

Additionally, there is strong evidence that water quality data is not normally distributed, therefore it is not appropriate to use statistics that are based on the assumption of normality. More information on the statistical issues can be found in *Information Sheet 3.3 - Statistics – Statistical Principles*.

In assessing and reporting long-term chemical performance against health-related guideline values, monitoring results for each chemical characteristic should be assessed and reported per water quality monitoring zone, listing the number of samples collected per zone, the minimum, maximum and mean result, and the number of results above the health-related guideline value.

Given that generally few data points will be available for evaluation, performance against the guideline value is determined based on the maximum result.

If a longer data set is being evaluated, or there are sufficient sample results from the 12 month review period to support a statistically valid evaluation of the results, then the 95th percentile statistic should also be listed and used to determine performance against the guideline value. *Information Sheet 3.5 – Number of Samples Required* provides advice on the number of samples required for a statistically valid evaluation. Advice from a statistician could also be sought.

The reporting statistics should not include results derived from repeat samples, or from emergency or investigative samples undertaken in response to an elevated result.

Monitoring data that are to be averaged or otherwise numerically adjusted should not be rounded prior to averaging or other manipulations[5]. Comparing monitoring data with a guideline value is described in Table 10.3, Section 10.2.2.

Any investigations and corrective actions arising from individual results that exceed a health-related guideline value should be incorporated into the long-term review and reported, to ensure that the cause of the exceedances is understood and appropriate actions have been taken or planned to prevent a recurrence. Where these investigations indicate that the system lacks robustness to provide confidence that exceedances will not recur, actions to improve system performance should be evaluated.

If a health-related guideline value is exceeded or regularly close to being exceeded, this may indicate a system deficiency, and the drinking water supplier needs to determine what is required to meet the guideline values and agreed levels of service consistently. Operational corrective actions to reduce the risk of health-related chemical exceedance should be implemented as quickly as practicable. Where corrective actions include major capital works, an acceptable schedule should be agreed in consultation with the health authority and/or other stakeholders, based on the risk to public health.

Review of operational monitoring – catchment to consumer

In Australian drinking water systems, many of the chemicals listed in these Guidelines are either not present or present at concentrations that are stable and sufficiently low that the risk of exceeding the health-related guideline value is very low. In these cases, the long-term evaluation of chemical performance consists primarily of confirming and reporting concentrations against the relevant guideline value.

5 Water suppliers may wish to retain monitoring data in its original state in order to permit future calculations.

There are likely to be a small number of chemicals that are consistently close to the guideline value, that exhibit high variability, or for which a control, such as treatment, has been implemented to manage the hazard. For these chemical characteristics, the long-term evaluation should incorporate review of any associated operational monitoring data throughout the system, to enhance system understanding and confirm the reliability of delivering drinking water that meets the specified requirements.

Depending on the chemical characteristic, relevant operational monitoring could include, for example:

- observational catchment monitoring, such as observation of pesticide and fertiliser application;
- events such as bushfires, which may increase organic load in source water, and subsequently, THMs in the distribution system;
- operational characteristics related to the performance of any treatment process used to remove a specific chemical (e.g. nitrate or arsenic removal); or
- operational characteristics relevant to the management of microbial quality that may, indirectly, have an impact on chemical quality (e.g. increasing chlorine dose to deal with higher source water turbidity resulting in elevated THMs).

For any chemical characteristics where there is risk of exceeding specified guideline values, it may be useful to establish target criteria or an operational limit at some value below the guideline value to provide warning and trigger prompt investigation before a health-related chemical exceedance occurs. Box 10.6 provides an example illustrating how these principles may be used to manage THMs concentrations in the distribution system.

The long-term evaluation of chemical performance, with inputs from drinking water quality monitoring data and any associated operational data, should increase understanding of overall system performance and provide input to any short- and long-term improvements to improve the management of any risk of exceeding health-related guideline values.

Box 10.6 *Assessing system performance for management of THMs*

Setting target criteria

The health-related guideline value for THMs at the point of supply to consumers is 0.250 mg/L. To ensure that the guideline value is not exceeded, an operational target for THM concentration in the distribution system can be set below this level, for example at 0.175 mg/L. If a drinking water quality result greater than 0.175 mg/L is obtained, then this can trigger a more detailed evaluation of the sample result and possible corrective action.

Short-term evaluation and response

If, for example, a drinking water quality monitoring result of 0.200 mg/L for THMs is obtained, the operational target is exceeded, and this will trigger a response. The first action may be to increase sampling frequency for a period to obtain a more comprehensive understanding of THM concentrations in the distribution system. If results are consistently above the target criterion, then further investigation is warranted to determine the THM formation potential of the source and treated water, and the relationship between chlorine dose and THM formation.

Operators can then set a maximum chlorine dose rate to avoid THM exceedances, as long as the dose rate does not compromise effective disinfection. This chlorine dose rate is then the operating target for disinfection. If the source water quality is highly variable, however, it may be necessary also to establish a surrogate for the organic content of the source water (e.g. UV absorbance), with target criteria and routine monitoring of this characteristic in the source water.

Long-term evaluation of THM performance

The objective of the long-term health-related chemical evaluation is to determine whether the water supply system can consistently deliver water that meets specified requirements. As one aspect of this long-term review, the long-term evaluation of THM monitoring data used in this example would involve reviewing:

- drinking water quality monitoring for guideline and target criterion exceedances;
- THM formation potential of source water and the likelihood of this changing in the future;
- whether the recommended maximum chlorine dose was adversely affecting disinfection or chlorine residuals in the distribution system, creating other health risks.

If the long-term evaluation concludes that the risk of THM exceedances is unacceptably high, then improvements should be considered. Considered first are operational options such as source water blending, lowering tank levels to reduce water age, or not using the source water when risk of THM formation is at its greatest. Alternatively, capital solutions may be required such as tank aeration or treatment to remove organics.

10.3.3 LONG-TERM EVALUATION OF AESTHETIC PERFORMANCE

The long-term evaluation of aesthetic performance is a system-wide assessment that includes evaluation of monitoring data for the distribution system, including the point of supply to consumers over a period against aesthetic guideline values, supplemented by all available operational, investigation and validation monitoring data from catchment to consumer.

The objective of the long-term evaluation is to understand system performance and confirm the robustness of the system to reliably deliver drinking water with characteristics that meet the physical and non-health-related chemical guideline values and/or requirements agreed with customers and other stakeholders. In addition, it is necessary to confirm that aesthetic water quality is not having an adverse impact on health-related system performance.

Review of aesthetic quality monitoring within the distribution system

When assessing and reporting the long-term performance of characteristics against aesthetic guideline values, each characteristic should be reported per water quality monitoring zone, listing the number of samples collected per zone, the minimum, maximum and mean result, and the number of results outside the relevant aesthetic guideline value. Any corrective actions that have been taken should also be listed.

The mean of the previous 12 months' monitoring results should be compared to the aesthetic guideline

value. If the mean for any aesthetic characteristic falls outside the guideline value, it should be further investigated to understand the reason for this outcome, and identify any trends that may be developing and the actions required to prevent recurrences. In such cases, investigative studies and additional monitoring may be required to improve characterisation of the water quality.

Review of operational monitoring – catchment to consumer

To ensure and/or improve aesthetic water quality performance and assess possible health impacts, review of operational monitoring data from catchment to consumer is required.

An important consideration in long-term evaluation is to assess if the overall performance of any aesthetic characteristic is likely to be associated with an adverse effect on the safety of the water supplied to customers (e.g. pH can affect chlorination effectiveness and the corrosiveness of the water). Typically this requires evaluating the aesthetic monitoring data in conjunction with other data, such as microbial monitoring data. If the safety of drinking water is unacceptably affected, then corrective actions should be included in the water quality improvement plan. If there is uncertainty about whether these characteristics are affecting system performance, additional operational monitoring should be planned.

The review should also assess the acceptability of the aesthetic quality of water received by consumers, taking into account that most non-health-related characteristics relate to the palatability and amenity of the water and, as such, that there is a degree of subjectivity involved in setting the guideline values for these characteristics. Where there is ongoing non-conformance with aesthetic guideline values, water suppliers should review customer complaints and consider consulting with customers to determine the impact.

Consumers notice and do not appreciate large variations in aesthetic water quality. Drinking water supplies subject to large variations in aesthetic quality should be assessed to identify the actions or investments needed to achieve greater consistency (e.g. selection of bore combinations, improved treatment). The decision to proceed with any aesthetic water quality improvements will depend on a number of factors including cost, technical difficulty, operator capability, and consumer feedback and willingness to pay for improvements. Where aesthetic quality improvements are programmed, progress on the improvements and their effectiveness should be reported.

10.3.4 LONG-TERM EVALUATION OF CONSUMER SATISFACTION

From the consumer's point of view, changes from the norm are particularly noticeable. At present, there are no guidelines for consumers' overall impressions or perceptions of physical water quality. Furthermore, consumer satisfaction may have a regional or even local context, and it needs to be assessed at this level.

The objective of the long-term evaluation of consumer satisfaction is to confirm that the complaint handling system is effective for picking up complaints, and particularly any clusters of complaints, related to water quality, and that action plans are adequate and linked suitably to operations.

Drinking water quality complaints by type (e.g. alleged illness, taste and odour, dirty water, stained laundry) should be reported over the period and evaluated against any internal or external performance targets (e.g. fewer than x complaints per 1000 households per year), noting any potential trends of concern. Investigations and response actions should be reviewed to ensure the actions were satisfactory, particularly with respect to any complaints of alleged illness, and that staff are adequately trained to respond effectively.

The long-term evaluation should include any reports of routine or one-off surveys of customer satisfaction and/or attitudes. Experience from major water suppliers indicates that consumer satisfaction has the following characteristics:

- Complaints and concerns about 'healthiness' are driven more by sudden noticeable changes in quality, particularly in taste, odour, colour and turbidity, than by the long-term average.
- Taste and odour associated with disinfectants are tolerated up to a point because they are associated with the protection of public health, although concerns sometimes arise about the health effects of added chemicals.
- It is unrealistic to expect to achieve complete satisfaction. For example, it is unlikely that more than 90 per cent of consumers will give a 'good to excellent' rating on taste and odour.
- The bulk of consumer complaints relate to taste and odour, discolouration and stained washing, many of which can stem from household plumbing or may be very localised. It is the unusual complaints such as the fishy smell generated by the presence of certain algae in source water, or blue discolouration due to corrosion of the consumer's copper service pipes, that may have wider implications for the water supply system, and these require immediate attention. In the event of blue discolouration linked to copper corrosion, a more detailed investigation should be undertaken to determine if the copper corrosion is widespread, or related to characteristics of the water being supplied.

Where programs are in place to improve consumer satisfaction or awareness, these should be evaluated in the long-term review for progress and effectiveness.

10.3.5 IMPROVEMENT PLAN

Any actions identified in the long-term review of performance that are needed to improve system management and overall drinking water safety and consumer satisfaction should be documented and incorporated into an improvement plan. Actions may apply in the short-term (e.g. enhanced mains flushing programs, provision of alarms on critical control points), or may be longer-term capital works projects (e.g. covering water storages, upgrading treatment).

The implementation of corrective and preventive actions will often have significant budgetary implications and may therefore require detailed evaluation and careful priority setting. Implemented actions should be documented and methods for monitoring the improvements should be developed, carried out, and subsequently reviewed for overall effectiveness and improvement.

10.3.6 PERFORMANCE REPORTING

Performance assessment, based on the long-term review of monitoring data, should be reported internally to relevant staff and departments, as well as to senior management.

Performance reporting on water supply systems is also an important issue for health and regulatory authorities, and for consumers. Providing assurance that performance is reviewed regularly and that improvements are made in response to identified problems contributes to confidence in the water supplied and the water supply organisation. External reporting ensures that system management and drinking water quality performance remains open and transparent. External reporting may be done through an annual report, the contents of which may be determined by a regulatory agency.

10.3.7 SUMMARY OF GUIDELINE VALUES FOR MICROBIAL, CHEMICAL AND PHYSICAL AND CHARACTERISTICS

Tables 10.4 and 10.5 summarise of the guideline values for microbial, chemical and physical and characteristics, to provide a ready reference when monitoring results are being evaluated. More detailed information on each characteristic can be found in the relevant fact sheet.

Table 10.5 *Performance measure for* Escherichia coli *within the distribution system*

- *Escherichia coli (E. coli)* should not be detected in a minimum 100 mL sample of drinking water.
- If detected, immediate corrective action must be taken

Table 10.6 *Guideline values for physical and chemical characteristics*

Characteristic	Guideline values (mg/L unless otherwise specified)		Comments
	Health	**Aesthetic**	
Acephate	0.008		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Acrylamide	0.0002		Minor impurity of polyacrylamide, used sometimes as a flocculant aid.
Aldicarb	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Aldrin & Dieldrin	0.0003 (combined)		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Aluminium (acid-soluble)	c	0.2	Guideline value based on post-flocculation problems; < 0.1 mg/L desirable. Lower levels needed for renal dialysis. No health-based guideline value can be established currently.
Ametryn	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Amitraz	0.009		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Amitrole	0.009		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Ammonia (as NH3)	c	0.5	Presence may indicate sewage contamination and/or microbial activity. High levels may corrode copper pipes and fittings.
Antimony	0.003		Exposure may rise with increasing use of antimony–tin solder.
Arsenic	0.01		From natural sources and mining/industrial/agricultural wastes.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Asbestos	c		From dissolution of minerals/industrial waste, deterioration of asbestos-cement pipes in distribution systems. No evidence of cancer when ingested (unlike inhaled asbestos).
Asulam	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Atrazine	0.02		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Azinphos-methyl	0.03		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Barium	2		Primarily from natural sources.
Benomyl	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns
Bentazone	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Benzene	0.001		Could occur in drinking water from atmospheric deposition (motor vehicle emissions) and chemical plant effluent. Human carcinogen.
Beryllium	0.06		From weathering of rocks, atmospheric deposition (burning of fossil fuels) discharges.
Bioresmethrin	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Boron	4		From natural leaching of minerals and contamination. <1 mg/L in uncontaminated sources; higher levels may be associated with seawater intrusion.
Bromacil	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Bromate	0.02		Possible by-product of disinfection using ozone, otherwise unlikely to be found in drinking water.
Bromophos-ethyl	0.01 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Bromoxynil	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Cadmium	0.002		Indicates industrial or agricultural contamination; from impurities in galvanised (zinc) fittings, solders and brasses.
Captan	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Carbaryl	0.03		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Carbendazim	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Carfentrazone-ethyl	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Carbofuran	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Carbon tetrachloride	0.003		Sometimes occurs as impurity in chlorine used for disinfection (it is not a disinfection by-product).
Carbophenothion	0.0005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Carboxin	0.3		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chloral hydrate (Trichloroacetaldehyde)	0.1 e		By-product of chlorination. Action to reduce chloral hydrate is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chloral hydrate.
Chloramine — see monochloramine			
Chlorantraniliprole	6		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chlorate	c e		By-product of chlorination. Insufficient data to set a health-related guideline value.
Chlordane	0.002		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chlorfenvinphos	0.002		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chloride	c	250	From natural mineral salts, effluent contamination. High concentrations more common in groundwater and certain catchments.
Chlorinated furanones (MX)	c e		By-product of chlorination. Insufficient data to set a health-related guideline value.
Chlorine	5	0.6	Widely used to disinfect water, and this can produce (free) chlorinated organic by-products. Odour threshold generally 0.6 mg/L, but 0.2 mg/L for a few people. In some supplies it may be necessary to exceed the aesthetic guideline in order to maintain an effective disinfectant residual throughout the system.
Chlorine dioxide	c	0.4	Oxidising agent and disinfectant in water treatment.
Chlorite	0.8		By-product of chlorine dioxide disinfection. Action to reduce chlorite is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chlorite.
Chloroacetic acids chloroacetic acid dichloroacetic acid trichloroacetic acid	e 0.15 0.1 0.1		By-product of chlorination. Action to reduce chloroacetic acids is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chloroacetic acids.
Chlorobenzene	0.3	0.01	Could occur in drinking water from spills or discharges. Taste/odour threshold (0.01 mg/L) is well below health level.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Chloroketones	e		By-product of chlorination.
1,1-dichloropropanone	c		
1,3-dichloropropanone	c		
1,1,1-trichloropropanone	c		
1,1,3-trichloropropanone	c		
Chlorophenols	e		By-product of chlorination of water containing phenol or related chemicals. Action to reduce chlorophenols is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chlorophenols.
2-chlorophenol	0.3	0.0001	
2,4-dichlorophenol	0.2	0.0003	
2,4,6-trichlorophenol	0.02	0.002	
Chloropicrin	c		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns. Data are inadequate to set a health-based guideline.
Chlorothalonil	0.05		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chloroxuron	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chlorpyrifos	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chlorsulfuron	0.2		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Chromium (as Cr(VI))	0.05		From industrial/agricultural contamination of raw water or corrosion of materials in distribution system/plumbing. If guideline value exceeded, analyse for hexavalent chromium.
Clopyralid	2		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Colour (true)		15 HU	An important aesthetic characteristic for customer acceptance. Treatment processes can be optimised to remove colour.
Copper	2	1	From corrosion of pipes/fittings by salt, low pH water. Taste threshold 3 mg/L. High concentrations colour water blue/green. >1 mg/L may stain fitings. >2 mg/L can cause ill effects in some people.
Cyanide	0.08		From industrial waste and some plants and bacteria.
Cyanogen chloride (as cyanide)	0.08		By-product of chloramination. Action to reduce cyanogen chloride is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than cyanogen chloride.
Cyfluthrin, Beta-cyfluthrin	0.05		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Cypermethrin isomers	0.2		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Cyprodinil	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
2,4-D [(2,4-Dichlorophenoxy) acetic acid]	0.03		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
DDT	0.009		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Deltamethrin	0.04		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Diazinon	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dicamba	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dichlobenil	0.01 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dichlorobenzenes			
1,2-dichlorobenzene	1.5	0.001	Could occur in drinking water from spills, discharges, atmospheric deposition, leaching from contaminated soils. Health levels are well above offensive taste/odour thresholds.
1,3-dichlorobenzene	c	0.02	
1,4-dichlorobenzene	0.04	0.0003	
Dichloroethanes			
1,1-dichloroethane	c		Could occur in drinking water from industrial effluents, spills, discharges.
1,2-dichloroethane	0.003		
Dichloroethenes			Rarely found in drinking water; found occasionally in groundwater from wells heavily contaminated by solvents.
1,1-dichloroethene	0.03		
1,2-dichloroethene	0.06		
Dichloromethane (methylene chloride)	0.004		Widely used solvent, commonly found in ground and surface waters overseas. Volatilises from surface waters and biodegrades in the atmosphere.
1,3-Dichloropropene	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dichloroprop / Dichlorprop-P	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dichlorvos	0.005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Diclofop-methyl	0.005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dicofol	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dieldrin see Aldrin			
Difenzoquat	0.1 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Diflubenzuron	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dimethoate	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified)		Comments
	Health	**Aesthetic**	
Diphenamid	0.3 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Diquat	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Dissolved oxygen	Not necessary	>85%	Low concentrations allow growth of nuisance microorganisms (iron/manganese/sulfate/nitrate-reducing bacteria), causing taste and odour problems, staining, corrosion. Low oxygen concentrations are normal in groundwater supplies and the guideline value may not be achievable.
Disulfoton	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Diuron	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
2,2-DPA	0.5		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
EDB	0.001 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Endosulfan	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Endothal	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Epichlorohydrin	0.0005d		Used in manufacture of some resins used in water treatment.
EPTC	0.3		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Esfenvalerate	0.03		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Ethion	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Ethoprophos	0.001		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Ethylbenzene	0.3	0.003	Natural component of petrol and petroleum products.
Ethylenediamine tetraacetic acid (EDTA)	0.25		Metal-complexing agent widely used in industry and agriculture, and as a drug in chelation therapy.
Etridiazole	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenamiphos	0.0005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenarimol	0.04		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenchlorphos	c		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenitrothion	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Fenoprop	0.01 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fensulfothion	0.01 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenthion	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fenvalerate	0.06		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fipronil	0.0007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Flamprop-methyl	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fluometuron	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fluoride	1.5		Occurs naturally in some water from fluoride-containing rocks. Often added at up to 1 mg/L to protect against dental caries. >1.5 mg/L can cause dental fluorosis. >4 mg/L can cause skeletal fluorosis.
Fluproponate	0.009		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Formaldehyde	0.5		By-product of ozonation.
Formothion	0.05 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Fosamine	0.03 f		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Glyphosate	1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Haloacetonitriles dichloroacetonitrile trichloroacetonitrile dibromoacetonitrile bromochloroacetonitrile	e c c c c		By-product of chlorination.
Haloxyfop	0.001		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Hardness (as $CaCO_3$)	Not necessary	200	Caused by calcium and magnesium salts. Hard water is difficult to lather. <60 mg/L $CaCO_3$ – soft but possibly corrosive. 60-200 mg/L $CaCO_3$ – good quality. 200-500 mg/L $CaCO_3$ – increasing scaling problems. >500 mg/L $CaCO_3$ – severe scaling.
Heptachlor	0.0003		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Hexachlorobutadiene	0.0007		Industrial solvent.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Hexaflurate	0.03 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Hexazinone	0.4		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Hydrogen sulfide	c	0.05	Formed in water by sulfate-reducing microorganisms or hydrolysis of soluble sulfide under anoxic conditions. Obnoxious 'rotten egg' odour, threshold 0.05 mg/L.
Imazapyr	9		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Iodide	0.5		From mineral and salt deposits.
Iodine	c		Can be used as an emergency water disinfectant. Taste threshold 0.15 mg/L.
Iprodione	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Iron	c	0.3	Occurs naturally in water, usually at <1 mg/L, but up to 100 mg/L in oxygen-depleted groundwater. Taste threshold 0.3 mg/L. High concentrations stain laundry and fittings. Iron bacteria cause blockages, taste/odour, corrosion.
Lanthanum	0.002		Rare earth element. Occurs naturally in water from rock weathering. Other sources include use as a phosphate binder to reduce algal blooms in water reservoirs, an agriculture fertiliser or leaching from the tailings of rare earth mining.
Lead	0.01		Occurs in water via dissolution from natural sources or household plumbing containing lead (e.g. pipes, solder).
Lindane	0.01		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Maldison (Malathion)	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Mancozeb	for ETU: 0.009		Mancozeb degrades in the environment to ethylene thiourea (ETU), hence the health-based guideline is based on the toxicity of ETU. Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Manganese	0.5	0.1	Occurs naturally in water; low in surface water, higher in oxygen-depleted water (e.g. groundwater at bottom of deep storages). >0.1 mg/L causes taste, staining. <0.05 mg/L desirable.
MCPA	0.04		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Mercury	0.001		From industrial emissions/spills. Very low concentrations occur naturally. Organic forms most toxic, but these are associated with biota, not water.
Metaldehyde	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Metham	for MTIC: 0.001		Metham degrades to methylisothiocyanate (MITC) in the environment, hence the health-based guideline is based on the toxicity of MITC. Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Methidathion	0.006		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Methiocarb	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Methomyl	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Methoxychlor	0.3 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Methyl bromide	0.001		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Metiram	for ETU: 0.009		Metiram degrades in the environment to ethylene thiourea (ETU), hence the health-based guideline is based on the toxicity of ETU. Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Metolachlor/s–Metolachlor	0.3		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Metribuzin	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns
Metsulfuron-methyl	0.04		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Mevinphos	0.005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Microcystins	1.3 µg/L		Hepatotoxic peptide produced by a range of cyanobacteria, expressed as microcystin-LR toxicity equivalents.
Molinate	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Molybdenum	0.05		Concentrations usually <0.01 mg/L; higher concentrations from mining, agriculture, or fly-ash deposits from coal-fuelled power stations.
Monochloramine	3		Used as water disinfectant. Odour threshold 0.5 mg/L.
Monocrotophos	0.002 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Naphthalophos	c		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns. No value set, as the health concerns have not been fully evaluated.
Napropamide	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Nicarbazin	1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Nickel	0.02		Concentrations usually very low; but up to 0.5 mg/L reported after prolonged contact of water with nickel-plated fittings.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Nitralin	0.5 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Nitrate (as nitrate)	50		Occurs naturally. Increasing in some waters (particularly groundwater) from intensive farming and sewage effluent. Guideline value will protect bottle-fed infants under 3 months from methaemoglobinaemia. Adults and children over 3 months can safely drink water with up to 100 mg/L nitrate.
Nitrilotriacetic acid	0.2		Chelating agent in laundry detergents (replacing phosphate). May enter water through sewage contamination.
Nitrite (as nitrite)	3		Rapidly oxidised to nitrate (see above).
N-Nitrosodimethylamine (NDMA)	0.0001 mg/L (100 ng/L)		By-product of chloramination and to a lesser extent chlorination. Action to reduce N-Nitrosodimethylamine is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than N-Nitrosodimethylamine
Norflurazon	0.05		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Omethoate	0.001		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Organotins dialkyltins tributyltin oxide	 c 0.001		Stabilisers in plastics. May leach from new polyvinyl chloride (PVC) pipes for a short time. Tributyltins are biocides used as antifouling agents on boats and in boiler waters.
Oryzalin	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Oxamyl	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns (for further information, see Information Sheet 1.6).
Ozone			As ozone used for disinfection leaves no residual, no guideline value or fact sheet has been provided.
Paraquat	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Parathion	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Parathion-methyl	0.0007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pebulate	0.03		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pendimethalin	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pentachlorophenol	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Permethrin	0.2		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
pH	c	pH 6.5–8.5	While extreme pH values (<4 and >11) may adversely affect health, there are insufficient data to set a health guideline value. <6.5 may be corrosive. >8 progressively decreases efficiency of chlorination. >8.5 may cause scale and taste problems. New concrete tanks and cement-mortar lined pipes can significantly increase pH and a value up to 9.2 may be tolerated provided monitoring indicates no deterioration in microbial quality.
Picloram	0.3		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Piperonyl butoxide	0.6		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pirimicarb	0.007		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Pirimiphos-ethyl	0.0005 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pirimiphos methyl	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Plasticisers di(2-ethylhexyl) phthalate di(2-ethylhexyl) adipate	0.01 c		Used in all flexible PVC products, and may leach from these over a long time. Could also occur in drinking water from spills.
Polihexanide	0.7		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Polycyclic aromatic hydrocarbons (PAHs) Benzo-(a)-pyrene	0.00001 (10 ng/L)		Widespread. Contamination can occur through atmospheric deposition, or leaching from bituminous linings in distribution systems.
Profenofos	0.0003		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Promecarb	c		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propachlor	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propanil	0.7		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propargite	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propazine	0.05		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propiconazole	0.1		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Propyzamide	0.07		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pyrasulfotole	0.04		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Pyrazophos	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Pyroxsulam	4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Quintozene	0.03		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Selenium	0.01		Generally very low concentrations in natural water..
Silica		80	An important characteristic for both aesthetics and treatment processes. Can form films on glass and can also affect reverse osmosis.
Silver	0.1		Concentrations generally very low. Silver and silver salts occasionally used for disinfection.
Simazine	0.02		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns.
Sodium	Not necessary	180	Natural component of water. Guideline value is taste threshold.
Spirotetramat	0.2		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns
Styrene (vinylbenzene)	0.03	0.004	Could occur in drinking water from industrial contamination.
Sulfate	c	250	Natural component of water, and may be added via treatment chemicals. Guideline value is taste threshold. >500 mg/L can have purgative effects.
Sulprofos	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns
2,4,5-T	0.1 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns
Taste and odour	Not necessary	Not offensive to most people	May indicate undesirable contaminants, but usually indicate problems such as algal or biofilm growths.
Temephos	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Temperature	Not necessary	No value set	Generally impractical to control; rapid changes can bring complaints.
Terbacil	0.2		Pesticide, has occasionally been reported in Australian drinking waters, but unlikely to be found at levels that may cause health concerns
Terbufos	0.0009		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Terbuthylazine	0.01		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Terbutryn	0.4		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Tetrachloroethene	0.05		Dry-cleaning solvent and metal degreaser. Could occur in drinking water from contamination or spills.
Tetrachlorvinphos	0.1 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified) Health	Aesthetic	Comments
Thiobencarb	0.04		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Thiometon	0.004 f		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Thiophanate	0.005		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Thiram	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Tin	Not necessary		Concentrations in water very low; one of the least toxic metals.
Toltrazuril	0.004		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Toluene	0.8	0.025	Occurs naturally in petrol and natural gas, forest-fire emissions. Could occur in drinking water from atmospheric deposition, industrial contamination, leaching from protective coatings in storage tanks.
Total dissolved solids	Not necessary	600	Based on taste: <600 mg/L is regarded as good quality drinking water. 600-900 mg/L is regarded as fair quality 900-1200 mg/L is regarded as poor quality >1200 mg/L is regarded as unacceptable.
Triadimefon	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Trichlorfon	0.007		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Trichlorobenzenes (total)	0.03	0.005	Industrial chemical.
1,1,1-Trichloroethane	c		Could occur in drinking water from contamination/spills.
Trichloroethylene	c		Industrial solvent, cleaning fluid, metal degreaser. Could occur in drinking water from direct contamination or via atmospheric contamination of rainwater.
Triclopyr	0.02		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Trifluralin	0.09		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.
Trihalomethanes (THMs) (Total)	0.25 e		By-product of chlorination and chloramination. Action to reduce trihalomethanes is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than trihalomethanes.
Turbidity	c	5 NTU	5 NTU is just noticeable in a glass. <0.2 NTU is the target for effective filtration of Cryptosporidium and Giardia. <1 NTU is the target for effective disinfection.
Uranium	0.017		Occurs naturally, or from release from mill tailings, combustion of coal and phosphate fertilizers.
Vernolate	0.04		Pesticide, unlikely to be found in drinking water at levels that may cause health concerns.

Characteristic	Guideline values (mg/L unless otherwise specified)		Comments
	Health	**Aesthetic**	
Vinyl chloride	0.0003		From chemical spills. Used in making PVC pipes. Human carcinogen.
Xylene	0.6	0.02	Could occur in drinking water as a pollutant, or from solvent used for bonding plastic fittings.
Zinc	c	3	Usually from corrosion of galvanised pipes/fittings and brasses. Natural concentrations generally <0.01 mg/L. Taste problems >3 mg/L.

HU = Hazen units; NTU = nephelometric turbidity units; THMs = trihalomethanes.

a Aesthetic values are not listed if the compound does not cause aesthetic problems, or if the value determined from health considerations is the same or lower.

b If present at all in Australian drinking waters, concentrations of all organic compounds other than disinfection byproducts are likely to be very low relative to the guideline value.

c Insufficient data to set a guideline value based on health considerations.

d The guideline value is below the limit of quantitation. Improved analytical procedures are required for this compound.

e The concentration of all chlorination byproducts can be minimised by removing naturally occurring organic matter from the source water, reducing the amount of chlorine added, or using an alternative disinfectant (which may produce other byproducts). Action to reduce trihalomethanes and other byproducts is encouraged, but must not compromise disinfection.

f No corresponding fact sheet for these pesticides. Guideline values for these pesticides appeared in a previous version of the ADWG and have been retained in Table 10.5 for information purposes only.

Note: All values are as 'total' unless otherwise stated.

Note: Routine monitoring for these compounds is not required unless there is potential for contamination of water supplies (e.g. accidental spillage).

Table 10.7 *Guideline values radiological quality of drinking water*

Guideline value
The total estimated dose per year from all radionuclides in drinking water, excluding the dose from potassium-40, should not exceed 1.0 mSv.
If this guideline value is exceeded, the water provider, in conjunction with the relevant health authority, should evaluate possible remedial actions on a cost-benefit basis to assess what action can be justified to reduce the annual exposure.
Screening of water supplies
Compliance with the guideline for radiological quality of drinking water should be assessed, initially, by screening for gross alpha and gross beta activity concentrations. The recommended screening level for gross alpha activity is 0.5 Bq/L. The recommended screening level for gross beta activity is 0.5 Bq/L after subtraction of the contribution from potassium-40.
If either of these activity concentrations is exceeded, specific radionuclides should be identified and their activity concentrations determined. The concentrations of both radium-226 and radium-228 should always be determined, as these are the most significant naturally occurring radionuclides in Australian water supplies. Other radionuclides should be identified if necessary to ensure all gross alpha and beta activity is accounted for, after taking into account the counting and other analytical uncertainties involved in their determination.

10.4 Reference

Mosse P, Murray B (2008). *Practical Guide to the Operation and Optimisation of Media Filters*. Water Industry Operators Association, Shepparton.

PART IV INFORMATION SHEETS

Introduction to water treatment

INFORMATION SHEET 1.1

Most Australian source waters require treatment prior to being supplied to consumers as drinking water. The level of treatment that is required will be a function of the quality of the source water and should be informed by a risk assessment process that is consistent with the approach described under Element 2 (Assessment of the drinking water supply system) of the Framework for Management of Drinking Water Quality (the Framework), which is detailed in Chapter 3 of the Guidelines.

Water treatment processes are covered by Element 3 (Preventive measures for drinking water quality management) and Element 4 (Operational procedures and process control) of the Framework.

Water treatment processes can be roughly divided into two groups of processes: the physical removal of particulate matter and other contaminants (that is, coagulation, sedimentation, clarification and filtration), and the inactivation of pathogenic microorganisms (that is, disinfection).

Some of the important concepts that need to be considered in relation to water treatment are:

the application of multiple barriers (section 3.3.1), which helps ensure that a failure of one barrier may be compensated by the effective operation of the remaining barriers, particularly where multiple hazards need to be managed, thus minimising the likelihood of contaminants passing through the entire treatment system and being present in sufficient amounts to cause harm to consumers;

identifying which treatment processes will be considered critical control points (section 3.3.2), and establishing target criteria and critical limits for each treatment process which is identified as a critical control point (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for each treatment process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the treatment processes operate to the established target criteria and critical limits.

The following Information Sheets provide an overview of the primary inactivation processes used to disinfect water to a drinking water standard. The information provided in the Information Sheets is general in nature and the specific disinfection processes for individual water supply systems should be determined by a risk assessment, and in consultation with water treatment specialists.

Overview of disinfection

INFORMATION SHEET 1.2

Disinfection of public water supplies commenced early in the 20th century and together with filtration is credited with substantial reductions in waterborne disease (Cutler and Miller 2005, CDC 1999). Most Australian public water supplies are disinfected and it is recognised as a fundamental barrier to microbial contamination.

Disinfection may be practiced alone in circumstances where the source water is of a high quality or as the final step in a water treatment process that utilises multiple barriers. The decision to use disinfection alone, or in combination with other water treatment processes, is determined by a system-specific risk assessment, which includes an assessment of the quality of the source water. In most cases disinfection will be used as part of a multiple barrier approach to the production of safe drinking water.

In all cases disinfection should be treated as a critical control point (CCP) (see section 3.3.2 for more information on CCPs). This means the failure of the disinfection process may lead to the water being unsafe to drink.

Common agents used to disinfect water include chlorine, chloramine, chlorine dioxide, ozone and ultra violet (UV) light. Information sheets 1.3 - 1.7 describe these disinfectants. This information sheet on disinfection should be read in conjunction with the relevant fact sheets for each disinfectant, which discuss the chemical by-products produced by each disinfectant.

PROPERTIES OF AN IDEAL DISINFECTANT

An ideal disinfectant should:

Effectively inactivate pathogenic micro-organisms over a wide range of physical and chemical conditions;

Be able to have its efficacy continuously monitored;

Provide ongoing protection from pathogenic micro-organisms that may be introduced into the distribution system;

Provide a disinfectant residual that is stable and easily measured in the field;

Produce minimal levels of by-products;

Be readily available, safe to handle and suitable for widespread use;

Not degrade in quality or strength during storage; and

Be affordable (in terms of both capital and operating costs).

No single disinfectant currently meets all these criteria. Despite the limitations, disinfectants are an essential step and widely used in the production of safe drinking water. Choosing the appropriate disinfectant is an important decision for effective drinking water quality management in any water supply system. Factors which influence that decision include:

The quality of the source water being treated including:

The type of micro-organisms present (e.g. pathogenic bacteria, viruses, and/or protozoa)

The type and concentration of organic matter present in the source water

Propensity of the source water to form disinfection by-products

Turbidity, colour, UV absorbance, pH, alkalinity and water temperature (i.e., factors that affect disinfectant effectiveness)

The characteristics of the water supply system including:

Volume and dose rate of disinfectant required to maintain effective disinfection

The type and concentration of organic matter present in the treated water

Propensity of the treated water to form disinfection by-products

Retention time within the distribution system

Pipe condition

Size and complexity of the distribution system

Specific local circumstances such as:

Personnel safety

Public safety

Supply logistics

Technical capacity of staff to operate the disinfection facility

Technical support for operators to operate and maintain the disinfection facility

Cost

EFFECTIVENESS OF DISINFECTION

Determining the effectiveness of chemical disinfectants

The C.*t* concept describes the relative effectiveness of a specific aqueous disinfectant against different microorganisms under specified conditions. It is determined by multiplying the concentration or residual of the disinfectant (in mg/L) by the contact time (in minutes). The C.*t* concept is expressed mathematically as:

$$k = Cn.t$$

where:

C = concentration of residual disinfectant

n = constant (also called the coefficient of dilution)

t = contact time required for a fixed per cent of inactivation

k= constant for a specific microorganism exposed under set conditions.

Reported values for "n" range from 0.5 to 1.8 for most aqueous disinfectants. Generally, however, "n" approximates 1, and the equation is simplified to k = C.*t*.

C.t values for specific organisms exposed to particular disinfectants can be calculated. A low C.*t* value indicates a strong primary disinfectant.

Comparative effectiveness of disinfection, based on the C.*t* concept, for the four major disinfectants for a range of micro-organisms, are given in Information Sheets 1.3 to 1.6. The figures presented represent published C.*t* values that achieve 99% (or 2 log) inactivation of the target microorganism. In summary, the published C.*t* values show that ozone and chlorine dioxide are very effective at inactivating most microorganisms. Chlorine is effective at inactivating bacteria and viruses, but is less effective against *Giardia*, and not effective at inactivating *Cryptosporidium* at a C.*t* value that could be applied to a drinking water supply. Chloramine requires a considerably higher C.*t* value than chlorine to inactivate bacteria and viruses; it is ineffective against *Cryptosporidium* and *Giardia* at C.*t* values that could be applied to a drinking water supply.

The relative merits of various disinfectants, and ultraviolet light (UV), are summarised in Table IS1.2.1.

Table IS1.2.1 - Applicability of disinfection techniques to different situations

Consideration	Chlorine	Chloramination	Ozone	Chlorine dioxide	Ultraviolet light
Relative complexity of technology	Simple to Moderate	Simple to Moderate	Complex	Moderate	Simple to Moderate
Safety Concerns	Yes	Yes	Yes	Yes	Minimal
Bactericidal	Good	Good	Good	Good	Good
Virucidal	Moderate	Poor	Good	Good	Good
Protozocidal	Poor	Poor	Good	Moderate	Good
By-products of possible health concern	Yes	Yes	Yes	Yes	No
Persistent residual	Moderate	Long	None	Moderate	None
Contact Time	Moderate	Long	Short	Moderate	Short
pH dependent	Yes	Yes	Slightly	Slightly	No
Process control	Well developed	Well developed	Developed	Developed	Well developed

Where contact tanks or clear water storages are used to achieve the desired contact time, the T_{10} contact time needs to be taken into consideration (Church and Colton 2013; USEPA 2003). The T_{10} contact time is the minimum detention time experienced by 90 percent of the water passing through the tank, and is based on a baffling factor or tracer studies (Church and Colton 2013).

Determining the effectiveness of ultraviolet disinfection

The C.*t* concept does not apply to UV disinfection. UV disinfection relies on exposure of pathogens to UV irradiation, measured as UV fluence or UV dose, in mJ/cm^2. Different pathogens respond differently to UV irradiation so the target UV dose is typically selected based on the main pathogen/s of concern for a given source water.

The UV dose achieved by a given unit is a function of the following:

UV transmittance (UVT %), i.e. water quality;

lamp power (Watts); and

exposure time (which is typically related to flow rate).

Wherever possible, a validated UV system should be used, and preferably those systems that have been validated in accordance with the requirements of the USEPA *Ultraviolet disinfection guidance manual for the final long term 2 enhanced surface water treatment rule* (UVDGM) (2006). Other validation processes for UV systems also exist (DVGW, 2006a, 2006b, 2006c; ONORM, 2001, 2003; and NWRI, 2012).

The monitoring, operation and maintenance requirements that must be applied to ensure the ongoing performance of the UV unit at the required dose, as detailed in USEPA *UV Disinfection Guidance Manual* (2006) include:

flow, UVT and lamp power set points;

maintenance and calibration of essential UVT or UV intensity instrumentation; and

UV lamp monitoring, cleaning and replacement.

Finally, it should be recognised that energy consumption is a significant contributor to operating cost for UV disinfection systems, particularly large scale systems. Consequently, the accurate and efficient setting and control of the UV dose is important to avoid any undue overdose and therefore energy wastage.

Ensuring the effectiveness of disinfection

As one of the most important processes in assuring safe drinking water, disinfection will always be a CCP (see section 3.3.2). This means the disinfection process should be continuously monitored to provide assurance that it is functioning correctly (see Appendices A1.7 and A1.8).

For chlorination, chloramination and chlorine dioxide this is best achieved by permanent online chlorine residual analysers installed downstream of disinfection.

With disinfection processes that do not provide a residual other parameters can be monitored to confirm disinfection effectiveness, e.g. the intensity of UV dose can be monitored for UV performance. A key consideration is ensuring that effective disinfection is achieved under the most extreme operating conditions of maximum flows and minimum detention times.

Given that disinfection will be a CCP, other important issues that will need to be considered to ensure the effectiveness of the disinfection process are:

establishing target criteria and critical limits for the disinfection process (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the treatment process operates to the established target criteria and critical limits.

It is important to note that when chlorine, chloramine, ozone and chlorine dioxide are used as disinfectants, if the disinfectant comes into contact with organic matter a range of disinfection by-products (DBP) can be formed. The by-products produced by individual disinfectants, and their potential health significance, are described in the Fact Sheets for each disinfectant

It is also important to note that the possible presence of microbial contaminants in drinking water poses a greater risk to public health than the possible presence of DBP. Therefore, disinfection should not be compromised in order to control DBP.

FACTORS AFFECTING DISINFECTION

Disinfection processes, and their associated C.*t* values, are affected by a range of external factors, such as the pH and temperature of the water. Particle shielding can also reduce disinfection.

Water quality operators and managers need to understand the factors which can adversely impact on effectiveness of the disinfection process and monitor these parameters, in order to achieve effective disinfection.

The table below summarises the parameters which might impact on the effectiveness of disinfection which should be monitored for common disinfectants.

Disinfectant Parameters which may impact the effectiveness of disinfectants

Chlorine	Turbidity, pH, temperature
Chloramine	Turbidity, pH, temperature
Chlorine dioxide	Turbidity, pH, temperature
Ozone	Turbidity, pH, temperature
Ultraviolet irradiation	Turbidity, colour, iron and UV absorbance or UV transmissivity (UVT)

Ideally these parameters should be monitored continuously, especially for source waters where water quality can change rapidly. If source waters are very stable (such as confined groundwater) it may be acceptable to monitor with regular grab samples.

The table above does not include all the operational parameters which should be monitored to demonstrate the effectiveness of disinfection process. Further details can be found in the Information Sheet for each disinfectant.

If the target criterion for an operational parameter is breached, it does not necessarily mean that disinfection has been compromised. In such instances the water utility needs to:

Validate that disinfection is still effective, specifically in the circumstance where supply has to be maintained during the period that the operational parameter is outside the target range;

Where possible, use an alternative source water;

Where possible, only take raw water that is within, or has returned to within, the target range for the water treatment processes that are being used; and

Verify that the water within the distribution system is still of a satisfactory quality.

DISINFECTION AND RESIDUAL MANAGEMENT

When operating a distribution system it is important to understand the difference between effective disinfection and maintaining a disinfectant residual. The water is effectively disinfected when the required C.*t* value has been achieved. After effective disinfection, enteric pathogens should not reappear within the distribution system, unless there is a failure in the integrity of the system. Therefore, unless there is a barrier breach within the distribution system the water should remain safe to drink even in the absence of adequate disinfectant residual. Barrier breaches could include such things as ingress, backflow, loss of pressure within the distribution system, or contamination within post-treatment storage tanks.

Operationally, a sudden loss of chlorine residual within the distribution system can also warn of an unusual event or potential contamination, such as backflow, within the distribution system.

Most Australian water utilities use chlorine as their disinfectant of choice with C.*t* target of at least 15 mg/L.min which is consistent with the World Health Organization's recommendation that effective disinfection can generally be achieved by maintaining a free chlorine concentration of 0.5 mg/L for 30 minutes (WHO, 2011). Based on data contained in Information Sheet 1.3 a C.t of 15 mg/L.min should achieve effective inactivation of enteric bacteria and viruses. With chlorinated/chloraminated supplies, it is common practice in Australia to endeavour to maintain adequate chlorine residual throughout the entire distribution system. Maintaining chlorine residual provides protection from backflow, ingress and *Naegleria*, and helps inhibit biofilm growth.

Where the aim is to maintain adequate disinfectant residual across the entire distribution system, the target chlorine residual set at the chlorinator will be based on achieving a desired residual concentration through most of the distribution system, including, where possible, the extremities of the system, after allowing for chlorine decay. As most chlorinators are designed to achieve a set residual to ensure a minimum C.*t* near the point of dosing the resultant C.*t* values will increase with longer contact time in the distribution system achieving a margin of safety.

The chlorine decay from the point of disinfection to the extremity of the distribution system is influenced by many factors including:

Water travel time/age

Chlorine demand of the water being disinfected

Water temperature

Biofilm growth

Asset condition of pipes and storages

All of these factors are dynamic. It is necessary to monitor the chlorine residual frequently within the distribution system, to enable adjustments of the chlorinator dosing rate to achieve the desired residual within the system.

If maintaining a consistent residual is difficult to achieve, or chlorine demand changes frequently, the water supplier should seek to understand the factors that are driving these changes. Some factors, such as water temperature, may be outside the water supplier's control. However, other factors which may contribute to a sudden increase in chlorine demand (e.g. algal growth) may indicate an adverse event in the source water.

Whilst the absence of sufficient disinfectant residual does not necessarily mean the water is unsafe to drink, if the desired chlorine residual is not being achieved then corrective action needs to be taken to ensure the target value is achieved.

MANAGING WATER SUPPLIES WITH NO RESIDUAL

When used as disinfectants, ozone and UV light do not produce residuals within the distribution system. As discussed in the previous section, after effective disinfection, enteric pathogens should not reappear within the distribution system, unless there is a failure in the integrity of the system. In systems which lack residual disinfection, there is an increased need to ensure the integrity of the system to compensate for the absence of a residual.

In the Australian context, distribution systems that are suited to operating with no residual are those which have high quality source water (e.g. groundwater from a confined aquifer), or are small distribution systems, with a low water age within the distribution system, and which do not have embedded post-treatment storage tanks.

The attractions of operating distribution systems with no residual disinfection are that costs associated with maintaining residual disinfection are avoided, there are no issues with disinfection by-products, and it addresses the concerns of some consumers in relation to the use of chlorine. The main issues with no residual are that if the integrity of system is breached, there is no effective barrier to microbial contamination, and there can be additional costs involved with the cleaning and disinfection of the distribution system.

REFERENCES

Austrian Standards Institute (ÖNORM) (2001). ÖNORM M 5873-1: Plants for the disinfection of water using ultraviolet radiation: Requirements and testing. Part 1. Low pressure mercury lamp plants. Österreichisches Normungsinstitut, A-1021 Vienna, Austria.

Austrian Standards Institute (ÖNORM) (2003). ÖNORM M 5873-2 Plants for the disinfection of water using ultraviolet radiation: Requirements and testing. Part 2. Medium pressure mercury lamp plants. Österreichisches Normungsinstitut, A-1021 Vienna, Austria.

Church J. and Colton J. (2013). Optimise chlorine contact Tank performance, *WaterWorks*, May 2013, 10-13, Water Industry Operators Association of Australia (WIOA), Shepparton, Australia.

Cutler D. and Miller G. (2005). The role of public health improvements in health advances: the twentieth-century United States. *Demography* 42:1-22.

German Association for Gas and Water (DVGW) (2006a). *UV Devices for Disinfection in the Water Supply Part 1: Requirements Related to Composition, Function and Operation, in Technical Rule, Code of Practice W294-1*. Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

German Association for Gas and Water (DVGW) (2006b). *UV Devices for Disinfection in the Water Supply Part 2: Testing of Composition, Function and Disinfection Efficiency in Technical Rule, Code of Practice W294-2* Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

German Association for Gas and Water (DVGW) (2006c). *UV Devices for Disinfection in the Water Supply; Part 3: Measuring Windows and Sensors for the Radiometric Monitoring of UV Disinfection Devices: Requirements, Testing and Calibration in Technical Rule, Code of Practice W 294-3* Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

National Water Research Institute (NWRI) (2012). *Ultraviolet Disinfection Guidelines for Drinking Water and Water Reuse*, Third Edition, California, United States of America.

US Centres for Disease Control and Prevention (1999). Morbidity and Mortality Weekly Report. Achievements in Public Health 1900-1999, *Control of Infectious Diseases 48 (29)* 621-629.

United States Environmental Protection Agency (US EPA) (2003). *Guidance Manual for Disinfection Profiling and Benchmarking* US EPA Office of Water: Washington D.C.

United States Environmental Protection Agency (US EPA) (2006). *Ultraviolet disinfection guidance manual for the final long term 2 enhanced surface water treatment rule*. US EPA Office of Water: Washington D.C.

World Health Organization (WHO) (2011). *Guidelines for Drinking-water Quality*, fourth edition, Geneva, Switzerland.

Disinfection with chlorine

INFORMATION SHEET 1.3

The possible presence of microbial contaminants in drinking water poses a greater risk to public health than the possible presence of disinfection by-products (DBP). Therefore, disinfection should not be compromised in order to control DBP.

Where the concentrations of chlorinated DBP consistently exceed associated health-based guideline values, the methods of water treatment, disinfection and distribution should be reviewed.

GENERAL DESCRIPTION

Chlorine was introduced widely as a water disinfectant early in the 20th century and still remains the most common drinking water disinfectant used around the world. It is a strong disinfectant with excellent bactericidal and virucidal properties and is effective at short contact times. Chlorine is less effective against protozoa and while it can inactivate *Giardia* at moderate doses and contact times it has little effect against *Cryptosporidium* at doses that can be practically used in drinking water. It is also a strong oxidising agent that can bleach colour compounds in water, oxidise soluble iron, manganese and sulfides, and remove the tastes, odours and some toxins produced by algae.

In water, chlorine reacts to form hypochlorous acid (HOCl) (see below), a very effective disinfectant which can dissociate to form the hypochlorite ion (OCl^-) in a pH dependent reaction with no dissociation below pH 6.5 and complete dissociation above pH 8.5. From a disinfection standpoint lower pHs are preferred as the hypochlorite ion is estimated to be 150 to 300 times less effective as a disinfectant than hypochlorous acid.

Cl_2 (gas) + H_2O $\rightleftharpoons$ $H^+ + Cl^- + HOCl$
$NaOCl + H_2O \rightleftharpoons NaOH + HOCl$
$Ca(OCl)_2 + 2H_2O \rightleftharpoons Ca(OH)_2 + 2HOCl$

HOCl then dissociates to the hypochlorite ion (OCl-) in a pH dependent reaction:
HOCl $\rightleftharpoons$ H+ + OCl- pKa = 7.5

APPLICATION

Chlorine is the most versatile of disinfectants used to treat drinking water supplies. It can be applied:

as a primary disinfectant at the point of entry into the drinking water distribution system;

as a secondary disinfectant within distribution systems to boost concentrations of chlorine residuals in the system as a barrier against regrowth of opportunistic free-living pathogens and ingress of faecal contamination;

to disinfect new and repaired water mains; and

to disinfect storage tanks as part of cleaning and maintenance or following the detection of contamination.

Chlorine can be applied alone or in combination with other disinfectants. For example, it can be used in combination with UV light disinfection as joint primary disinfectants where UV light is used primarily to inactivate *Cryptosporidium* and *Giardia*, and chlorine is used to inactivate viruses and bacteria. In this combination chlorination also provides a residual disinfectant to provide protection of distribution systems against regrowth and recontamination. Chlorine can also be used for this purpose in conjunction with ozone and chlorine dioxide.

Chlorine can also be used in combination with chloramines either as a primary disinfectant before production of persistent chloramine residuals or as a secondary disinfectant in subsections of water distribution systems.

PRACTICAL CONSIDERATIONS

Chlorine can be applied as a gas, liquid (sodium hypochlorite) or solid (calcium hypochlorite). Due to the strict safety requirements associated with the use of gaseous chlorine, liquid chlorine, which is easier to use, is often used in preference to gaseous chlorine. The disadvantages of sodium hypochlorite are that concentrations degrade over time, chlorate can be formed during storage and it is a corrosive solution. Calcium hypochlorite needs to be stored in a cool dry environment and kept away from moisture and heat. Chlorine residuals and chlorination by-products can produce distinctive tastes and odours (see Taste and Odour Fact Sheet).

Advantages of chlorination include its common and long-standing use and the availability of reliable dosing and monitoring equipment. Reliable and robust field kits for measuring chlorine residuals within the distribution system are also available. In addition to being a proven disinfectant against most enteric pathogens (excluding *Cryptosporidium*) chlorine is also a strong oxidising agent that can bleach colour compounds in water, oxidise soluble iron, manganese and sulfides, and remove the tastes and odours and some toxins produced by algae.

PERFORMANCE VALIDATION

Table IS1.3.1 presents published C.*t* values for chlorine that have been demonstrated as achieving a two log reduction in the target microorganism. These values are supplied for illustrative purposes only. For chlorine C.*t* values that achieve a greater log reduction, the cited references should be consulted. The C.*t* value that is applied at a particular water treatment plant should be based on the microbial risk assessment for that particular water supply system.

Table IS1.3.1 Published C.t values for 99% (2 log) inactivation of various microorganisms by chlorine 1,2

Microorganism	Free chlorine C.t value (mg/L.min)	Reference
Escherichia coli	<1 (10-15°C)	LeChevallier and Au 2004
CB5 virus	3.29 (10°C)	Keegan et al. 2012
Naegleria fowleri	30 (30°C)	Robinson and Christy 1984
Giardia	60 (15°C)	USEPA 1991
Cryptosporidium	7200 (25°C)	Korich et al. 1990

Notes: (1) pH is within the range of 6-9 for *E.coli* and pH7 for the other organisms.

(2) The values in the table are based on published values and should be viewed as the minimum values necessary to achieve effective disinfection.

The important conclusion to draw from Table IS1.3.1 is that, at the typical chlorine C.*t* values used in Australian drinking water supplies, which are usually based on the World Health Organization's recommendation that effective disinfection can generally be achieved by applying a 30 minute contact time to a free chlorine concentration of 0.5 mg/L (WHO 2011) (i.e. equivalent to a chlorine C.*t* value of 15 mg/L.min), chlorine will inactivate bacteria and viruses, but will not inactivate *Giardia* or *Cryptosporidium*.

Chlorine is also effective against *Naegleria fowleri*, but the elevated C.*t* requirement means dosing must be adjusted to provide a sufficient residual throughout the distribution system. *Naegleria* can encyst and when in this state are more resistant to disinfection. Unless the chlorine residual is continuous, the cysts are also able to survive in tank sediments and pipe biofilm. A free chlorine residual at 0.5 mg/L or higher will control *N. fowleri*, provided the disinfectant residual persists throughout the water supply system at all times.

WATER QUALITY CONSIDERATIONS

Evidence from various studies indicates that pH influences disinfection, with lower pHs being optimal, as the hypochlorous acid is far more effective than the hypochlorite ion. Temperature also influences efficacy, with disinfection times reduced at higher temperatures. Although it has been suggested that particles may act as a protective shield for micro-organisms, and that turbidity should be kept below 1 NTU for effective disinfection, the relationship between turbidity and the effectiveness of chlorine has not been established for all pathogens. Increasing the turbidity from <1 to 20NTU increased the Ct for 2 log inactivation of CB5 from 3.29 to 5.95 mg.min/L at pH 7 (Keegan et al. 2012).

Whilst many water suppliers often achieve satisfactory inactivation of bacteria at turbidities that are greater than 1 NTU, generally, the lower the turbidity of the water at the time of chlorination the more effective chlorination will be. Where chlorination is routinely occurring at turbidities that are greater than 1 NTU, the effectiveness of the chlorination process should be validated.

Relationships with other parameters, such as natural organic matter or colour, have not been well studied; however, it is known that these parameters adversely impact on the chlorine dose required to achieve a free chlorine residual and effective disinfection.

Where contact tanks or clear water storages are used to achieve the desired contact time, the T_{10} contact time needs to be taken into consideration (Church and Colton 2013; USEPA 2003). The T_{10} contact time is the minimum detention time experienced by 90 percent of the water passing through the tank, and is based on a baffling factor or tracer studies (Church and Colton 2013).

PERSISTENCE

A major advantage of chlorination is that it produces a residual disinfectant that is moderately persistent, with longevity limited by chlorine being a highly reactive oxidant. Chlorine can be used to provide a residual disinfectant in distribution systems, with persistence dependent on the chlorine demand imparted by natural organic matter (and inorganic compounds, such as iron) in drinking water, and other factors such as temperature and sunlight (if the system incorporates open storages). The persistence of chlorine also makes it suitable for the control of themophilic *Naegleria*, including *N. fowleri*, particularly where a sufficient residual can be maintained throughout the distribution system.

Chlorine will not persist in long distribution systems, particularly those incorporating long above-ground pipelines, because of the elevated water temperature that occurs in these pipelines. Such distribution systems lend themselves to chloramination (see Disinfection with chloramine Information Sheet).

BY-PRODUCTS

Chlorine, in reaction with natural organic matter present in source water, can form a wide range of halogenated disinfection by-products, with over 600 identified to date (Hrudey 2009, Itoh et al. 2011). These include trihalomethanes, haloacetic acids, haloacetonitriles and trichloroacetaldehyde (chloral hydrate). The chemistry of the reactions is complex and not fully understood. Factors that influence the formation of disinfection by-products include the chlorine dose, the concentrations and types of natural organic matter that are present, temperature, pH and detention time. Chlorate can be produced in association with degradation of concentrated sodium hypochlorite solutions.

Guideline values have been developed for a number of disinfection by-products (see Chapter 10 and associated Fact Sheets). While every effort should be taken to minimise the formation and concentration of chemical disinfection by-products this should never be done in a manner that compromises disinfection, as poor microbiological quality represents a greater and more immediate risk to human health than short term exposure to disinfection by-products (Hrudey 2009).

OPERATIONAL CONSIDERATIONS

Given that the chlorination process will be a critical control point (CCP), other important issues that will need to be considered to ensure the effectiveness of the process are:

establishing target criteria and critical limits for the chlorination process (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the chlorination process operates to the established target criteria and critical limits.

OPERATIONAL MONITORING

The table below summarises the operational monitoring that should be undertaken for chlorine.

Operational Parameter	**Monitoring**
pH	Online monitoring
Turbidity	Online monitoring
Contact time	Calculated
Chlorine residual (total)	Online monitoring

REFERENCES

Church J. and Colton J. (2013). Optimise chlorine contact Tank performance, *WaterWorks*, May 2013, 10-13, Water Industry Operators Association of Australia (WIOA), Shepparton, Australia.

Hrudey S. (2009). Chlorination disinfection by-products, public health risk tradeoffs and me. Water Research 43: 2057-2092.

Itoh S, Gordon BA, Callan P and Bartram J. (2011). Regulations and perspectives on disinfection by-products: Importance of estimating overall toxicity. J. Water Supply: Research and Technology-Aqua 60, 261-274.

Keegan A, Wati S, Robinson B. (2012). Chlor(am)ine disinfection of human pathogenic viruses in recycled waters. Smart Water Fund Project SWF62M-2114, Smart Water Fund, Melbourne, Australia.

Korich DG, Mead JR, Madore MS, Sinclair NA and Sterling CR (1990). Effects of ozone, chlorine dioxide, chlorine, and monochloramine on Cryptosporidium parvum oocyst viability. Applied and Environ Micro. 56, 1423-1428.

LeChevallier MW and Au K-K. (2004). Water treatment and pathogen control. World Health Organization, Geneva, Switzerland.

Robinson BS and Christy PE. (1984). Disinfection of water for control of amoebae. Water 11: 21-24.

United States Environmental Protection Agency (US EPA) (1991). *Guidance manual for compliance with the filtration and disinfection requirements for public water systems using surface water sources*. US EPA Office of Water: Washington D.C.

United States Environmental Protection Agency (US EPA) (2003). *Guidance Manual for Disinfection Profiling and Benchmarking* US EPA Office of Water: Washington D.C.

World Health Organization (WHO) (2011). *Guidelines for Drinking-water Quality*, fourth edition, Geneva, Switzerland.

Chloramines

INFORMATION SHEET 1.4

The possible presence of microbial contaminants in drinking water poses a greater risk to public health than the possible presence of disinfection by-products (DBP). Therefore, disinfection should not be compromised in order to control DBP.

Where the concentrations of DBP consistently exceed associated health-based guideline values, the methods of water treatment, disinfection and distribution should be reviewed.

GENERAL DESCRIPTION

Chloramines are formed when chlorine and ammonia are added to water:

$NH_3 + HOCl \longrightarrow NH_2Cl + H_2O$
monochloramine

$NH_2Cl + HOCl \longrightarrow NHCl_2 + H_2O$
dichloramine

$NHCl_2 + HOCl \longrightarrow NCl_3 + H_2O$
nitrogen trichloride (trichloramine)

There are three types of chloramines; monochloramine, dichloramine and trichloramine, with production of the three types dependent on pH and the ratio of chlorine to ammonia. Monochloramine is the preferred choice because it is the most stable and produces the lowest tastes and odours. Dichloramine is a stronger disinfectant than monochloramine, but is less stable and produces distinctive odours. Trichloramine is the least stable and produces offensive odours. Formation and stability of monochloramine is favoured at Cl_2/NH_3 ratios of 3 to 5 (with 4 typically used) and a pH above 8 (UWRAA 1990, USEPA 1999).

The primary reason for using monochloramine rather than chlorine is its much greater persistence. In Australia chloramination has provided persistent residuals through very long drinking water pipelines (>100kms) and provides protection against growth of free-living organisms such as *Naegleria fowleri* (UWRAA 1990).

APPLICATION

Chloramination is used in drinking water supplies where persistence is a key advantage and can be particularly useful in long or complex distribution systems that have extended detention times. It has been particularly effective in reducing the occurrence of *Naegleria fowleri* in systems incorporating long above ground pipelines, and has also been shown to reduce the occurrence of *Legionella* in buildings (Flannery et al. 2006).

In conjunction with other disinfectants, such as chlorine or ultraviolet (UV) light, chloramines can also be used as a secondary (or booster) disinfectant to provide persistent residuals within the distribution system.

Where chloramination is used as the primary disinfectant, due to the relatively high C.*t* values required to inactivate microbial pathogens, it is important to determine the minimum C.*t* values before the first customer in the drinking water distribution system. This is often done by calculating minimum C.*t* values at maximum flow rates.

An emerging practice is to dose chlorine followed by ammonia separately, at different points within the water treatment process. The advantage of this practice is that achieves better upfront inactivation of microbial pathogens, whilst still delivering a longer-lasting residual within the distribution system. To maximise the benefits of the practice there needs to sufficient contact time for the chlorine to achieve inactivation, prior to the addition of the ammonia.

PRACTICAL CONSIDERATIONS

Chloramines are formed by the addition of either liquefied anhydrous ammonia or aqueous ammonia (UWRAA 1990, USEPA 1999), either before or after chlorine dosing. The addition of ammonia first reduces the production of chlorinated disinfection by-products, but it also reduces initial inactivation by reducing the contact time between the free chlorine and the treated source water.

Chloramination has a long history of use and was introduced in Brisbane in 1935. Robust and reliable dosing and monitoring equipment is available. Reliable field kits for measuring residuals within the distribution system are also available; these kits generally measure concentrations of chloramines as total chlorine. There have been reports of false free chlorine readings with tablet-based methods (UWRAA 1990). The DPD-Ferrous titrimetric method is less prone to false readings (see monochloramine Fact Sheet).

PERFORMANCE VALIDATION

Table IS 1.4.1 presents published C.*t* values for preformed monochloramine (that is, monochloramine that is formed off-site) that have been demonstrated as achieving a two log reduction in the target microorganism. These values are supplied for illustrative purposes only. For chloramine C.*t* values that achieve a greater log reduction, the cited references should be consulted. The C.*t* value that is applied at a particular water treatment plant should be based on the microbial risk assessment for that particular water supply system.

Table IS1.4.1 Published C.t values for 99% (2 log) inactivation of various microorganisms by preformed monochloramine 1,2

Microorganism	Preformed monochloramine C.t value (mg/L.min)	Reference
Escherichia coli	95-180 (5°C)	LeChevallier and Au 2004
Adenovirus 2	1688 (10°C at 2 NTU)	Keegan *et al.* 2012
Giardia	1470 (5°C)	USEPA 1999
Naegleria fowleri	320 (30°C)	Robinson and Christy 1984

Notes: (1) pH is within range of 8-9 for *E. coli*, pH 7 for Adenovirus 2 and *Naegleria fowleri*, pH 6-9 for *Giardia*.

(2) The values in the table are based on published values and should be viewed as the minimum values necessary to achieve effective disinfection

The important conclusion to draw from Table IS1.4.1 is that chloramines require a much higher C.*t* value than chlorine to inactivate microorganisms. Therefore, for distribution systems where chloramine is going to be used, the need for much greater C.*t* values will be an important consideration (i.e. water needs to be held in the pipe network and/or tanks for longer to allow sufficient time for effective disinfection).

WATER QUALITY CONSIDERATIONS

The influence of pH on the effectiveness of disinfection appears to be variable (UWRAA 1990, USEPA 1999, Cromeans et al. 2010) and could depend on the target microorganism. Varying the pH will influence the species of chloramine present and this could impact on the effectiveness of disinfection. The target range for pH during chloramination is usually 8.5 ± 0.2. Monochloramine stability improves with increased pH, with optimum stability occurring at pH 9. There is no benefit from chloramination at a pH greater than 9 (UWRAA 1990).

As for chlorine, C.*t* requirements are reduced at higher temperatures (USEPA 1999, Kahler et al. 2011). Monochloramine appears to remain effective at high turbidity but inactivation rates decrease as turbidity increases (UWRAA 1990). This finding was confirmed by more recent work by Keegan et al. (2012), using water that varied from 2 to 20 NTU.

PERSISTENCE

Persistence is the principal advantage of chloramination, and it has been used successfully to produce disinfectant residuals through long pipeline systems exceeding 100km in length.

Persistence can be reduced by nitrification, particularly at the ends of distribution systems. Nitrification is caused by bacterial oxidation of ammonia to nitrite, and nitrite to nitrate (Cunliffe 1991). Nitrifying bacteria are naturally-occurring sediment and biofilm organisms. Nitrification can accelerate chloramine decay, and at the ends of distribution systems can lead to complete loss of residual and the replacement of chloramines with oxidised nitrogen (nitrate and nitrite). A number of factors have been associated with nitrification including detention times, excess ammonia (low chlorine:ammonia ratios) and low chloramine residual. The common practice to reduce the likelihood of nitrification is maintaining minimum chloramine residuals of 1.5-2 mg/L (Cunliffe 1991, USEPA 1999).

Work in South Australia indicates that the risk of nitrification can be mitigated by ensuring that the concentration of free ammonia after chloramination does not exceed 0.3 mg/L. Additionally, the concentrations of nitrate and nitrite can be monitored within the distribution network to given an early indication of the onset of nitrification.

The replacement of chlorination with chloramination improves the microbiological quality of the water in large distribution systems (UWRAA 1990). The general reason for this is that biocidal residuals of monochloramine travel further into the system, providing effective disinfection. If nitrification occurs within the system, then a return to chlorination for about a week may be necessary in order to control nitrifying bacteria.

BY-PRODUCTS

Chloramination generally reduces chlorinated DBP, including trihalomethanes (THMs), but does not eliminate them. A similar range of DBP will be produced as when chlorine is used, particularly when chlorine is added before the ammonia (WHO 2000). Dichloroacetic acid can be formed from monochloramine and cyanogen chloride formation is greater than with free chlorine (USEPA 1999).

In addition, chloramination can be associated with the production of nitrosamines, including NDMA (see NDMA Fact Sheet). Factors that influence the formation of NDMA include the chloramine dose, the concentrations and types of organic nitrogen-containing compounds that are present, pH and detention time (WQRA 2013).

Health-based guideline values have been developed for a number of DBP (see Chapter 10 and associated Fact Sheets). Whilst every effort should be taken to minimise the formation and concentration of DBP this should never be done in a manner that compromises disinfection as poor microbiological quality represents a greater and more immediate risk to human health than short term exposure to DBP (Hrudey 2009).

OPERATIONAL CONSIDERATIONS

Given that the chloramination process will be a critical control point (CCP), other important issues that will need to be considered to ensure the effectiveness of the process are:

establishing target criteria and critical limits for the chloramination process (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the chloramination process operates to the established target criteria and critical limits.

OPERATIONAL MONITORING

The table below summarises the operational monitoring that should be undertaken for chloramine.

Operational Parameter	**Monitoring**
pH	Online monitoring
Turbidity	Online monitoring
Chlorine-to-ammonia ratio	Calculated
Contact time	Calculated
Chlorine residual (total)	Online monitoring
Chloramine concentration	Field kit

REFERENCES

Cunliffe DA. (1991). Bacterial nitrification in chloraminated water supplies Applied and Environ. Micro 57: 3399-3402.

Cromeans TL, Kahler AM, and Hill VR. (2010). Inactivation of Adenoviruses, Enteroviruses, and Murine Norovirus in Water by Free Chlorine and Monochloramine. Applied and Environ. Micro 76: 1028-1033.

Flannery B, Gelling LB, Vugia DJ, Weintraub JM, Salerno JJ, Conroy MJ, Stevens VA, Rose CE, Moore MR, Fields BS, and Besser RE. (2006). Reducing *Legionella* colonization of water systems with monochloramine. Emerging Infectious Diseases 12: 588-596.

Hrudey S. (2009). Chlorination disinfection by-products, public health risk tradeoffs and me. Water Research 43: 2057-2092.

Kahler AM, Cromeans TL, Roberts JM, Hill VR. (2011). Source water quality effects on monochloramine inactivation of adenovirus, coxsackievirus, echovirus, and murine norovirus. Water Research 45:1745-51.

Keegan A, Wati, S, Robinson B. (2012). Chlor(am)ine disinfection of human pathogenic viruses in recycled waters. Smart Water Fund Project SWF62M-2114, Smart Water Fund, Melbourne, Australia.

LeChevallier MW and Au K-K. (2004). Water treatment and pathogen control. World Health Organization, Geneva.

Robinson BS and Christy PE. (1984). Disinfection of water for control of amoebae. Water 11: 21-24.

United States Environmental Protection Agency (USEPA) (1999). Alternative disinfectants and oxidants guidance manual. Washington DC.

Urban Water Research Association of Australia (UWRAA) (1990) Chloramination of water supplies. UWRAA, Melbourne Australia.

Water Quality Research Australia (WQRA) (2013). Guidance Manual for the Minimisation of NDMA and other Nitrosamines in Drinking and Recycled Water. Research Project 1018, Water Quality Research Australia Limited, Adelaide, Australia.

World Health Organization (WHO) (2000). Disinfectants and disinfection by-products. Environmental Health Criteria 216. WHO, Geneva.

Disinfection with chlorine dioxide

INFORMATION SHEET 1.5

The possible presence of microbial contaminants in drinking water poses a greater risk to public health than the possible presence of disinfection by-products (DBP). Therefore, disinfection should not be compromised in order to control DBP.

Where the concentrations of DBP consistently exceed associated health-based guideline values, the methods of water treatment, disinfection and distribution should be reviewed.

GENERAL DESCRIPTION

Chlorine dioxide is a strong oxidant that in addition to being an effective biocide can be used to oxidise iron and manganese, and control taste- and odour-causing compounds. It has also been used as a secondary disinfectant in many European countries (Le Chevallier and Au 2004).

Chlorine dioxide is highly soluble in water (particularly at low temperatures), and is effective over a range of pH values (pH 5–10). Theoretically, chlorine dioxide undergoes five valence changes in oxidation to chloride ion:

$ClO_2 + 5e– = Cl– + 2O^2–$

However, in practice, chlorine dioxide is rarely reduced completely to the chloride ion (White 1999).

Chlorine dioxide is thought to inactivate microorganisms through direct oxidation of tyrosine, methionine, or cysteine-containing proteins, which interferes with important structural regions of metabolic enzymes or membrane proteins (Gates 1998). In water treatment, chlorine dioxide has the advantage of being a strong disinfectant, but of not forming trihalomethanes (THMs) or oxidizing bromide to bromate (Le Chevallier and Au 2004). Whilst not producing THMs, the by-products chlorate and chlorite can be produced.

APPLICATION

Chlorine dioxide is a suitable disinfectant for a small to medium sized water treatment plant. It has been used mainly as a preoxidant (rather than as a primary disinfectant, due mainly to its relative cost, and lack of a persistent residual) to control taste and odour, oxidise iron and manganese, and more recently, remove the precursors of THMs and total organic halogen (TOX). In some supplies chlorine dioxide has been used in combination with chloramination.

PRACTICAL CONSIDERATIONS

Reliable equipment is available for disinfection with chlorine dioxide. However, the technology involved is moderately complex, but more effective controls for the process are developing. Chlorine dioxide is highly reactive and can be rapidly consumed.

PERFORMANCE VALIDATION

Table IS 1.5.1 presents published C.t values for chlorine dioxide that have been demonstrated as achieving a two log reduction in the target microorganism. These values are supplied for illustrative purposes only. For chlorine dioxide C.t values that achieve a greater log reduction, the cited references should be consulted. The C.t value that is applied at a particular water treatment plant should be based on the microbial risk assessment for that particular water supply system.

Table IS1.5.1 Published C.t values for 99% (2 log) inactivation of various microorganisms by chlorine dioxide 1,2,3

Microorganism	Chlorine Dioxide C.t value (mg/L.min)	Reference
Escherichia coli	0.4-0.75	USEPA 1999
Enteric viruses	5.6	USEPA 1999
Giardia	17	USEPA 1999
Cryptosporidium	858	USEPA 2010

Notes: (1) Water temperature is 5°C.

(2) pH is within the range of 6-9.

(3) The values in the table are based on published values and should be viewed as the minimum values necessary to achieve effective disinfection

The important conclusion to draw from Table IS1.5.1 is that the C.*t* values required to inactivate bacteria and viruses, and to some extent *Giardia*, are comparable to those for chlorine, but the C.*t* value required to inactivate *Cryptosporidium* is unlikely to be able to be achieved in most drinking water supply systems.

WATER QUALITY CONSIDERATIONS

Chlorine dioxide is a reactive gas that cannot be easily stored or transported, and must be generated on site; this is usually done by acid treatment of sodium chlorite, which generates the gas with little or no chlorine contamination and so avoids the formation of chlorinated by-products during disinfection.

It has excellent oxidising ability, which reduces taste, minimises colour and oxidises iron and manganese complexes.

Turbidity at the time of disinfection should be less than 1 NTU.

The effectiveness of chlorine dioxide is also not as sensitive to changes in pH as chlorine. There is some evidence that effectiveness against protozoa increases from pH 6 to 8 (USEPA 1999).

PERSISTENCE

Chlorine dioxide provides a moderately persistent residual.

BY-PRODUCTS

By-products from the use of chlorine dioxide include chloride ions, chlorite ions, chlorate ions (see Fact Sheet on Chlorine dioxide/ chlorate/ chlorite for more information). Whilst not a by-product, in some cases residual chlorine dioxide may also be present.

OPERATIONAL CONSIDERATIONS

Given that the dosing point for chlorine dioxide will be a critical control point (CCP), other important issues that will need to be considered to ensure the effectiveness of the process are:

establishing target criteria and critical limits for the dosing process (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the

operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the dosing process operates to the established target criteria and critical limits.

OPERATIONAL MONITORING

The table below summarises the operational monitoring that should be undertaken for chlorine dioxide, based on recommendations from the New Zealand Ministry of Health (NZ MoH 2008).

Operational Parameter	**Monitoring**
pH	Online monitoring
Turbidity	Online monitoring
Chlorine dioxide concentration	Online monitoring

Regular monitoring for chlorite and chlorate should also be undertaken.

REFERENCES

Gates D (1998). The chlorine dioxide handbook: water disinfection series. American Water Works Association, Denver, CO.

LeChevallier MW and Au K-K (2004). Water treatment and pathogen control. World Health Organization, Geneva.

New Zealand Ministry of Health (2008). Drinking-water Standards for New Zealand 2005 (Revised 2008).

United States Environmental Protection Agency (USEPA) (1999). Alternative disinfectants and oxidants guidance manual. Washington DC.

United States Environmental Protection Agency (USEPA) (2010). Long term 2 enhanced surface water treatment rule toolbox guidance manual. Washington DC.

White GC. (1999). ***Handbook of Chlorination and Alternative Disinfectants.*** John Wiley & Sons, Inc, New York.

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Disinfection with ozone

INFORMATION SHEET 1.6

The possible presence of microbial contaminants in drinking water poses a greater risk to public health than the possible presence of disinfection by-products (DBP). Therefore, disinfection should not be compromised in order to control DBP.

Where the concentrations of DBP consistently exceed associated health-based guideline values, the methods of water treatment, disinfection and distribution should be reviewed.

GENERAL DESCRIPTION

Ozone is generated on site by passing an electric discharge through clean dry air or oxygen. The resultant ozone is a very strong biocide and oxidising agent, and is effective in reducing colour, taste and odour, and oxidising iron and manganese.

The mechanism by which ozone inactivates microorganisms is not well understood. Ozone in aqueous solution may react with microorganisms either by direct reaction with molecular ozone or by indirect reaction with the radical species formed when ozone decomposes (Le Chevallier and Au 2004). Ozone is known to attack unsaturated bonds, forming aldehydes, ketones or carbonyl compounds (Langlais et al. 1991).

Free radicals formed by the decomposition of ozone are generally less effective for microbial inactivation than molecular ozone, because microbial cells contain a high concentration of bicarbonate ions that quench the free radical reaction, and many microbial cells also contain catalase, peroxidase, or superoxide dismutase to control free radicals produced by aerobic respiration. In addition, some bacteria contain carotenoid and flavonoid pigments that protect them from ozone. These factors can account for reports that heterotrophic bacteria may be less susceptible to ozone inactivation than *Giardia* (Wolfe et al. 1989).

APPLICATION

Ozone can be used in medium to large treatment plants, although it has not been used in Australia to date for the primary disinfection[1] of a sizeable drinking water supply. It reacts with natural organics to produce lower molecular weight compounds that are more biodegradable and promote the growth of bacteria in distribution systems, which may have significant consequences for many Australian distribution systems where elevated water temperatures create a predisposition for bacterial growth. To avoid fouling, a biological filtration step is advisable after ozonation of water containing a DOC concentration of >1 mg/L (von Gunten, 2003).

The production of lower molecular weight compounds has been used to advantage in biological filtration processes. Ozonation can break up high molecular weight organics before filtration through a bed of granular activated carbon. The resulting low molecular weight compounds increase the amount of assimilable organic carbon (AOC) that can be used by bacteria that grow on the carbon, thereby reducing organic concentrations in the water. Ozone has a long history of use for disinfection, and for the control of taste, odour and colour. Ozone is more expensive than chlorine and has low solubility in water.

1 Ozone has been used in NSW for removal of algal toxins and for taste and odour control by Orange Council for over ten years. Similarly, ozone has been used for such control by MidCoast Water, Rous Water and Tweed Council for over four years.

PRACTICAL CONSIDERATIONS

Even though ozone systems are complex, using highly technical instruments, the process is highly automated and very reliable, requiring only a modest degree of operator skill and time to operate (USEPA 1999). Maintenance of ozone generators requires skilled technicians. If trained maintenance staff are not available at the plant, this work can be done by the equipment manufacturer.

Ozone is a toxic gas and the ozone production and application facilities should be designed to generate, apply, and control this gas, so as to protect plant personnel. Ambient ozone levels in plant facilities should be monitored continuously.

PERFORMANCE VALIDATION

Table IS 1.6.1 presents published C.*t* values for ozone that have been demonstrated as achieving a two log reduction in the target microorganism. These values are supplied for illustrative purposes only. For ozone C.*t* values that achieve a greater log reduction, the cited references should be consulted. The C.*t* value that is applied at a particular water treatment plant should be based on the microbial risk assessment for that particular water supply system.

Table IS1.6.1 Published C.t values for 99% (2 log) inactivation of various microorganisms by ozone 1,2,3

Microorganism	Ozone C.t value (mg/L.min)	Reference
Escherichia coli	0.02	USEPA 1999
Enteric viruses	0.6	USEPA 1999
Giardia	0.5-0.6	Wickramamayake *et al.* 1984
Cryptosporidium	32	USEPA 2010

Notes: (1) Water temperature is 5°C.

(2) pH 7 for *Giardia* and within the range of pH 6-9 for the other organisms.

(3) The values in the table are based on published values and should be viewed as the minimum values necessary to achieve effective disinfection.

The important conclusion to draw from Table IS1.6.1 is that ozone is more effective than chlorine, chloramines, and chlorine dioxide for the inactivation of viruses, *Cryptosporidium*, and *Giardia*.

WATER QUALITY CONSIDERATIONS

Ozone is highly sensitive to turbidity. Turbidity should be less than 1 NTU at the time of ozonation. The pH should be less than 8 for effective disinfection because ozone is unstable above pH 8 (at pH 8, half of the ozone is lost in less than 30 minutes).

PERSISTENCE

Due to its low solubility in water and instability above pH 8, an ozone residual cannot be maintained in a distribution system, particularly as temperature increases.

BY-PRODUCTS

Ozone is a powerful oxidant and can convert naturally-occurring bromide to bromine, and this can lead to the formation of brominated trihalomethanes (THMs), brominated acetic acids, bromopicrin, brominated acetonitriles, as well as the formation of bromate (USEPA 1999). However, the brominated THMs produced in ozonation usually occur in lower concentrations than chlorinated THMs produced by chlorination. The ADWG health-based guideline value for bromate is 0.02 mg/L, and bromate formation

can become a serious problem for waters containing bromide levels above 0.1 mg/L (von Gunten, 2003). Bromate formation can be reduced to a certain extent by ammonia addition and pH depression, but bromate is very difficult to remove once formed (von Gunten, 2003). An alternative method of disinfection should be used with high bromide waters.

Low molecular weight aldehydes, such as formaldehyde and acetaldehyde, have also been detected as by-products of ozonation.

OPERATIONAL CONSIDERATIONS

Given that where ozonation is used as a primary disinfectant it will be a critical control point (CCP) important operational considerations to ensure the effectiveness of the process are:

establishing target criteria and critical limits for the ozonation process (section 3.4.2);

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the ozonation process operates to the established target criteria and critical limits.

OPERATIONAL MONITORING

The table below summarises the operational monitoring that should be undertaken for ozone, based on recommendations from the New Zealand Ministry of Health (NZ MoH 2008).

Operational Parameter	**Monitoring**
pH	Online monitoring
Turbidity	Online monitoring
Ozone concentration	Online monitoring
Residual Concentration(1)	Online monitoring

(1) measured at a point representing the end of the contact period

REFERENCES

Langlais B, Reckhow DA, Brink DR. (1991). *Ozone in Water Treatment, Applications and Engineering*, Lewis Publishers, Inc., Chelsea, MI

LeChevallier MW and Au K-K. (2004). Water treatment and pathogen control. World Health Organization, Geneva.

New Zealand Ministry of Health (2008). Drinking-water Standards for New Zealand 2005 (Revised 2008).

United States Environmental Protection Agency (USEPA) (1999). Alternative disinfectants and oxidants guidance manual. Washington DC.

United States Environmental Protection Agency USEPA (2010). Long term 2 enhanced surface water treatment rule toolbox guidance manual. Washington DC.

von Gunten U. (2003). Ozonation of drinking water: Part II. Disinfection and by-product formation in presence of bromide, iodide or chlorine. *Water Research* 37: 1469-1487.

Wickramamayake GB, Rubin AJ, Sproul OJ. (1984). Inactivation of *Naegleria* and *Giardia* cysts in water by ozonation. *Journal of the Water Pollution Control Federation*, 56:983–988.

Wolfe RL, Stewart MH, Scott KN, McGuire MJ. (1989). Inactivation of *Giardia muris* and indicator organisms seeded in surface water supplies by peroxone and ozone. *Environmental Science and Technology*, 23:744–745.

Disinfection with ultraviolet light

INFORMATION SHEET 1.7

GENERAL DESCRIPTION

Germicidal ultraviolet (UV) light is generated by low and medium pressure mercury vapour lamps. UV irradiation disrupts the chemical bond of many organic molecules and damages nucleic acid and hence can be a potent disinfectant. The UV light effective for inactivating microorganisms is in the UV-B and UV-C ranges of the spectrum (200–300 nm), with maximum effectiveness around 265 nm.

The mechanism of disinfection by UV light differs considerably from the mechanisms of chemical disinfectants such as chlorine and ozone. Chemical disinfectants inactivate microorganisms by destroying or damaging cellular structures, interfering with metabolism, and hindering biosynthesis and growth (Snowball and Hornsey 1988). UV light inactivates microorganisms by damaging their nucleic acid, thereby preventing them from replicating and disrupting their ability to infect hosts.

UV irradiation has a minimal effect on the chemical composition or taste of water. Unlike chemical disinfectants, high dosage or over-dosing with UV light presents no danger, and is sometimes considered as a safety factor.

APPLICATION

UV light disinfection is a treatment option that can contribute to the effective implementation of a multi-barrier approach that reduces microbial risk in drinking water supplies. UV light disinfection can be used as the primary disinfectant for the inactivation of chlorine resistant pathogens (e.g. *Cryptosporidium* and *Giardia*), thereby reducing disinfection by-product formation.

However, UV light disinfection typically should not completely replace the use of chemical disinfection. This is because there are a number of other aspects to consider in managing the microbial risk of drinking water supplies, such as:

maintaining a disinfection residual within the distribution system;

management of taste and odour compounds;

controlling cyanobacteria;

deactivation of viruses that are not easily treated by UV light alone; and

ensuring there is an adequate multi-barrier approach for the entire drinking water system.

PRACTICAL CONSIDERATIONS

The equipment required for UV irradiation is fairly reliable, the technology required is relatively simple and controls for the process are being developed.

There are a number of factors that should be considered in relation to UV light, such as:

reliability of power supply, in particular the start-up and restart times should be factored into operational and response plans;

water quality aspects, such as algae, high colour and turbidity, hardness and organic matter, as they can reduce the amount of UV irradiation reaching microorganisms and necessitate higher doses of applied irradiation for effective disinfection (which can be managed if the percentage UV transmittance is known);

a site-specific mercury spill response plan should be established to minimise mercury release in the rare event of a lamp breakage;

units require regular cleaning and maintenance to remain effective; and

once the appropriate UV dose is determined and matched to the flow rate, exceeding the validated flow rate could result in the application of an insufficient UV dose, as a result of short-circuiting.

PERFORMANCE VALIDATION

Table IS1.7.1 presents published dosage rates for UV light that have been demonstrated as achieving a two log reduction in the target microorganism. These values are supplied for illustrative purposes only. For UV dosage rates that achieve a greater log reduction, the cited references should be consulted. Further information can be obtained from a review of existing data on the effectiveness of UV light against a range of specific pathogens undertaken by Chevrefils et al. (2006). The UV dosage rate that is applied at a particular water treatment plant should be based on the microbial risk assessment for that particular water supply system.

Table IS1.7.1 Published dosage rates to achieve 99% (2 log) inactivation of various microorganisms by UV irradiation

Target Pathogen	Dosage for drinking water (mJ/cm^2) to achieve 2 log removal
Cryptosporidium	**5.8**
Giardia	5.2
Viruses	100
E.coli	9

Based on Hijnen et al.(2006) and USEPA (2006)

The important conclusion to draw from Table IS1.7.1 is that, UV light will effectively inactivate protozoa and bacteria, but is less effective against viruses.

Wherever possible, a validated UV light system should be used, and preferably those systems that have been validated in accordance with the requirements of the USEPA *Ultraviolet disinfection guidance manual for the final long term 2 enhanced surface water treatment rule* (UVDGM) (2006). Other validation processes for UV light systems also exist (DVGW, 2006a, 2006b, 2006c; ONORM, 2001, 2003; and NWRI, 2012).

WATER QUALITY CONSIDERATIONS

The performance of UV disinfection is not affected at turbidity levels of 1 NTU, and UV light may remain effective at higher turbidities than 1 NTU, as long as the transmittance of UV light through the water is not compromised. However, the lower the turbidity of the water the more effective the performance of UV light will be. This reinforces the importance of percentage UV transmittance as a measure of the effectiveness of the applied UV dose to inactivate targeted pathogens.

UV irradiation is not pH dependent and the temperature effect between the ranges of 5 to 35°C is minimal (USEPA 2006). The presence of algae in the water being treated may reduce the UV transmittance and interfere with the UV disinfection process and should be considered in the design phase if the supply is prone to algal blooms.

Highly coloured water is not suitable for UV disinfection as the dissolved organic matter which gives the water its colour strongly absorbs the UV light, greatly reducing the effectiveness of the UV disinfection process. This again highlights the importance of measuring UV transmittance.

PERSISTENCE

At the proper dosage, UV light requires only a short contact time, but has the disadvantage that it leaves no residual disinfectant, which would provide an additional barrier within the distribution system.

BY-PRODUCTS

Few data are available on the by-products of UV disinfection. At the UV doses typical for drinking water supplies (less than 200 mJ/cm2), there is no evidence of the formation of by-products (DBP) or exacerbation of DBP if post UV disinfection occurs (USEPA 2006).

UV light has been reported to convert nitrate to nitrite (Sharpless and Linden, 2001). Given the typical values of Australian waters, the nitrate to nitrite conversion is unlikely to result in the exceedance of health guidelines for drinking water.

OPERATIONAL CONSIDERATIONS

Given that the UV process will be a critical control point (CCP), other important issues that will need to be considered to ensure the effectiveness of the process are:

establishing target criteria (section 3.4.2) and critical limits for the UV irradiation process, including UV transmittance and intensity;

preparing and implementing operational procedures (section 3.4.1) and operational monitoring (section 3.4.2) for the process;

preparing corrective action procedures (section 3.4.3) in the event that there are excursions in the operational parameters; and

undertaking employee training (section 3.7.2) to ensure that the UV irradiation process operates to the established target criteria and critical limits.

It is recommended that validation is undertaken for each system to ensure appropriate treatment is in place for the water quality and level of risk.

OPERATIONAL MONITORING

As there is no disinfection residual to measure following UV light treatment, other operational aspects of UV light systems should be monitored to ensure that the treatment system is operating as expected. Examples of monitoring parameters are listed below, recognising that each system will need to develop its own operational monitoring specifications reflecting its unique circumstances.

UV dose

Flow rate

UV transmittance

Lamp outage

Lamp age

UV intensity

Another issue that needs to be considered with respect to UV light is that the performance of the UV lamps deteriorates over time, so that lamps should be changed at the frequency recommended by the manufacturer. Typically the loss of UV light output is around 25% over 12,000 hours operation.

Furthermore, biofilm, which can accumulate on the sleeve surrounding the lamp, should be regularly removed from the sleeve. Many UV light systems now have automated cleaning systems.

REFERENCES

Austrian Standards Institute (ÖNORM) (2001). ÖNORM M 5873-1: Plants for the disinfection of water using ultraviolet radiation: Requirements and testing. Part 1. Low pressure mercury lamp plants. Österreichisches Normungsinstitut, A-1021 Vienna, Austria.

Austrian Standards Institute (ÖNORM) (2003). ÖNORM M 5873-2: Plants for the disinfection of water using ultraviolet radiation: Requirements and testing. Part 2. Medium pressure mercury lamp plants. Österreichisches Normungsinstitut, A-1021 Vienna, Austria.

Chevrefils G and Caron E (2006). UV Dose Required to Achieve Incremental Log Inactivation of Bacteria, Protozoa and Viruses, Trojan Technologies Inc., London, ON, Canada.

German Association for Gas and Water (DVGW) (2006a). *UV Devices for Disinfection in the Water Supply Part 1: Requirements Related to Composition, Function and Operation*, in *Technical Rule, Code of Practice W294-1*. Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

German Association for Gas and Water (DVGW) (2006b). *UV Devices for Disinfection in the Water Supply Part 2: Testing of Composition, Function and Disinfection Efficiency* in *Technical Rule, Code of Practice W294-2* Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

German Association for Gas and Water (DVGW) (2006c). *UV Devices for Disinfection in the Water Supply; Part 3: Measuring Windows and Sensors for the Radiometric Monitoring of UV Disinfection Devices: Requirements, Testing and Calibration* in *Technical Rule, Code of Practice W 294-3* Deutsche Vereinigung des Gas- und Wasserfaches, Bonn, Germany.

Hijnen WAM, Beerendonk EF and Medema G.J. (2006). Inactivation credit of UV radiation for viruses, bacteria and protozoan (oo)cysts in water: A review. *Water Research* 40: 3-22.

National Water Research Institute (NWRI) (2012). *Ultraviolet Disinfection Guidelines for Drinking Water and Water Reuse*, Third Edition, California, United States of America.

Sharpless CM and Linden KG. (2001). UV photolysis of nitrate: Effects of natural organic matter and dissolved inorganic carbon, and implications for UV water disinfection. *Environmental Science and Technology* 35:2949–2955.

United States Environmental Protection Agency (US EPA) (2006). *Ultraviolet disinfection guidance manual for the final long term 2 enhanced surface water treatment rule.* US EPA Office of Water: Washington D.C.

Other disinfectants

INFORMATION SHEET 1.8

This information sheet provides information on a number of other chemicals that have been used, or are proposed for use, as disinfectants of drinking water. Currently, these chemicals are not recommended for use as disinfectants in municipal water systems, but may be of use in specific, small scale applications, where the use of more traditional disinfectants may not be practical. Prior to use, expert advice should be sought regarding the appropriateness of using these chemicals as disinfectants.

BROMINE

Definition and uses

Bromine has been widely used to disinfect swimming pools through the addition of solid bromine-releasing agents such as N-bromo-N-chloro-5,5-dimethylhydantoin or dibromocyanuric acid. Bromine chloride (BrCl) is under investigation for large-scale use, such as the control of biofouling in cooling towers or wastewater disinfection, as it is much less corrosive than liquid bromine and has sufficient vapour pressure to enable it to be metered in equipment similar to that used for chlorine.

Relative to chlorine, bromine is costly to use on a municipal-scale water supply system.

Health effects

Bromine is corrosive to human tissue in a liquid state and its vapours irritate the eyes and the throat. Bromine vapours are very toxic if they are inhaled. Bromine concentrations of around 0.5 mg/L in swimming pools cause eye and mucous membrane irritation and can lead to odour nuisance. Bromine aggressively reacts with metals and it is a corrosive material. Security measures should be taken when bromine is transported, stored or used.

IODINE

Definition and uses

Iodine has been used as a disinfectant for small drinking water supplies; however, like bromine, it is costly to use on a municipal scale. It is not recommended for regular use as a disinfectant due to possible health effects associated with long-term consumption. It can, however, be used for emergency water disinfection.

Health effects

Iodine is an essential trace element for humans and is used in the synthesis of thyroid hormones. The recommended dietary intake for adults ranges from 0.03 mg/day to 0.15 mg/day. Iodine is efficiently absorbed by the gastrointestinal tract and deposited in the thyroid gland, the eye, and muscle tissue. More than 70% is found in the thyroid gland. High oral doses (more than 30 mg/kg body weight) of iodine can be lethal. Lower doses (3.3 mg/kg body weight) have been used to treat asthmatic patients without adverse effects.

Chronic exposure to high amounts of iodide in the diet (over 2 mg/day) can result in a condition known as iodism. Symptoms resemble those of a sinus cold. Long-term consumption of iodinated drinking water has not been associated with adverse health effects in humans. Experiments with humans who drank water containing up to 1 mg/L iodine for five years showed no signs of iodism or hypothyroidism, but some changes in uptake of iodine by the thyroid gland were observed.

Animal studies using chickens susceptible to autoimmune thyroiditis reported an increase in the incidence of the disease when they were given high doses of iodide in their drinking water (200 mg/L). Excessive iodide consumption may increase the incidence of this disease in humans.

Iodide has not been shown to increase the incidence of cancer of the thyroid in laboratory animals.

No data are available on the mutagenic activity of iodine.

For further information refer to the Iodine Fact Sheet contained in Part V of these guidelines.

SILVER

Definition and uses

Silver is a weak biocide/bacteriostat that has been used occasionally for disinfection, particularly in point-of-use devices. However, there is no reliable evidence that these products worked effectively to kill micro-organisms. A long exposure time of several hours to days is required for any biocidal effect to be observed (Bosch et al, 1993). This is generally not practical in the supply of potable water. In addition, for protozoan pathogenic microorganisms, antiprotozoan activity does not appear to be significant for metal ions acting alone (Cassels et al. 1995). The Australian Pesticides and Veterinary Medicines Authority provide a comprehensive review on the effectiveness of silver as a disinfectant (http://www.apvma.gov.au/use_safely/pool/background.php).

For the reasons outlined above silver is not recommended for use as a disinfectant for municipal drinking water supplies.

Health effects

Although silver can be found in many biological substances, it is not considered an essential trace element for mammals. It has been estimated that less than 10% of dietary silver is absorbed by the gastrointestinal tract.

Silver is stored mainly in the liver and skin and is capable of binding to amino acids and proteins. The best-known clinical condition of silver intoxication is argyria, which results in a bluish-grey metallic discolouration of the skin, hair, mucous membranes, mouth and eye. Most cases have been associated with self-administration of silver preparations, or occupational exposure to silver and silver compounds.

Experiments with laboratory rats and mice have reported similar results. Very high concentrations of silver in drinking water (over 600 mg/L) for a lifetime caused discolouration in the thyroid and adrenal glands, the choroids of the eyes, the choroid plexus of the brain, and the liver and kidney. Some hypoactive behaviour was also reported.

No data are available on the carcinogenicity of silver. Silver salts are not mutagenic in tests with bacteria, but can induce damage in mammalian DNA.

For further information refer to the Silver Fact Sheet contained in Part V of these guidelines.

REFERENCES

Bosch, A., Diez, J.M.and Abad, F.X. (1993). Disinfection of human enteric viruses in water by copper:silver and reduced levels of chlorine. *Water Science and Technology*, 27, 3-4: 351-356.

Cassells, J.M., Yahya, M.T., Gerba, C.P. and Rose, J.B. (1995). Efficacy of a combined system of copper and silver and free chlorine for inactivation of *Naegleria fowleri* amoebas in water. *Water Science and Technology*,31: 119-122.

Sampling Information – handling requirements and preservation

INFORMATION SHEET 2.1

This information sheet gives information on general handling requirements in sampling for chemical, physical, radiological and microbial characteristics.

SAMPLING FOR CHEMICAL, PHYSICAL AND RADIOLOGICAL CHARACTERISTICS

Table IS2.1.1 provides general advice on the handling requirements for chemical, physical and radiological characteristics, based on Australia Standard *AS/NZS 5667.1:1998 Water Quality – Sampling Part 1: Guidance on the design of sampling programs, sampling techniques and the preservation and handling of samples*. To ensure that samples are collected and transported in an appropriate manner, advice of an analyst should be sought before taking a sample.

Metal Fractions

Metals can be divided into various fractions as determined by the analytical information:

filterable metals (soluble or dissolved, macromolecular and colloidal metals) – those constituents of an unacidified sample that pass a 0.45µm membrane filter;

suspended metals – those constituents of an unacidified sample that are retained on a 0.45µm membrane filter;

total metals – the concentration of metals determined on an unfiltered sample after vigorous digestion, or the sum of the concentrations of metals in both the filterable and suspended fractions. Total metals include all metals inorganically and organically bound, both filterable and particulate;

acid-extractable metals – the concentration of metals in solution after treatment of an unfiltered sample with hot mineral acid;

readily acid-soluble aluminium – see Fact Sheet on Aluminium.

The fraction(s) to be analysed will determine the requirements for sample handling and preservation. It is generally advisable to collect two samples, one for total metals and one for dissolved metals.

SAMPLING FOR MICROBIAL CHARACTERISTICS

As per Australian Standard AS/NZS 2031:2012 – Selection of containers and preservation of water samples for microbiological analysis, when sampling for microbial characteristics the following dot points must be taken into consideration:

The sample container used to collect the sample must be sterile.

Sufficient sodium thiosulfate should be added to the sterile sample container to neutralise all residual chlorine.

When collecting water samples that may contain concentrations of heavy metals that may be toxic to bacteria, addition of EDTA to the sample containers is recommended.

The size of the container used depends upon the number and type of tests to be carried out, but the containers must be of sufficient volume for the all the tests required, with adequate head space to allow for sample mixing.

All samples should be refrigerated or chilled in ice coolers during transport.

Drinking water samples should be taken directly from a service pipe, or a dedicated sampling point, not from an intermediate tank or cistern. As described in *AS/NZS 5667.5:1998 Water Quality – Sampling Part 5: Guidance on sampling of drinking water and water used for food and beverage processing*, sampling taps should be sterilised by flame or alternative methods of equivalent efficacy, for example soaking in chlorine solution, and should be maintained in good order. The water discharged by flushing should be able to run off freely.

On days of total fire ban, where sterilisation by flame is not possible, an alternative method of equivalent efficacy should be used.

Samples should be analysed within six hours of collection. If this is not possible, then the samples must be transported and stored at between 2°C and 10°C. Samples must not be frozen.

Where logistics do not allow examination within six hours, storage may be prolonged and samples may be examined up to 24 hours after collection, provided that they are kept cool (between 2°C and 10°C) and in the dark.

As noted in AS/NZS 2031:2012, if samples are to be analysed for free-living protozoans, including amoebae, the samples should not be refrigerated during transport and storage, but should be held at ambient temperature, preferably near 20°C, but the sample temperature should not exceed 30°C.

Further advice on appropriate sampling technique can be found in Australian Standards *AS/NZS 5667.1:1998* and *AS/NZS 5667.5:1998*.

Table IS2.1.1 *Special handling requirements in sampling for chemical, physical and radiological characteristics (data compiled from AS/NZS 5667.1:1998)*

Characteristic	Container	Minimum sample size (mL)	Preservation procedure	Maximum holding period	Comments
Aluminium	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Arsenic	P(A), G(A)	500	Add HNO_3 to pH <2	28 days	
Boron	P	100	None required	28 days	Fill container completely to exclude air
Cadmium	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Chloride	P, G	100	None required	28 days	
Chlorine residual	P, G	500	Analyse immediately	5 minutes	Keep sample out of direct sunlight
Chromium (total)	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Chromium (VI)	P(A), G(A)	100	Refrigerate	24 hours	Sample container should be thoroughly rinsed. Avoid adding reagents
Colour	P, G	500	Refrigerate and store in the dark	2 days	
Copper	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Cyanide	P, G	500	Add NaOH to pH >12. Refrigerate in the dark	24 hours	Remove sulfide
Fluoride	P	200	None required	28 days	PTFE containers are not suitable
Hardness	P	100	None required	7 days	Fill container completely to exclude air

Characteristic	Container	Minimum sample size (mL)	Preservation procedure	Maximum holding period	Comments
			Add HNO_3 to pH <2 and refrigerate	28 days	Fill container completely to exclude air. Acidification permits the determination of calcium and other metals from the same sample
Iron	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Lead	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Manganese	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Mercury	G(A)	500	Add HNO_3 to unfiltered sample to pH <1. Add $K_2Cr_2O_3$	28 days	Consult analyst for further instruction
Metals (general)	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Metals (filterable)	P(A), G(A)	100	Filter immediately, add HNO3 to pH <2	28 days	0.45 m filter
Nitrate	P, G	500	Refrigerate	24 hours	Unfiltered samples
			Filter on site (0.45 m cellulose acetate membrane filter) and freeze	1 month	Consult analyst – depends on analytical method
Odour	P, G	500	Refrigerate	6 hours	Analyse as soon as possible
Oxygen, dissolved	P or G	300	None required	Determine in the field	Avoid excessive turbulence, to minimise oxygen entrainment
	G		Winkler acidification	24 hours	Store in dark
Pesticides (organochlorine, organophosphorous, and nitrogen-containing)	G(s)	1000 to 3000	Refrigerate[1]	7 days	Extract on site where practical. Consult with analyst
pH	P, G	100	Refrigerate	6 hours	
Poly aromatic hydrocarbons (PAHs)	G(S)	1000	Refrigerate and store in dark[2]	7 days	Extract on site where practical. Consult with analyst
Radioactivity gross *alpha* and *beta* activity	P, G	1000	Add HNO_3 to pH <2	28 days	Fill container completely to exclude air. Consult with analyst
Selenium	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	
Sodium	P	100	None required	28 days	
Sulfate	P, G	200	Refrigerate	7 days	
Taste	G	500	None required	24 hours	Analyse as soon as possible
Temperature	-	-	None required	Analyse immediately	Determine *in situ*
Total dissolved solids	P, G	500	Refrigerate	24 hours	Fill container completely to exclude air
Trihalomethanes	G, vials with PTFE-faced septum	100	Add 2 mL of 5% ascorbic acid solution	14 days	Fill container completely to exclude air

Characteristic	Container	Minimum sample size (mL)	Preservation procedure	Maximum holding period	Comments
Turbidity	P, G	100	None required	24 hours	Preferably determine on site or *in situ*
Zinc	P(A), G(A)	100	Add HNO_3 to pH <2	28 days	

Container	P	= Plastic (polyethylene or equivalent)
	G	= Glass
	G(B)	= Glass, borosilicate
	P(A), G(A)	= Rinsed with 50% HNO_3
	G(S)	= Glass, rinsed with organic solvent, PTFE cap liner
	PTFE	= Polytetrafluoroethylene
Preservation	Refrigerate	= Store between 1° and 4°C in the dark, do not freeze
	HNO_3	= Nitric acid (hydrochloric acid may be used in this context but nitric acid is preferred)
	NaOH	= Sodium hydroxide solution (40% w/v)
	$K_2Cr_2O_3$	= Potassium dichromate

2 If sample is chlorinated, for each 1000 mL of sample add 80 mg of sodium thiosulfate to container prior to sample collection.
3 If sample is chlorinated, for each 1000 mL of sample add 80 mg of sodium thiosulfate to container prior to sample collection.

Radiological monitoring and assessment of performance

INFORMATION SHEET 2.2

SCREENING OF WATER SUPPLIES

The process of identifying individual radioactive species and determining their concentration requires sophisticated and expensive analysis, which is normally not justified because concentrations in most circumstances are very low. A more practical approach is to use a screening procedure, where the total radioactivity present in the form of alpha and beta radiation is determined without regard to the identity of specific radionuclides.

The 'screening' levels that are recommended for both gross alpha and gross beta activity are 0.5 becquerels per litre (Bq/L). The gross beta measurement includes a contribution from potassium-40, a natural beta emitter, which occurs naturally in a fixed ratio to stable potassium. Potassium is an essential element for humans, and is absorbed mainly from ingested food. Potassium-40 does not accumulate in the body but is maintained at a constant level independent of intake. The contribution of potassium-40 to beta activity is therefore subtracted following a separate determination of total potassium. The specific activity of potassium-40 is 30.7 becquerels per gram of potassium. However, not all the radiation from potassium-40 appears as beta activity. The beta activity of potassium-40 is 27.6 becquerels per gram of stable potassium, which is the factor that should be used to calculate the beta activity due to potassium-40. Although potassium-40 can make a significant contribution to the gross beta activity of drinking water (in Bq/L), this translates into a trivial dose in mSv. For example, assuming potassium-40 activity was at the 0.5 Bq/L screening level for total beta activity, the dose would only be 0.003 mSv per year, based on the calculation given in Section 7.6.2 and using the ICRP (1996) 'dose per unit intake' value for potassium-40 of 6.2 x 10-6 mSv/Bq.

If the 'screening' levels are not exceeded, there is no need for further assessment. The recommended screening levels provide a good margin of safety against the dose-based guideline values. The likely worst case leading to the highest exposure is where these gross activities are due entirely to radium-226 (an alpha emitter) and radium-228 (a beta emitter). The total dose corresponding to total alpha and total beta activities that are just within the respective screening level of 0.5 Bq/L will be approximately 0.35 millisieverts (mSv) per year, with 0.1 mSv from radium-226 and 0.25 mSv from radium-228. Water that meets the screening guideline will result, at worst, in an annual dose of approximately one-third of the minimum dose at which intervention should be considered (see Table IS2.2.1). The worst case exposure is based on a combination of the likelihood of occurrence in the Australian environment and the relatively high mSv/Bq ratio.

If either or both screening levels are exceeded, further investigation is necessary to identify the nature of the radioactivity. It should be emphasised that the screening level is intended only as a practical means to ascertain if further consideration of the radiological quality of the water supply is needed. It should never be regarded as a guideline value, or even as an indicative water quality target.

DOSE ASSESSMENT

If the screening level for gross alpha or gross beta activity is exceeded, specific radionuclides should be identified and their activity concentrations determined. This may involve taking a resample if the volume of the original sample is inadequate to allow specific radionuclide analysis. Activity concentrations for the most common sources of emissions, radium-226 and radium-228, should be evaluated at this stage. If radium does not account for all the gross alpha and beta emissions, then additional radionuclides will need to be identified. In accounting for all gross alpha and beta activity, the analytical service provider needs to provide information on counting and other errors associated with the determinations.

The annual dose rate from each radionuclide can be calculated using the method described in Section 7.6.2.

If the sum of the annual doses from all radionuclides is less than 0.5 mSv, no further action is required and routine monitoring can continue. If the sum of the annual doses from all radionuclides exceeds 0.5 mSv, it is not appropriate to rely on a single analysis to determine annual exposure. In this case, radionuclides should be sampled quarterly to obtain a profile of radiological water quality, as some water supplies show seasonal variations.

The quarterly results should be reviewed as they become available, to ensure that there are no immediate problems. Otherwise, a final assessment of annual dose can be made when at least four results are available and the average concentration of each radionuclide can be used to calculate the annual doses.

OPERATIONAL RESPONSE

The operational response will depend on the estimated annual dose determined by the sum of the contribution from each radionuclide present in the water.

If the total annual dose is less than 0.5 mSv, the guideline level for intervention has not been exceeded and routine monitoring can be maintained.

If the total annual dose lies between 0.5 and 1.0 mSv, the guideline value for intervention has not been exceeded; however, discussions should be held with the relevant health authority to determine the frequency of ongoing sampling.

If the total annual dose exceeds 1.0 mSv, the guideline exposure for considering intervention has been exceeded. The water service provider and the relevant health authority should assess the results and examine options to reduce the levels of exposure. Water supply providers should consider operational changes that can be implemented at minimal cost to reduce annual exposures. For example, water could, wherever possible, be taken preferentially from bores with the lowest radionuclide concentrations.

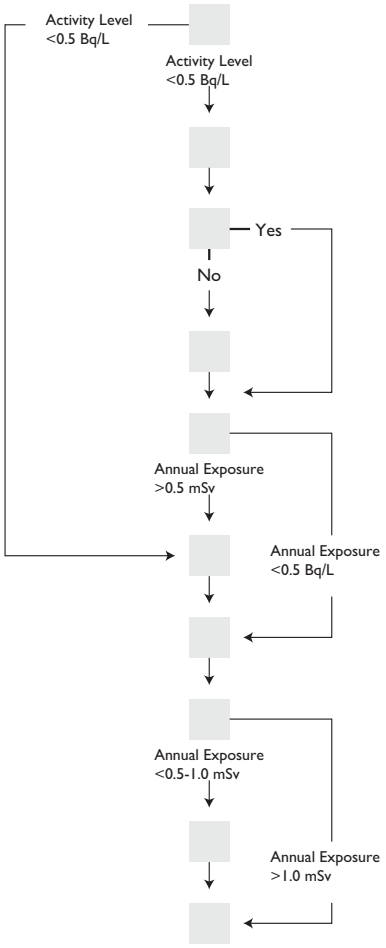
A total annual dose that exceeds 10 mSv is unacceptable for drinking water and immediate action should be taken to reduce the dose to below guideline levels.

Recommendations on the response process are presented in Table IS2.2.1. The monitoring and assessment process is further illustrated in Figure IS2.2.1.

***Table IS2.2.1** Summary of operational responses*

Dose level (mSv per year)	**Response**
< 0.5	1. Continue routine monitoring.
0.5-1	1. Consult with relevant health authorities. 2. Review frequency of ongoing sampling. 3. Evaluate operational options to reduce exposure.
>1-10	1. Consult with relevant health authorities. 2. Assess in detail possible remedial actions, taking into account potential cost-effectiveness of actions. 3. Implement appropriate remedial action on the basis of the cost-benefit evaluation.
> 10	1. Water not suitable for consumption on the basis of radioactivity levels. 2. Consult with relevant health authorities. 3. Immediate intervention is expected and remedial action must be taken to reduce doses to below the guideline value of 1.0 mSv.

Figure IS2.2.1 *Flowchart showing how to determine whether the radiological quality of drinking water complies with the Guidelines*

Step	Activity	Process flow	Guidance note
1	Determine gross alpha and [gross beta excluding - K40].		
2	Retest for gross alpha and [gross beta excluding - K40] and determine Ra-226 and Ra-228 activity levels.		
3	Are all gross alpha and gross beta accounted for?		
4	Determine the activity levels of additional radionuclides.		
5	Calculate annual exposure.		
6	Complies with Guidelines. Continue rountine monitoring.		
7	Commence quarterly monitoring.		Monitoring includes steps 2 to 4.
8	Calculate annual exposure based on quarterly monitoring.		
9	Complies with ADWG. Continue monitoring at frequency agreed with health departement.		
10	Exceeds guideline. Consider intervention.		

METHODS OF ANALYSIS

Gross alpha and beta activity concentration

For analysis of drinking water for gross alpha and beta activity, the most common approach is to evaporate a known volume of the sample to dryness and measure the activity of the residue. As alpha radiation is easily absorbed within a thin layer of solid material, the reliability and sensitivity of the method for alpha determination may be degraded in samples with a high content of total dissolved solids.

Where possible, standard methods should be used to determine concentrations of gross alpha and beta activities. Table IS2.2.2 lists the three procedures that are recommended.

Table IS2.2.2 *Recommended methods for the analysis of gross alpha and beta activities in drinking water*

Method reference	Technique	Detection limit	Application
ISO 9696 (2007)	Evaporation	0.02-0.1 Bq/L	Groundwater with TDS greater than 0.1 g/L
AS 3550.5 (1990)	Evaporation	0.02 Bq/L	Surface water and groundwater with TDS 0.1 g/L
APHA, AWWA, WEF(1998)	Co-precipitation	0.02 Bq/L	Surface and groundwater (low Fe) (TDS not a factor)

AS = Australian Standard (of Standards Australia)
AWWA = American Water Works Association
APHA = American Public Health Association
Bq = becquerel
ISO = International Organization for Standardization
TDS = total dissolved solids
WEF = Water Environment Federation.

The determination of gross beta activity using either of the evaporation methods in Table IS2.2.2 includes the contribution from potassium-40. An additional analysis of total potassium is therefore required.

The co-precipitation technique shown in Table IS2.2.2 excludes the contribution due to potassium-40, so determination of total potassium is not necessary. This method is not suitable for assessment of water samples containing fission products such as caesium-137. However, under normal circumstances, concentrations of fission products in Australian drinking water supplies are so low that they cannot be detected.

Analytical methods for specific radionuclides

Generally, Australian standard or international standard methods are not available for key natural radionuclides such as radium-226 and radium-228; however, suitable methods have been published in the literature. Suggested methods for specific radionuclides are included the relevant Fact Sheets.

Sample handling and pretreatment

Water samples should be pretreated to prevent significant losses of radionuclides from solution following collection and in transit to a laboratory.

Details of appropriate procedures for the handling of water samples, including suitable containers and pretreatment methods, are described in the relevant Australian/New Zealand Standards (AS/NZS 1998a and 1998b).

Analytical methods for potassium-40

It is impractical to use a radioactive measurement technique to determine the concentration of potassium-40 in a water sample. This is because gamma ray analysis is not very sensitive and it is difficult chemically to isolate the radionuclide from solution. Because the ratio of potassium-40 to stable potassium is fixed, chemical analysis for potassium is recommended. A measurement sensitivity of 1 mg/L for potassium-40 is adequate for monitoring purposes; this can readily be achieved by atomic absorption spectrophotometry or specific ion analysis. The activity due to potassium-40 can then be calculated using a factor of 0.0276 Bq of beta activity per milligram of potassium.

Sampling frequency

New water supplies and those not previously sampled should be sampled often enough to characterise the radiological quality of the water supply and to assess any seasonal variation in radionuclide concentrations. This should include analysis for radon. Quarterly sampling over the first year should provide sufficient data to establish the baseline. Once the radiological quality of a supply has been established, sampling can be less frequent – every two years for groundwater supplies, every five years for surface water supplies.

Reporting of results

The analytical results for each sample should contain the following information:

sample identifying code or information;

reference date and time for the reported results (e.g. sample collection date);

identification of the standard analytical method used or a brief description of any non-standard method used;

identification of any radionuclides or type of total radioactivity determined;

calculated concentration or activity value using the appropriate blank for each radionuclide;

estimates of the counting uncertainty and total propagated uncertainty;

decision level (in units consistent with the counting uncertainty) and nominal minimum detectable concentration for each radionuclide or parameter analysed.

The estimate of total propagated uncertainty of the reported result should include the contributions from all the parameters within the analytical method (i.e. counting and other random and systematic uncertainties).

REFERENCES

APHA/AWWA/WEF (American Public Health Association/American Water Works Association/Water Environment Federation) (2005). *Standard methods for the examination of water and wastewater*. 21st edition, Washington DC.

AS (Australia Standards) AS 3550.5 (1990). Waters – Determination of gross alpha and gross beta activities. Standards Association of Australia, Sydney.

AS/NZS (Australia and New Zealand Standards) (1998a). AS/NZS 5667.1:1998 Water quality – Sampling – Guidance on the design of sampling programs, sampling techniques and the preservation and handling of samples.

AS/NZS (Australia and New Zealand Standards) (1998b). AS/NZS 5667.5:1998 Water quality – Sampling – Guidance on sampling of drinking water and water used for food and beverage processing.

ICRP (International Commission on Radiological Protection) (1996). Age-dependent doses to members of the public from intake of radionuclides: Part 5 Compilation of ingestion and inhalation dose coefficients. ICRP Publication 72, Pergamon Press, Oxford, United Kingdom.

ISO (International Organization for Standardization 9096 (2007). Water quality – measurement of gross alpha activity in non-saline water – thick source method. International Standard 9696. International Organization for Standardization, Geneva, Switzerland.

Statistics – Visualising data

INFORMATION SHEET 3.1

Visualization is critical to data analysis. While tables are necessary to record the data, it is usually very difficult to distinguish pattern in tables of numbers, particularly for large data sets. Graphs, however, allow the reader to see complex data sets simply and concisely. Plots can reveal hidden structure in the data, and outlying or unusual results, and they enable preconceived ideas to be challenged. Visualization of data is best described using an example.

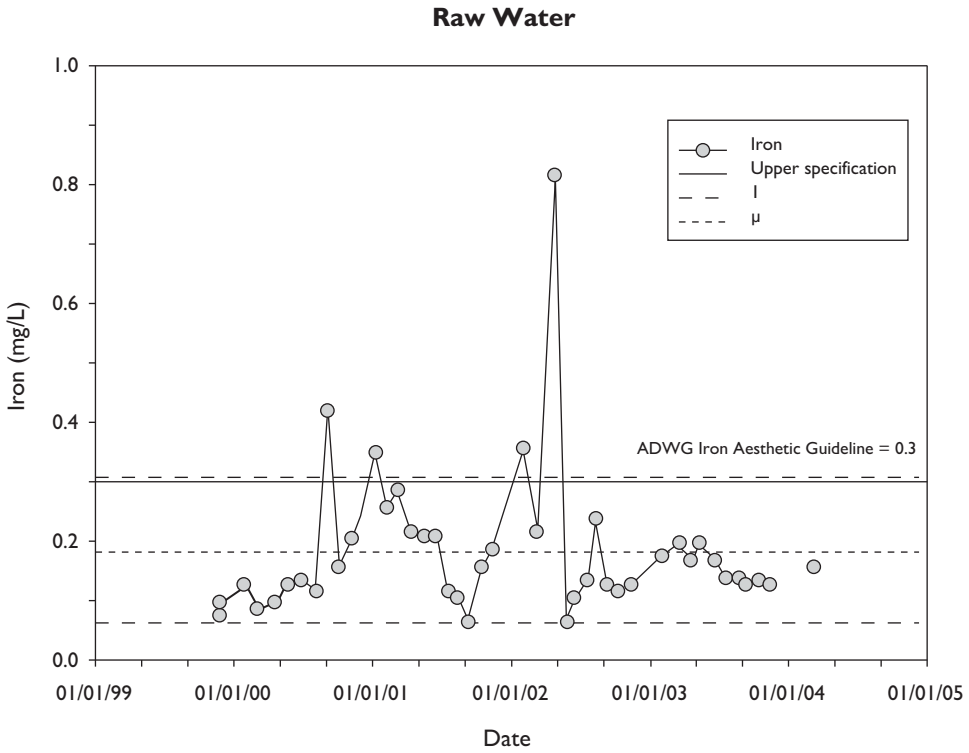
Table IS3.1.1 contains a typical water data set that may have been collected as part of an operational monitoring program. A simple graphical representation of one analyte (iron in this case) from these data is presented in Figure IS3.1.1. The graph makes clear a number of features of the data that are lost in the table. The average of the data can be seen to be close to 0.2 mg/L and the majority of data lie between ± one standard deviation of the mean. Of note are 4 data points that exceed the aesthetic guideline value listed in the Guidelines, as well as periods where consecutive increases or decreases in the data have occurred. Such a chart is often referred to as a quality control or Shewhart chart, and is discussed further in Information Sheet 3.5.

Table IS3.1.1 *Example of a water quality data set*

Date	Iron (mg/L)	Manganese (mg/L)	Total Hardness as $CaCO_3$ (mg/L)	Colour (PCU)	Turbidity (NTU)	Electrical Conductivity (µS/cm)
28/11/1999	0.08	0.02				
28/11/1999	0.1	0.005				
30/01/2000	0.13	0.02				
5/03/2000	0.09	0.01	37	15	0.5	125
16/04/2000	0.1	0.01				
21/05/2000	0.13	0.01	18			105
25/06/2000	0.14	0.01	29	24	1	110
6/08/2000	0.12	0.005	35	12	0.7	120
3/09/2000	0.42	0.02	15	145	13	75
3/10/2000	0.16	0.01	15	27	1.6	99
6/11/2000	0.21	0.01	26	9	0.6	120
4/12/2000	0.25	0.01	27	18	0.6	125
8/01/2001	0.35	0.03	27		0.5	125
5/02/2001	0.26	0.02	24		0.8	120
5/03/2001	0.29	0.01	14	58	4.6	84
9/04/2001	0.22	0.01	24		0.9	110
15/05/2001	0.21	0.01	25		0.6	115
17/06/2001	0.21	0.01	26		0.6	115
16/07/2001	0.12	0.01	23		0.9	105
12/08/2001	0.11	0.01	23	8	0.7	115
9/09/2001	0.07	0.01	33		0.9	130
15/10/2001	0.16	0.01	24	19	0.8	110
11/11/2001	0.19	0.01	29	5	0.6	120
3/02/2002	0.36	0.03	28	5	0.9	136
11/03/2002	0.22	0.01	32	6	1	155
28/04/2002	0.81	0.04	43	5	2.6	218
26/05/2002	0.07	0.01	28	8	0.3	125

Date	Iron (mg/L)	Manganese (mg/L)	Total Hardness as $CaCO_3$ (mg/L)	Colour (PCU)	Turbidity (NTU)	Electrical Conductivity (µS/cm)
16/06/2002	0.11	0.01	25	6	0.6	116
21/07/2002	0.14	0.01	27	4	0.4	112
11/08/2002	0.24	0.01	18	18	12	82
8/09/2002	0.13	0.01	24	7	1.2	110
13/10/2002	0.12	0.01	31	5	0.4	131
10/11/2002	0.13	0.01	31	5	0.5	130
2/02/2003	0.18	0.01	16	19	1.7	85
16/03/2003	0.2	0.01	21	6	0.6	98
20/04/2003	0.17	0.01	24	5	0.5	108
11/05/2003	0.2	0.01	24	4	0.5	110
22/06/2003	0.17	0.01	31	3	0.9	111
21/07/2003	0.14	0.01	21	10	1	92
24/08/2003	0.14	0.01	27	4	0.5	106
7/09/2003	0.13	0.01	27	5	0.4	108
12/10/2003	0.14	0.01	32	5	0.5	125
16/11/2003	0.13	0.01	27	7	0.4	105
7/03/2004	0.16	0.01	31	4	0.6	128

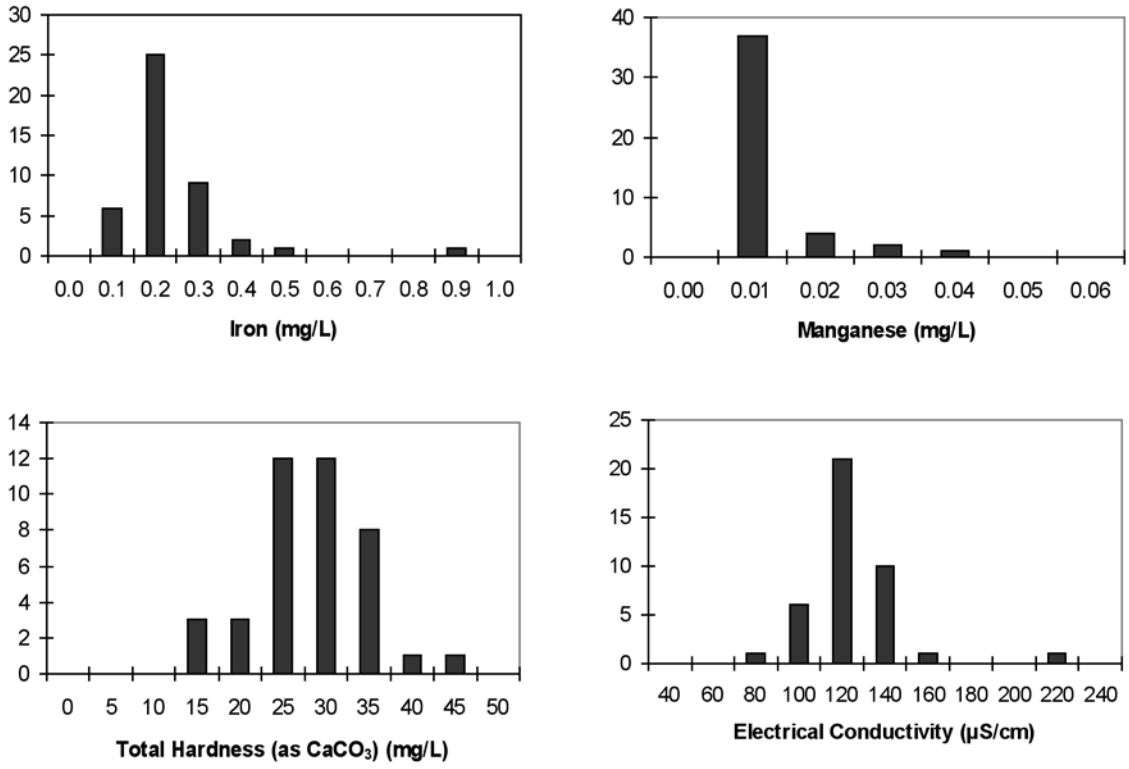
Figure IS3.1.1 *Graphical presentation of iron data from Table IS3.1.1*



Another useful graphical representation of data is a frequency histogram (Figure 3.1.2) which can identify individual features of the distribution of the data and their relationship with other analytes. Frequency histograms can provide an indication of the normality of the data (or lack thereof in most cases). For example, compare the frequency histogram for total hardness with that for iron and manganese; the latter two show significant departure from normality. Tests of normality (e.g. using Anderson's test or Maximum Likelihood) can be considered but are unlikely to be definitive when sample sizes are small, as is usually

the case in water quality data. Further aspects of the distribution (e.g. skewness and kurtosis) also need to be considered. In practice, however, water quality data are rarely normally distributed. Accordingly, the use of alternative distributions (most notably, lognormal distributions) should be considered when significant departures from normality are observed.

Figure IS3.1.2 *Histogram of data for selected analytes from Table IS3.1.1. Note that most data sets shown are evidently non-normal and skewed*



In summary, the first step in data analysis is to present the data graphically, ideally both for the 12-month reporting period and for the full period for which data is available, or for a ten-year period.

If more detailed or involved data analysis techniques are to be considered the advice of a statistician should be sought.

REFERENCE

AS/NZS 5667.1:1998 (1998). Water Quality – Sampling Part 1: Guidance on the design of sampling programs, sampling techniques and the preservation and handling of samples. Standards Association of Australia

Statistics – Assessing performance

INFORMATION SHEET 3.2

PRELIMINARY CONSIDERATIONS

In deciding how the performance of a water supply system should be assessed, it is necessary to consider:

the statistical implications of the assessment mechanism;

possible health implications of using different statistical measures;

community perceptions of what constitutes good quality water.

Three commonly used procedures measure performance respectively against a maximum value, a mean, or a percentile (Ellis 1989).

ASSESSING PERFORMANCE AGAINST A MAXIMUM VALUE

Using this approach, performance is measured by quoting the percentage of scheduled samples tested that are below the guideline value. Although the approach is used often and is superficially easy to understand, it has some serious deficiencies:

While measurements will show how a system is performing at the time of sampling, there is no way of determining what the water quality is like between sampling events. Statistical procedures cannot be used to indicate whether or not the measurements are representative of the quality at other times. (Other methods of assessing performance, however, can provide this information.)

There is no way of reliably estimating what the true maximum value is, as this may well occur between samples. Any sampling program can only provide a biased estimate of the true maximum value, which it will invariably underestimate. There is always the possibility that the next sample analysed may have a higher value.

ASSESSING PERFORMANCE AGAINST A MEAN

Performance is assessed by comparing the mean value of measurements with the guideline value over a period (usually 12 months). Such an approach has a number of attractions:

For characteristics not related to health, the guideline values are generally set at values that have the potential to generate a change that is noticeable by the customer. In many cases, it is sudden large increases in a value that can bring an increased number of consumer complaints. Therefore, when looking at trends over time, it can be argued that it is the mean or average value that is the significant value in relation to system performance.

Simple and well recognised statistical procedures can be used to provide statistically unbiased estimates of the mean with a known degree of confidence. The degree of confidence (expressed as the confidence interval) will indicate how much the values are likely to vary from the mean between sampling events.

The disadvantage of this approach is that a few high values can be offset by a number of low values.

ASSESSING PERFORMANCE AGAINST A PERCENTILE

Using this approach, performance is satisfactory if a large percentage of results (although not necessarily all) are less than the guideline value. Like the use of a mean, this approach has a number of attractions:

For health-related characteristics, performance could not be regarded as satisfactory if the guideline values were exceeded more than rarely. This is consistent with using a high percentile such as a 95^{th} percentile.

It is possible, using statistical procedures, to estimate with a known degree of confidence how well the

results of sampling represent the quality of water at other times.

Using a percentile to assess performance against the guideline is consistent with the requirement that the upper control limit of the control chart be equal to or less than the guideline value. For example, for normally distributed data, if the 95th percentile is used, control limits can be placed at 1.64 times the standard deviation on either side of the mean. These control limits will then encompass about 90% of the data, and of the remaining 10%, about 5% will be above the upper control limit and 5% below the lower. This means that if the upper control limit is the same as, or less than, the guideline value, then 95% or more of the data should be below the guideline value.

More samples need to be analysed to assess performance against a percentile than are needed for a mean. This is reasonable for health-related characteristics, as exceeding the guideline may, in some cases, have significant health effects. More sampling provides a greater degree of protection.

The main disadvantage of this approach is that estimates of percentiles are inherently more uncertain than estimates of means.

If more detailed or involved data analysis techniques are to be considered the advice of a statistician should be sought.

REFERENCE

Ellis JC (1989). *Handbook on the Design and Interpretation of Monitoring Programmes*. Water Research Centre, Medmenham, United Kingdom, Technical Report NS29.

Statistics – Statistical principles

INFORMATION SHEET 3.3

This information sheet sets out some general statistical principles and considerations for designing and interpreting water quality monitoring programs. Ideally, expert statistical advice should be sought in devising and interpreting such a program. Further information and references can be found in the *Australian Guidelines for Monitoring and Reporting*, National Water Quality Management Strategy Paper No. 7 (ANZECC/ARMCANZ 2000).

SUMMARY STATISTICS

A fundamental task in many statistical analyses is to characterise the *location* and *variability* of a data set. This is usually described by the mean (μ) and standard deviation (σ). Most statistical packages can produce these values for a given data set. In addition, percentiles are simple to derive. None of these three statistics require an underlying assumption of normality and all can be derived using simple statistical tools and commonly available spreadsheet packages. However, there are a number of data transformations that are usually required before the statistics can be estimated, as follows.

OUTLIERS AND 'LESS THAN' VALUES

Two persistent problems cause difficulties in the use of the mean in assessing water quality data:

outliers – that is, numbers that appear to be extreme when compared with other data in the data set. These are not numbers generated by some malfunction of measuring equipment or transcription errors, which clearly ought to be discarded. They are numbers that seem anomalous, although there is no obvious explanation and they cannot be discarded on technical grounds; and

values that are recorded as less than the limit of detection.

As an example, consider the following set of data:

<0.5, <0.5, 1.2, 1.4, 1.45, 2.1, 21.3

The first problem is what to do about the less-than values. Should they be ignored, replaced by 0.25, replaced by 0, or should the < symbol be ignored? There is no clear answer except that it can be shown that using L/2, where L is the limit of detection, is effectively a worst-case method and not the even-handed approach it appears to be at first sight (Ellis 1989). If the values below the limit of detection are critical in determining how a supply performs against the guidelines, then steps should be taken to reduce the limit of detection. Statistical treatment of values below the detection limit is possible but is complex and not entirely satisfactory. In the absence of any alternative, however, it is recommended that detection limit values be replaced by L/2, as a conservative approach. The original data set should be kept intact so that it can be seen which data were substituted in this way, and when presenting results, the substitution should be noted. Further information on dealing with less than values can be found in Croghan and Egeghy (2003) and Smith *et al.* (2006).

For determining the 95th percentile, up to 95% of the reported results can be less than the limit of detection and the statistic can still be found readily. The lowest 95% of reported values simply identify which value is reported as 95th ranked value and do not arithmetically contribute to it. If more than 95% of the reported values are below the detection limit, the 95th percentile should be reported simply as less than the limit of detection. Percentiles are discussed further in the following section.

For determining averages, it is necessary to substitute the values at less than the detection limit with L/2 and to note that this substitution was made, as well as noting what proportion of data was below the detection limit; for example: Average: 0.3 mg/L (notes: detection limit 0.2 mg/L, 12 samples taken, 3 samples below detection limit which were substituted with a value of 0.1 mg/L). The substitution of censored data will necessarily introduce biases to the calculated means and standard deviations, and this approach should be used only when the proportion of censored data is relatively low (e.g. 3 out of 12 results, in the above example).

The second problem with the data set is the very high 21.3 value. Is it genuine, or an analytical error? If it is genuine, is it valid to include it in the calculation of the mean (and hence the 95th percentile) when it will clearly have a marked effect on the result? The answer is that it must be included in the calculation as it may have an impact on the health of people receiving the water. To remove it would have the same effect as censoring the data set. Only those data points that have been clearly shown to be in error should be removed.

Simple worksheet packages, such as Excel™, will have some difficulty in deriving percentiles and averages if some of the data are shown as $<x$, since the software may simply ignore these values and see them as non-numerical. Therefore, substitution with numerical values, such as L/2, or manual calculation, may be necessary, to avoid producing misleading results.

If the detection limit is above the guideline value, the assay should be changed to provide a more sensitive one. Detection limits are also discussed in Chapter 9 (Section 9.10.3).

SKEWNESS AND KURTOSIS

A further characterization of the data can include the measure of *skewness* and *kurtosis*.

Skewness is a measure of symmetry, or more precisely, the lack of symmetry. A distribution, or data set, is symmetrical if it looks the same to the left and right of the centre point.

The skewness for a *normal distribution* is zero, and any symmetrical data should have a skewness near zero. Negative values for the skewness indicate data that are skewed left (i.e. the left tail is long compared to the right tail); positive values indicate data that are skewed right (i.e. the right tail is long compared to the left tail). Further advice on how to treat strongly skewed data sets can be found in McBride (2005).

Kurtosis is a measure of whether the data are peaked or flat relative to a normal distribution. Data sets with high kurtosis tend to have a distinct peak near the mean, decline rather rapidly, and have heavy tails. Data sets with low kurtosis tend to have a flat top near the mean rather than a sharp peak. A uniform distribution would be the extreme case.

There are different definitions used for kurtosis. The standard *normal distribution* has a kurtosis of zero. Which definition of kurtosis is used is a matter of convention. When using software to compute the sample kurtosis, it is necessary to be aware of which convention is being followed.

The *histogram* (Information Sheet 3.1) is an effective graphical technique for showing both the skewness and kurtosis of a data set. This may be sufficient to indicate whether the data are approximately normal, or are skewed and need further statistical treatment (e.g. transformation) before statistics are calculated and performance assessments undertaken.

MEASUREMENT ERROR

A set of results is no more than a series of snapshots of some process over the period of sampling. A statistic calculated from these results, such as a percentile, a mean, or a standard deviation, can never exactly coincide with the true statistic, except by chance. The true statistic could only be determined by continuous error-free measurement of every drop of water – an impossibility in water quality analysis.

Values determined experimentally from a set of measurements are, thus, often referred to as estimates of the true statistic. These estimates may be too high or too low – there is no way of knowing. This uncertainty is known as the measurement error (although the term 'error' is unfortunate as it really means 'small departures from the true result', not mistakes made in analysis), and quantification of this error is an important component of statistical methods.

If more detailed or involved data analysis techniques are to be considered, the advice of a statistician should be sought.

REFERENCES

Croghan CW, Egeghy PP (2003). Methods of dealing with values below the limit of detection using SAS. South Eastern SAS Users Group Conference. http://analytics.ncsu.edu/sesug/2003/SD08-Croghan.pdf

Ellis JC (1989). *Handbook on the Design and Interpretation of Monitoring Programmes.* Water Research Centre, Medmenham, United Kingdom, Technical Report NS29.

McBride GB (2005). *Using Statistical Methods for Water Quality Management. Issues, problems and solutions.* Wiley Series in Statistics in Practice.

Smith D, Silver E, Harnly M (2006). Environmental samples below the limits of detection – comparing regression methods to predict environmental concentrations. SAS Conference Proceedings: Western Users of SAS Software. http://www.lexjansen.com/wuss/2006/Analytics/ANL-Smith.pdf .

Sokal RR, Rohlf FJ (1969). *Biometry*. WH Freeman and Company, San Francisco.

Statistics – Control charts and trends

INFORMATION SHEET 3.4

PURPOSE AND CONTENTS OF A CONTROL CHART

A control chart displays monitoring data for a given characteristic against either time or sample sequence number. It has the following important features clearly marked:

control limits;

each measured data point;

the mean value of the measurements.

PURPOSE OF CONTROL LIMITS

Control limits can be based on long-term monitoring data (including data from the reporting period). They are horizontal lines parallel to the mean but shifted from it by a number of standard deviations (at least 1.64 times the standard deviation), and they are calculated from the long-term standard deviation. They thus define the area within which most of the long-term data fall. Provided that the system is 'in control', most of the data for the reporting period will also lie between these limits. In addition, an alert limit can also be set, which is more stringent than the control limit. The alert limit is the point at which corrective action is initiated to avoid breaching a control limit.

Alternatively, the control limits can be set at some pre-determined target criteria or critical limits (see Sections 9.4.2 and 9.4.3). The target criteria reflect the effectiveness of a process. A critical limit is a prescribed tolerance that distinguishes acceptable from unacceptable performance.

Control charts can also be used to assess long-term performance on an ongoing basis (rather than for a given reporting period), in which case the control limits and mean should be calculated from all the available data over previous years, and recalculated periodically. They should be used in relation to operational monitoring data, but are of no use when assessing drinking water quality monitoring data.

ADVANTAGES OF USING CONTROL CHARTS TO ASSESS PERFORMANCE

When using control charts to assess long-term operational performance:

It is easy to see if data exceed an alert limit (target criteria) or control limit (critical limit), and by what amount.

The variability in the data can be quickly determined. Characteristics with low variability may be of less concern than those that vary markedly. Trends or 'runs' of consecutive high or low values in the data may also be observable.

SETTING CONTROL LIMITS

A decision must be made on where to place the control limits; that is, on the percentage of the long-term measured data that they will contain (see Table IS3.4.1). It is suggested that the control limits should be not less than 1.64 times the long-term standard deviation; for normally distributed data, this will encompass approximately 90% of the long-term data and, provided the system remains in control, approximately 90% of the data for the reporting period. The distances for other percentages of the data are shown below. These figures are constants for any normal distribution curve, and can be determined from cumulative normal probability tables given in most statistical textbooks.

Alternatively, the control or alert limits can be set based on an operational target or limit that must be met, for example, the need to maintain a fluoride concentration below 1.0 mg/L. In this example, the upper control limit may be 0.8 mg/L and the alert limit 1.0 mg/L. By plotting the data on a control chart, emerging trends in fluoride concentrations can be identified, and preferably managed before either the control limit (critical limit) or alert limit (target criteria) is reached.

Table IS3.4.1 Relationship between control limits and multiples of the standard deviation[4]

Standard deviations(s)	% of data expected to fall within the bounds
1.64 x s	90.00
1.96 x s	95.00
3.00 x s	99.85

DETERMINING THE STANDARD DEVIATION

In order to establish control limits, it is necessary to determine a reliable mean and long-term standard deviation. To obtain initial estimates of these statistics, no less than 7 and preferably 15 or more measurements are required from independent representative samples. Therefore, a monthly sampling program would collect sufficient data to allow for meaningful control limits to be set after a 12-month period. It is clearly unsatisfactory to bias the results by selecting sampling times or locations that are favourable (or unfavourable).

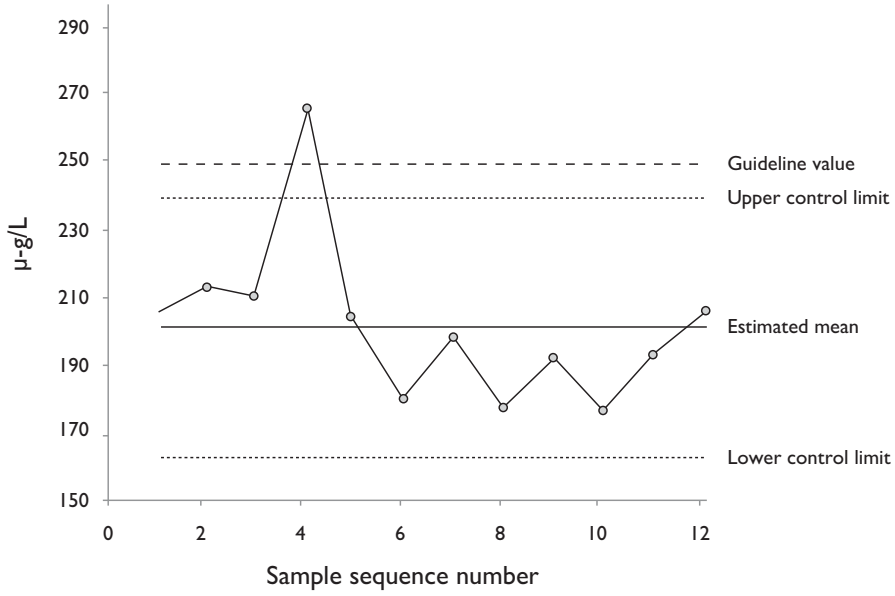
To ensure the ongoing relevance of the control limits that are set, the mean and standard deviation should be periodically recalculated based on either a longer data set, or another representative period of collected data.

EXAMPLE OF A CONTROL CHART

Figure IS3.4.1 shows an example of a control chart using trihalomethanes data, using 12 monthly measurements. In the example, the control limits have been placed at two standard deviations away from the mean. The guideline value would act as the critical limit. Excursions above either the control limit or the critical limit would prompt an investigation. The control chart shows that, except for the one excursion above the critical limit, trihalomethanes levels were generally under control during the period under review.

4 Taylor JK (1987). *Quality assurance of chemical measurements*, Lewis Publishers, Chelsea, Michigan.

Figure IS3.4.1 *Example of a control chart for trihalomethanes data*



REFERENCES

APHA (American Public Health Association) (2005). Method 1010B General Introduction: Statistics. In: *Standard Methods for the Examination of Water and Wastewater*, 21st Edition. APHA, Washington, United States.

Taylor JK (1987). *Quality Assurance of Chemical Measurements*. Lewis Publishers, Chelsea, Michigan.

ANZECC, ARMCANZ (Australia and New Zealand Environment and Conservation Council, Agriculture and Resource Management Council of Australia and New Zealand) (2000). *Australian Guidelines for Monitoring and Reporting*. National Water Quality Management Strategy Paper no. 7. ANZECC, ARMCANZ.

Number of samples required

INFORMATION SHEET 3.5

NON-MICROBIAL

Poor quality water supplies should be more frequently monitored than good quality water supplies; this is supported by statistical arguments as shown below.

If the data are normally distributed, the minimum number of samples required to achieve a desired level of precision with a known degree of confidence can be determined using the following formula:

$$n = \left\{\frac{t(a) \times h \times s}{D}\right\}^2$$

Where:

$t(a)$	=	Student's t statistic with infinite degrees of freedom corresponding to a single tail probability of a. At the 95% confidence level this value is 1.96
h	=	an uncertainty factor in estimating percentiles: for the 95th percentile the value is 1.64 (at the 95% confidence level): for means the value is 1.0
s	=	standard deviation
D	=	precision in measurement
n	=	number of samples required.

Most water quality data are skewed. Where the data are skewed, it is still possible to calculate the number of samples required but the calculation is more complex (Ellis 1989). Boxes IS3.5.1 to IS 3.5.3 detail this more complex method.

***Box IS3.5.1** Samples required to meet a guideline based on a 95th percentile*

Suppose that in the past a characteristic has been running with a mean of 0.02 mg/L with a standard deviation of 0.02 mg/L, and that for this characteristic the guideline value is 0.1 mg/L. The 95th percentile can be estimated as follows:

95^{th} percentile = mean + 1.64 x s = 0.02 + 1.64 x 0.02 = 0.0528

This is well below the guideline value. It would be possible to take fewer samples and still be confident that the guideline has been met.

To estimate the minimum number of samples necessary, the first step is to calculate the necessary precision by halving the difference between the 95^{th} percentile and the guideline value:

(0.1 - 0.0528)/2 = 0.0236 mg/L

The lower limit of the confidence interval is the estimated 95^{th} percentile, and the upper limit is the guideline value. The number of samples required to achieve this can then be calculated as follows:

$$1.96\left\{\frac{\times 1.64 \times 0.02}{0.0236}\right\}^2 = 8 \text{ samples with rounding up}$$

Thus, a precision of 0.0236 mg/L can be achieved (with 95% confidence) by taking 8 samples over the year. Alternatively, 8 samples per year will be sufficient to be sure (with 95% confidence), that the 95^{th} percentile is less than the guideline value.

***Box IS3.5.2** Samples required to meet guidelines based on 95th percentile, with a different mean*

Suppose that after taking these 8 samples it is found that the mean has drifted up to 0.04 mg/L, but the standard deviation remains the same at 0.02 mg/L. The 95th percentile is now:

$$95^{th} \text{ percentile} = \text{mean} + 1.64 \times s = 0.04 + 1.64 \times 0.02 = 0.0728$$

The precision now required is 0.014 mg/L (as (0.1–0.072/2)=0.014 mg/L). This is a smaller value and hence the number of samples required to achieve it with the same degree of confidence will increase:

$$\left(\frac{1.96 \ 1.64 \ 0.02}{0.014}\right)^2 = 22 \text{ samples (with rounding)}$$

Therefore, the sampling frequency would have to be increased to 22 per year, or about 1 per fortnight, to meet this change in precision.

***Box IS3.5.3** Number of samples based on meeting a mean*

Using the same data given in Example 2 above, the precision required can be calculated by halving the difference between the mean and the guideline value, i.e. (0.100– 0.040)/2 = 0.03 mg/L (the lower limit of the confidence interval in this example is the mean, and the upper limit is the guideline value). The number of samples required is then:

$$\left(\frac{1.96 \ 0.02}{0.03}\right)^2 = 2 \text{ samples (with rounding)}$$

Thus, 2 samples per year would be sufficient to be sure (with 95% confidence) that the mean is less than the guideline value. Using a mean instead of a 95th percentile can make a substantial difference to the number of samples required.

MICROBIAL

One of the aims in any sampling program, particularly microbiological sampling, is to have a high degree of confidence that the water quality as measured in the laboratory is representative of that actually used by the consumer, not just at the time of sampling, but all the time. Unless all water is sampled, it is not possible to be 100% confident that this condition is met. A properly designed sampling program, testing only a very small percentage of the total amount of water in a system, can give a high degree of confidence about the overall water quality. The degree of confidence is related to the number of samples analysed. (This assumes, of course, that the sampling locations selected are representative of the water supplied to the consumer.)

Even if all samples tested are free of bacterial indicators, no sampling program can guarantee that *all* the water in a system is free of indicator organisms. In fact, it can be shown that for any reasonable sampling program, the degree of confidence in achieving a situation where 100% of the water in a system is free of bacterial contamination is close to zero (Ellis 1989).

It is far better to have a high degree of confidence that a large proportion of the water is free of contamination, than to have no confidence that all the water is uncontaminated. Realistic monitoring programs can give a high degree of confidence that 98% of all the water in a system is fee of bacterial contamination.

This does *not* mean that the other 2% of water is contaminated. All it indicates is that the sampling program is statistically unable to show a high degree of confidence that more than 98% of all the water in the system is free of contamination.

Even if all samples tested are uncontaminated, it does not follow that there is necessarily a high degree of confidence that the water is free from contamination. The number of samples required to meet a target, and the degree of confidence that this confers when all samples are free of contamination, is shown in Figure IS3.5.1 (Ellis 1989).

For example, if 50 samples are tested per year and all are free of contamination, then there is only 65% confidence that 98% of the water in the system is free of contamination. It would be necessary to take 150 samples, each free of contamination, before the degree of confidence reached 95%. Fewer than 50 samples per year, even if each sample was free of contamination, give a low degree of confidence that the water system as a whole is 98% free of contamination.

If one or more samples taken over a year are positive, then the degree of confidence that 98% of water in the system is free of contamination is reduced. This is shown in Figure IS3.3.2 (Ellis 1989). Suppose, for example, that 150 samples were collected in a year but some of those samples showed faecal contamination. The degree of confidence that 98% of the water in the system is free of contamination drops from 95% with a positive result to 80% with one positive result, and 60% with two positive results.

The plateau shown in Figure 2 at the 50% confidence level is an artefact of the difficult computation procedure used to derive these graphs. The graphs should only be regarded as an approximate guide, but they nevertheless provide a highly informative summary.

Figure IS3.5.1 *Level of confidence that 98% of water in a supply is free of faecal contamination for different numbers of samples when all samples tested are free of faecal contamination (Source: Ellis 1989, reprinted with permission of the Water Research Centre, Medmenham)*

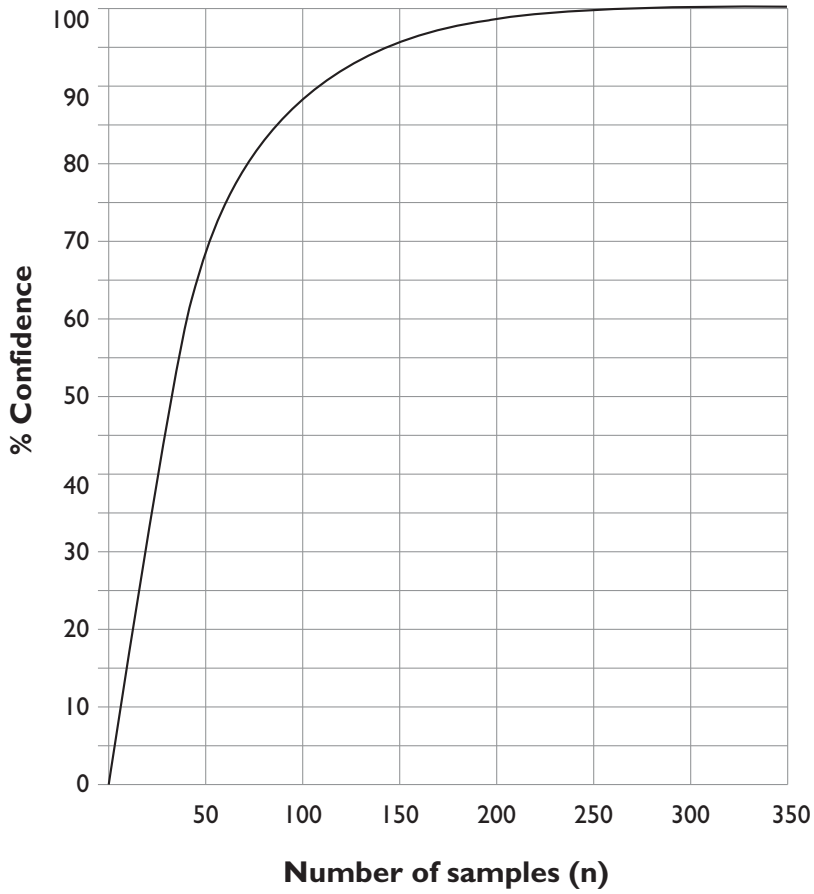
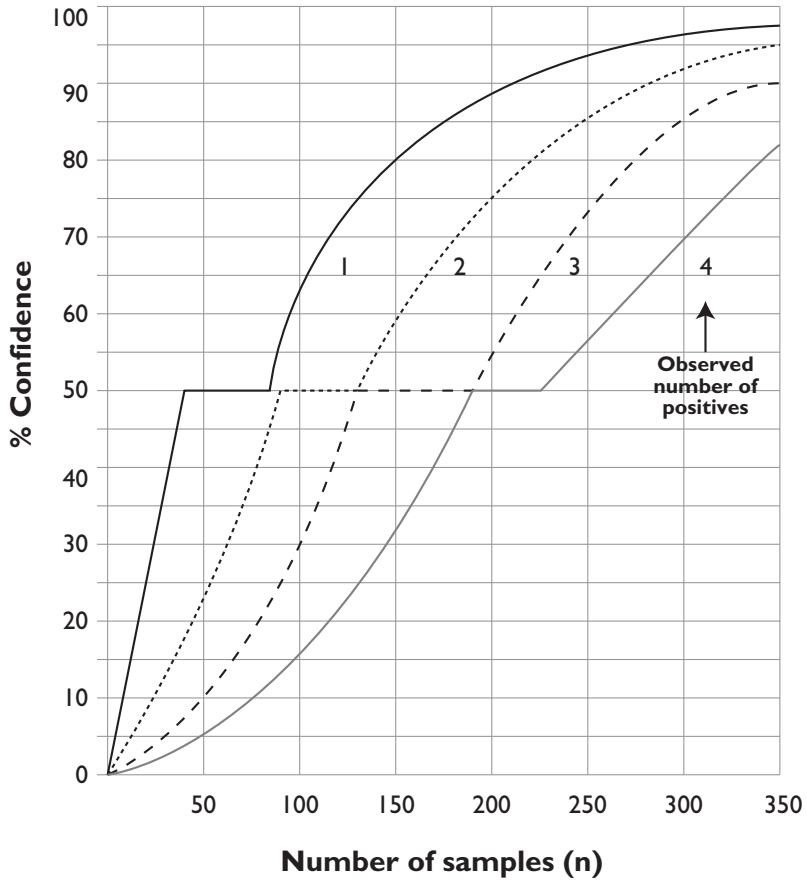


Figure IS3.5.2 *Level of confidence that 98% of water in a supply is free of faecal contamination for different numbers of samples when 1, 2, 3 or 4 samples give positive results (Source: Ellis 1989, reprinted with permission of the Water Research Centre, Medmenham)*



REFERENCE

Ellis JC (1989). *Handbook on the Design and Interpretation of Monitoring Programmes*. Water Research Centre, Medmenham, UK, Report NS No 29.

Guidance for issuing and lifting boil water advisories

This guidance will help water suppliers, in conjunction with public health authorities, develop procedures for boil water advisories.

Boil water advisories are public announcements advising that drinking water should be boiled (or otherwise disinfected) before being consumed or used to wash uncooked food (e.g. salad vegetables and fruit), make ice, gargle or clean teeth. They are generally issued on the basis of suspected or confirmed contamination by potentially pathogenic micro-organisms.

Boil water advisories are serious measures and although they are intended to protect public health they can also have adverse consequences including risks of scalding (Mayon-White and Frankenberg 1989) and non-compliance particularly when issued frequently or for extended periods of time (consumer fatigue) (Hrudey and Hrudey 2004, WHO 2011). The decision to issue a boil water advisory should follow the same risk management principles applied in ensuring safe drinking water. A boil water advisory should be used when consideration of all available information leads to the conclusion that the risk to public health is persistent, unacceptably high and is greater than the adverse consequences of an advisory.

Although the decision to issue a boil water advisory needs careful consideration timeliness is also important with delays potentially leading to increased disease (Powell et al. 2002, O'Connor 2002). Preparation is essential to prevent unnecessary delays. Drinking water suppliers and public health agencies should have contingency plans dealing with the issuing and lifting of boil water advisories. Contingency plans should be included in incident and emergency protocols (see Chapter 3.6). Plans should include reporting criteria and procedures for ensuring ongoing communication between water suppliers, public health agencies, and the public during events.

In order to be adequately prepared for the implementation of a boil water advisory, it may be useful for water suppliers and health authorities to run mock boil water exercises.

POTENTIAL CAUSES

A number of factors may lead to consideration of a boil water advisory including environmental emergencies, failure of critical control points and other preventive measures, adverse results from monitoring, detection of pathogenic micro-organisms or detection of drinking water-borne disease (confirmed or suspected).

Environmental emergencies/events

Environmental events such as severe storms, flooding, bushfires or earthquakes can lead to:

inundation or failure of water treatment plants

inundation and contamination of distribution systems

destruction/damage of infrastructure

deterioration in physical or microbiological quality of source waters that overwhelms treatment capability

loss of power, which results in treatment processes failing to work.

Matters to consider include the level and duration of impact.

Failure of critical control points/preventive measures

Issues that may lead to the failure of critical control points and other preventive measures include:

impaired or inadequate filtration due to mechanical breakdown or operational failure

impaired or inadequate disinfection

failure to protect distribution system integrity from ingress of contamination (e.g. through mains breaks or unintended cross-connections).

In most cases failure of critical control points/preventive measures should be detected by well-designed operational monitoring programs. For example:

impaired or inadequate filtration should be detected by failure to comply with operational criteria and critical limits for turbidity

impaired or inadequate disinfection should be detected by failure to comply with operational criteria and critical limits for disinfectant residuals, C.t or UV light transmission

failure to protect distribution system integrity may be detected by unexpected loss of chlorine residual, loss of pressure, changes in turbidity and other physical characteristics or increased customer complaints.

The duration of faults, the extent of non-compliance, performance of other relevant preventive measures, availability of buffering water storages and available data and information on source water quality should be considered when assessing the impacts of poor performance or failure on safety of drinking water supplies. For example:

minor deviations in filtration performance may be compensated for by downstream disinfection. However, where filtration is being used as the primary control against *Cryptosporidium*, downstream disinfection with chlorine or chloramines will not be effective

impacts of interruptions to chlorination may be reduced by buffering in downstream treated water storages.

Detection of E. coli and pathogens

Adverse results from monitoring could lead to consideration of a boil water advisory. For example:

repeated or persistent detection of *E. coli* in distribution systems

the detection of enteric pathogens (e.g. *Cryptosporidium*) in samples collected for investigative purposes.

As described in Chapter 10 occasional detections of E. coli may occur and in the absence of evidence of any other failures (e.g. inadequate disinfection), should not trigger a boil water advisory. The initial response should be to urgently identify and rectify any sources of contamination and immediately collect further samples. If repeat samples contain E.coli the response should be escalated, and could include increased or supplementary disinfection as well as more widespread monitoring to determine the extent of contamination. If investigations indicate that a systematic failure exists a boil water advisory should be considered.

Similarly, detection of low numbers of *Cryptosporidium* oocysts in a single sample of drinking water in the absence of evidence of any other failures/incidents (e.g. increased or new challenges in source water, major rainfall events, impaired filtration) would not normally trigger a boil water advisory, but should lead to further investigations and immediate re-sampling. Potential viability and infectivity of detected oocysts is important information to enhance risk assessment. If speciation is not available on the original sample this should be considered for follow-up samples.

The repeated detection of enteric pathogens should lead to consideration of a boil water advisory.

Detection of waterborne disease

Detection of confirmed disease associated with drinking water from a community water supply means that a major fault has occurred. A boil water advisory should be issued, unless there is a high level of certainty that the fault has been rectified and all contaminated water has been flushed/removed from the distribution system.

In the case of disease suspected to be associated with drinking water the decision to issue a boil water advisory will depend on a number of factors including the strength of evidence that drinking water is the

cause, when and where the disease occurred and information about the water supply, including changes in operation/faults/rectification etc.

Guidance in a boil water advisory

Bringing water to a rolling boil[5] is sufficient to inactivate enteric micro-organisms (see Attachment C). The advisory should indicate that the affected drinking water can be made microbiologically safe by bringing the water to a rolling boil. This can be achieved by a number of methods, although care should be taken to avoid scalding. Kettles with automatic shut off switches are sufficient for this purpose and should reduce the risk of scalding, although their use relies on a power supply being available. Variable temperature kettles should be set to boil.

Water should be boiled before being used for drinking, mixing of cold beverages, washing of uncooked food (e.g. salad vegetables and fruit), making ice, brushing teeth and gargling. Water must be cooled before use.

Under most circumstances it is not necessary to boil water used for other household purposes., As a guide, if water complies with the NHMRC *Guidelines for Managing Risks in Recreational Water* highest two categories for primary contact (<200 enterococci or E. coli per 100 mL) it should be safe for showering and bathing for all except the very young, who can swallow more bath or shower water than older children and adults. As a precaution toddlers and infants should be sponge bathed.

Washing of dishes by machine or hand is acceptable, provided dishes are air-dried. No additional precautions are required for washing clothes.

When it is not possible to boil water

If it is not possible to boil water, for example if there is no electricity, commercial products are widely available for point-of-use disinfection. These include “chlorine tablets”, which have been widely used in disaster relief situations and also by travellers in areas without good water sanitation.

Alternatively, unscented household bleach (containing sodium hypochlorite) can be used. This will be effective against bacteria, viruses and some protozoa (but not *Cryptosporidium*). For clear water add one teaspoon (about 5 mL) of household bleach containing 4-5% available chlorine per 30 litres of water (or two drops bleach per litre of water). For cloudy water add one teaspoon of bleach per 15 litres (or four drops bleach per litre of water). Further information on how to use sodium hypochlorite and other methods of disinfection for water is provided in Table 6.1 of the World Health Organization’s Guidelines for Drinking Water Quality (2011).

LIFTING A BOIL WATER ADVISORY

Criteria for lifting a boil water advisory will require a risk assessment based on the cause of the advisory. It is difficult to provide prescriptive guidance for all events and circumstances. In general terms, lifting an advisory requires evidence that the identified environmental or operational causes of contamination that led to the boil water advisory being issued have been resolved/rectified and that contaminated water has been cleared from the water supply.

5 A rolling boil is defined as a continuous and rapid stream of air-bubbles rising from the bottom of a pot or kettle.

There must be evidence that contaminated water has been cleared from the distribution system by flushing and/or disinfection. This should include results for disinfectant residuals and *E. coli* from affected zones of distribution systems. Sample locations should be chosen to ensure adequate coverage of the affected area and not necessarily be limited to collection of samples from routine locations. It may be possible to lift boil water advisories on a single set of *E. coli* results providing there is sufficient supporting data to justify such a decision. For example, data demonstrating persistent chlorine residuals being present in affected zones. However, in cases of major contamination, the advisory should not be lifted until two sets of samples, collected on separate days, have returned negative results

Where a boil water advisory has been issued because of detection of an enteric pathogen (e.g. *Cryptosporidium*) it will generally be necessary to demonstrate that a treatment barrier (e.g. filtration) has been restored and that the organism is no longer present. This will require absence of the organism from, ideally, consecutive sets of samples and a review of barrier performance.

COMMUNICATION

Appropriate communication with water consumers and users is key to ensuring a boil water advisory is effective and consumers are protected. Communication procedures should be prepared in advance of any incident or emergency and should include:

who is responsible for issuing the boil water advisory

how it should be implemented

templates for communicating information on boil water advisories and

establishing communication networks with major water users.

Boil water advisories should include the reason for the advisory, recommended actions to be taken by consumers, potential health consequences of disregarding advice, and action being taken by the water utility and the health agency. A contact phone number for enquiries should be included. Evidence has shown that providing clear explanatory information from the outset improves compliance with advisories (Angulo et al. 1997, O'Donnell et al. 2000). A generic template for communicating with consumers is provided in Attachment A.

Large water users and specialist users should be contacted directly (see section on "How should the boil water advisory be issued?").

Who should issue the boil water advisory?

Responsibility for issuing a boil water advisory will vary and may rest with the water utility, the relevant health authority or a separate regulatory agency. Who is responsible needs to be clearly identified in pre-established incident protocols. A boil water advisory should only be issued after consultation between the drinking water supplier and the relevant health authority.

When should the boil water advisory be issued?

When required, boil water advisories should be issued as soon as possible after detection of evidence of a serious fault. Delays will result in prolonged exposure of consumers to potentially unacceptable levels of risk and undermine the confidence of consumers.

How should the boil water advisory be issued?

Generally, boil water advisories will be issued through a broad range of media outlets including print, radio and television. These should include media outlets directed toward non-English speaking communities. Where available, electronic/social media and communication tools should also be used. Consideration should be given to the development of graphics for low literacy consumers (see for example http://www.health.qld.gov.au/disaster/documents/safe-water-poster.pdf), and to providing translations for culturally and linguistically diverse communities.

Large water use customers (e.g. food manufacturers), vulnerable users (e.g. hospitals, residential care facilities), specialist users (e.g. medical practitioners and dentists) and agencies and organizations that provide support services for those with limited vision or hearing, should be notified directly. Consideration should be given to notifying water carters (who may have carried contaminated water outside the affected area).

While it is expected that large water use customers will have developed their own response plans for water contamination incidents, water suppliers should establish and maintain contact lists for these users, and ensure they are informed of the advisory so they can implement these management plans.

For smaller community-based supplies, other mechanisms of communication with consumers including posting signs, door knocking, door hangers or letter box drops should be used where practical in addition to issuing advice through traditional media outlets.

Consideration should also be given as to how to deal with publicly-accessible water supply points, such as drinking fountains/bubblers. This may be managed through appropriate signage.

Hotels, motels and other accommodation businesses should be reminded to provide the boil water advice to all customers.

If large areas are affected or if the advisory is likely to be in place for many days, issuing specific advice to specific users may be considered (e.g. hotels, food businesses, GPs, dentists, schools, swimming pool operators).

Regular reminders should be issued throughout the incident.

How should a boil water advisory be lifted?

A similar approach to that taken in issuing a boil water advisory should be applied when lifting it. A decision to lift a boil water advisory should only be taken after agreement between the water supplier and the relevant public health agency.

The actions taken to ensure drinking water safety and to minimise the likelihood of recurrence of the incident should be provided in notices lifting the advisory. This is important to underpin public confidence in the drinking water supply and the overall management of water quality.

Guidance should be provided to consumers about measures they are required to undertake to clear potentially contaminated water from private plumbing systems including residential properties, for example flushing systems to ensure contaminated water is removed. In buildings used by vulnerable populations (e.g. hospitals) testing for *E. coli* and/or chlorine residuals could be required to verify that contaminated water has been removed.

A generic template is provided in Attachment B.

After the event

As described in Chapter 3.6.2, following the lifting of a boil water advisory, a full investigation, debrief and review of the event/incident should occur. All staff, functional groups and agencies involved in the event/incident should be included in this process.

In addition to identifying operational, technical or environmental causes and responses, the review should include an assessment of the timeliness and effectiveness of communications, and the level of compliance with the advisory. In the case of large scale events an external review could be useful.

The review should identify what worked well, what failed, what needs to be changed and whether additional resources were required.

REFERENCES

Angulo FJ, Tippen S, Sharp DJ, Payne BJ, Collier C, Hill J, Barrett TJ, Clark RM, Geldreich EE, Donnell HD and Swerdlow DL (1997) A community waterborne outbreak of salmonellosis and the effectiveness of a boil water order. American Journal of Public Health 87, 580-584.

Hrudey SE and Hrudey EJ (2004) Safe Drinking Water: Lessons from recent outbreaks in affluent nations. IWA publishing.

Mayon-White RT and Frankenberg RA (1989) Boil the water. Lancet 334, 216.

National Health and Medical Research Council (2008). Guidelines for Managing Risks in Recreational Water. Australian Government, Canberra

O'Connor DR (2002) Report of the Walkerton Inquiry, Part 1. The events of May 2000 and related issues. The Attorney General of Ontario, Toronto, The Walkerton Inquiry.

O'Donnell M, Platt C and Aston R (2000) Effect of a boil water notice on behaviour in the management of a water contamination incident. Communicable Disease and Public Health 3, 56-59

Powell DA, Blaine KA, Gomes L, Grant SE, LaCroix and Morris S (2002) Water warnings: communication in drinking water-related public health emergenciesThe Walkerton Inquiry. Commissioned Paper 12.

World Health Organization (2011) Guidelines for Drinking Water Quality.

Attachment A

GENERIC BOIL WATER ADVISORY TEMPLATE

This template can be used as the basis for informing consumers that a boil water advisory has been issued. The water supplier or public health agency issuing the advisory can add or delete information as appropriate.

Boil water {alert / advisory} for (name area/water supply)

The (*Water Supplier or Public Health Agency*) advises that consumers in (*identify affected area, list towns/suburbs*) should boil drinking water until further notice. A map of the affected area is available at (*identify web-site – a map should also be attached to the advisory*)

This advice has been issued following (*state reason for the advice, including when the fault was detected*).

Customers should bring water to a boil by heating the water until a continuous and rapid stream of air-bubbles is produced from the bottom of a pan or kettle. Kettles with automatic cut-off switches are suitable. Variable temperature kettles should be set to boil. After heating, water must be allowed to cool before using it, and be stored in a clean, closed container for later use. Care should be taken to avoid scalding {injuries}.

Customers should boil all water used for:

drinking

brushing teeth

washing and preparing food or beverages

preparing baby formula

making ice.

Unboiled water can be used for:

showering and bathing (avoid swallowing water). As a precaution babies and toddlers should be sponge bathed to prevent them swallowing water

washing dishes by hand or in a dishwasher, providing dishes are air-dried before being used after washing

washing clothes.

Consumption of unboiled water could lead to (*provide list of symptoms*). If you are concerned that you may have been affected by contaminated water please contact your GP and advise them about this notice. There have been (*state number*) illnesses reported to date (*if any identified*)The water supplier is working closely with the public health agency to identify conditions that will enable the boil water advice to be lifted. To correct the problem the (*water supplier*) is (*state what is being done and why*). It is expected that this will take (*if possible give estimated times to resolve problem*). The advisory will be in effect until the (*water supplier*) and the (*public health agency*) are confident that there is no longer a public health concern.

Please share this advice with neighbours and friends in the affected area.

For more information go to (*identify web-site*) or call (*phone number of water utility, public health agency or dedicated hot-line if established*). Regular updates will be provided.

Attachment B

GENERIC TEMPLATE FOR LIFTING A BOIL WATER ADVISORY

This template can be used as the basis for informing consumers that a boil water advisory has been lifted. The water supplier or public health agency responsible for the advisory can add or delete information as appropriate.

Boil water advisory for (name area/water supply) has been lifted

The (*Water Supplier or Public Health Agency*) advises that the boil water advisory issued on (*identify date*) has been lifted and there are no restrictions remaining on the normal uses of drinking water supplied to (*identify affected area, list towns/suburbs*). This action has been taken following consultation with (*Public health agency*) **or** based on advice provided by the (*water supplier*).

The boil water advice was issued following (*state reason*). It has been lifted after (*state remedial action*) has restored water safety. This has been confirmed by tests results showing (*state results*).

Home owners and residents are advised that internal taps should be run for 2-3 minutes to ensure that any contaminated water is flushed from their plumbing. Owners and managers of large buildings should ensure that their entire system is flushed and that storage tanks are drained and refilled. Building managers and owners can contact (*water supplier's phone number*) if advice is required.

The (*public health agency*) has advised that there have been (include number) illnesses reported (*if any identified*).

The (*water supplier*) is working closely with the (*public health agency*) to investigate the causes of the contamination/incident and to identify procedures to prevent recurrence.

For more information go to (*identify web-site*) or call (*phone number of water utility, public health agency or dedicated hot-line if established*).

Attachment C

INACTIVATION OF MICRO-ORGANISMS AT ELEVATED TEMPERATURES

Organism	Temp (°C)	Inactivation time (secs)	Reference
Bacteria			
Campylobacter spp	60	16.2 (3.9 log10)	D'Aoust et al. (1988)
	63	16.2 (> 5log10)	D'Aoust et al. (1988)
	62	15 (3.5-5log10)	FSANZ 2007
Coxiella burnettii	79.4	25 (>5log10)	FSANZ 2007
E. coli	60	16.2 (1.5 log10)	D'Aoust et al. (1988)
	65	1800 (6 log10)	Moce-Llivina et al. (2003)
		< 2 (per log10)	Spinks et al. (2006)
E. coli O157	64.5	16.2 (> 5log10)	D'Aoust et al. (1988)
	65	3 (per log10)	Spinks et al. (2006)
	62	15 (<1 – 5log10)	FSANZ 2007
Enterococcus faecalis	65	7-19 (per log10)	Spinks et al. (2006)
Klebseilla pneumoniae	65	< 2 (per log10)	Spinks et al. (2006)
L.pneumophila	65	9 (per log10)	Dennis et al. (1984)
Legionella spp	80	18-42 (per log10)	Stout et al. (1986)
Mycobacterium paratuberculosis	72	15 (>4 log10)	FSANZ 2007
Pseudomonas aeruginosa	65	5 (per log10)	Spinks et al. (2006)
Salmonella spp (mixed)	68.3	16.2 (> 5log10)	D'Aoust et al. (1987)
Salmonella Typhimurium	65	< 2 (per log10)	Spinks et al. (2006)
Salmonella Cholerasuis	60	300 (per log10)	Moce-Llivina et al. (2003)
Serratia marcesans	65	< 2 (per log10)	Spinks et al. (2006)
Shigella sonnei	65	3 (per log10)	Spinks et al. (2006)
Yersinia enterocolitica	63	16.2 (> 5log10)	D'Aoust et al. (1988)
Viruses			
Adenovirus 5	70	1260 (>8 log10)	Maheshwari et al. (2004)
Coxsackievirus B4	60	1800 (5.1 log10)	Moce-Llivina et al. (2003)
Coxsackievirus B5	60	1800 (4.8 log10)	Moce-Llivina et al. (2003)
Echovirus 6	60	1800 (4.3 log10)	Moce-Llivina et al. (2003)
Enteroviruses	60	1800 (4.3 log10)	Moce-Llivina et al. (2003)
Hepatitis A	63	1800 (>6 log10)	Millard et al. (1987)
	65	120 (2 log10)	Parry & Mortimer (1984)
		1320 (3 log10)	Bidawid et al. (2000)
	75	30 (>5 log10)	Parry & Mortimer (1984)
		30 (>6 log10)	Millard et al. (1987)
	80	5 (5log10)	Parry & Mortimer (1984)
	85	<30 (>5 log10)	Bidawid et al. (2000)
		5 (>6 log10)	Millard et al. (1987)
Poliovirus 1	60	1800 (5.4 log10)	Moce-Llivina et al. (2003)
	62	1800 (>5 log10)	Strazynski et al. (2002)
	72	30 (>5 log10)	Strazynski et al. (2002)
	95	15 (>5 log10)	Strazynski et al. (2002)
Protozoa			
Cryptosporidium parvum	60	300 (3.4 log10)	Fayer. R. (1994)
	72	60 (3.7 log10)	Fayer. R. (1994)
		5-15 (>3 log10)	Harp et al. (1996)

Giardia	55	300	Jarrol et al. (1984)
	70	600 (100%)	Ongerth et al. (1989)

The results show that bacteria are particularly sensitive to heat and rapid kills (less than 1 minute per log) are achieved at temperatures above 65 °C. Viruses are inactivated at temperatures between 60 and 65 °C but more slowly than bacteria. However, as shown for poliovirus and hepatitis A that as temperatures increase above 70 °C greater than 5 log inactivations are achieved in less than a minute.

Cryptosporidium parvum is inactivated in less than 1 minute once temperatures exceed 70 °C. Data is more limited for *Giardia* but it is generally more sensitive to environmental pressure than *Cryptosporidium* (Sattar et al. 1999) and it is likely that it would at least be as sensitive to thermal inactivation as *Cryptosporidium*.

Based on these results it is considered that the process of heating water to a rolling boil and then cooling it to room temperature or below would provide more than enough time to inactivate pathogenic bacteria, viruses and protozoa. This approach is endorsed by the World Health Organization (WHO 2011).

REFERENCES

Bidawid S, Farber J, Sattar S, Hayward S (2000) Heat inactivation of Hepatitis A virus in dairy foods. Journal of Food Protection. 63(4):522-528.

D'Aoust J-Y, Emmons DB, McKellar R, Timbers GE, Todd ECD, Sewell AM and Warburton DW. (1987). Thermal inactivation of *Salmonella* species in fluid milk. J. Food Prot. 50, 494-501.

D'Aoust J, Park C, Szabo R, Todd E. (1988) Thermal inactivation of *Campylobacter* species, *Yersinia enterocolitica*, and haemorrhagic *Escherichia coli*. J Dairy Science 71:3230-3236.

Dennis PJ, Green D and Jones BP (1984) A note on the temperature tolerance of Legionella. J.Applied Bacteriol 56, 349350.

Fayer. R. (1994) Effect of High temperature on infectivity of *Cryptosporidium parvum* oocysts in water. Applied and Environmental Microbiology 60:2732-2735.

Food Standards Australia New Zealand (FSANZ, 2007) Scientific evaluation of pasteurisation for pathogen reduction in milk and milk products.

Harp J, Fayer R, Pesch B and Jackson G. (1996) Effect of pasteurisation on infectivity of *Cryptosporidium parvum* oocysts in water and milk. Applied and Environmental Microbiology 62: 2866-2868.

Jarroll EL, Hoff JC and Meyer EA (1984). Resistance of cysts to disinfection agents. In: Erlandsen SL, Meyer EA, eds Giardia and giardiasis: biology, pathogenesis and epidemiology pp 311-328. Plenum Press, New York.

Maheshwari G, Jannat R, McCormick L and Hsu D. (2004) Thermal inactivation of Adenovirus type 5. J Virol Methods 118:141-145.

Millard J, Appleton H and D Parry J. (1987) Studies on heat inactivation of Hepatitis A virus with special reference to shellfish. Epidemiol Infect. 98:397-414.

Moce-Llivina L, Muniesa M, Pimenta-Vale H, Lucena F and Jofre J (2003) Survival of bacterial indicator species and bacteriophages after thermal treatment of sludge and sewage. Applied and Environmental Microbiology. Vol 69, No 3: 1452-1456.

Ongerth J, Johnson R, MacDonald S, Frost F, Stibbs H (1989) Backcountry Water Treatment to Prevent Giardiasis. American Journal of Public Health. 79(12):1633-1637.

Parry J and Mortimer P. (1984) The heat sensitivity of Hepatitis A virus determined by a simple tissue culture method. Journal of Medical Virology 14(3):277-283.

Sattar SA, Chauret C, Springthorpe VS, Battigelli D, Abbaszadegan M and LecChevallier M (1999) *Giardia* cyst and *Cryptosporidium* oocyst survival in watersheds and factors affecting inactivation. AWWA Research Foundation and American Water Works Association. Denver Colorado.

Spinks A, Dunstan H, Harrison T, Coombes P, Kuczera G. (2006). Thermal inactivation of water-borne pathogenic and indicator bacteria at sub-boiling temperatures. Water Research 40: 1326-1332.

Stout J, Best M and Yu V. (1986) Susceptibility of members of the family Legionellaceae to thermal stress: Implications for heat eradication methods in water distribution systems Applied and Environmental Microbiology 52: 396-399.

Strazynski M, Kramer J and Becker B. (2002) Thernal inactivation of poliovirus type 1 in water, milk and yoghurt. Int J Food Microbiol. 74:73-78.

World Health Organization (2011) Guidelines for Drinking Water Quality.

PART V FACTSHEETS

MICROORGANISMS

MICROORGANISMS Microbial indicators

Bacteroides

(endorsed 2011)

GUIDELINE

No guideline value has been established for Bacteroides in drinking water. If used as an indicator and detected in drinking water, immediate action should be taken including investigation of potential sources of faecal contamination. The primary value of these organisms is as a tool for tracking and identifying sources of contamination.

GENERAL DESCRIPTION

Bacteroides is an anaerobic Gram-negative, non-spore-forming rod from the family *Bacteroidaceae*. *Bacteroides* is primarily found in the intestinal tract of humans and other animals, comprising up to one third of faecal flora and far outnumbering concentrations of *Escherichia coli* (Finegold *et al*. 1983, Layton *et al*. 2006). *Bacteroides* can be present at concentrations of up to 10^{10} to 10^{11} per gram of faeces. It is involved primarily in digestion and plays a role in excluding potential pathogens from the human gut. Due to its anaerobic nature, *Bacteroides* does not survive or grow in aerobic conditions, including in water supplies.

Although *Bacteroides* is not a frank pathogen, some species, for example *B. fragilis*, are opportunistic pathogens and can cause bacteraemias and abscess formation at multiple body sites (Wexler 2007).

Recent advances in molecular technology have resulted in the development of several human- and animal-specific genetic markers for *Bacteroides*. These methods have the potential not only to identify faecal contamination but also to discriminate between sources of contamination. Specific *Bacteroides* markers for humans and ruminants have been developed (Layton *et al*. 2006) and work is in progress on a marker specific to birds.

SOURCE AND OCCURRENCE

Bacteroides is present in very high numbers in faecal material from animals and humans. It does not grow or survive in oxygenated water. There appears to be a high degree of host specificity.

METHOD OF IDENTIFICATION AND DETECTION

Bacteroides can be cultured using anaerobic media (Kator and Rhodes 2003). In addition there is a rapid fluorescent antibody technique that can be used (Fiksdal and Berg 1987).

Specific molecular methods can distinguish various specific markers within *Bacteroides*. These techniques have been published but are not yet available as routine diagnostic methods. Most *Bacteroides* methods involve quantitative PCR of the 16SrRNA of the organism (Bernard and Field 2000, Layton *et al*. 2006, Reischer *et al*. 2007, Kildare *et al*. 2007, Ahmed *et al*. 2009).

INDICATOR VALUE AND APPLICATION IN PRACTICE

Bacteroides has been proposed as a suitable indicator of contamination of water supplies. It is present in high numbers in faeces and does not grow in water supplies. In addition, *Bacteroides* can be used as a tool in faecal source tracking (USEPA 2005). Understanding sources of contamination in source waters is important in developing sound risk management plans. *Bacteroides* can be used to assess source water quality and the relative impacts of human and livestock waste. Such information can improve the

accuracy of microbial risk assessments and identification of appropriate control measures. Faecal source tracking can also be used to investigate sources of post-treatment ingress of human and animal waste into distribution systems.

The presence of *Bacteroides* provides evidence of recent faecal contamination. Detection in drinking water should always lead to investigation of the cause, which could include inadequate treatment or ingress of contamination.

REFERENCES

Ahmed W, Goonetilleke A, Powell D, Gardner T (2009) Evaluation of multiple sewage-associated *Bacteroides* PCR markers for sewage pollution tracking. *Water Research* 43:4872-4877.

Bernhard AE, Field KG (2000). Identification of non-point sources of faecal pollution in coastal waters by using host-specific 16S ribosomal DNA genetic markers from faecal anaerobes. *Applied and Environmental Microbiology,* 66:1587-1594.

Finegold SM, Sutter VL, Mathisen GE (1983). Normal indigenous intestinal flora. In: ***Human Intestinal Microflora in Health and Disease***. Academic Press, London, pp. 3-31.

Fiksdal L, Berg JD (1987). Evaluation of a fluorescent antibody technique for the rapid enumeration of ***Bacteroides fragilis*** group of organisms in water. ***Journal of Applied Microbiology,*** 62(4):377-383.

Kator H, Rhodes M (2003). Detection enumeration and identification of environmental microorganisms of public health significance. In: Mara D, Horan N (eds), ***The Handbook of Water and Wastewater Microbiology.*** Academic Press London.

Kildare BJ, Leutenegger CM, McSwain BS, Bambic DG, Rajal VB, Wuertz S (2007). 16s rRNA-based assays for quantitative detection of universal, human-, cow-, and dog-specific faecal *Bacteroides*: A Bayesian approach. *Water Research* 41:3701-3715.

Layton A, McKay L, Williams D, Garrett V, Gentry R, Sayler G (2006). Development of ***Bacteroides*** 16S rRNA gene TaqMan-based real-time PCR assays for estimation of total, human and bovine faecal pollution in water. ***Applied and Environmental Microbiology,*** 72:4214-4224.

Reischer GH, Kasper DC, Steinborn R, Farnleitner AH, Mach RL (2007). A quantitative real-time PCR assay for the highly sensitive and specific detection of human faecal influence in spring water from a large alpine catchment area. *Letters in Applied Microbiology,* 44:351-356.

USEPA (United States Environmental Protection Agency) (2005). ***Microbial source Tracking Guide Document.*** EPA 600/R-05/064, USEPA, Cincinatti, USA.

Wexler H (2007). ***Bacteroides:*** the good, the bad, and the nitty-gritty. ***Clinical Microbiology Reviews,*** 20(4):593-621

NOTE: Important general information is contained in PART II, Chapter 5

Coliphages

(endorsed 2011)

GUIDELINE

Coliphages should not be detected in any 100 mL sample of drinking water. If used as an indicator and detected in drinking water, immediate action should be taken including investigation of potential sources of faecal contamination.

GENERAL DESCRIPTION

Bacteriophages (also known as phages) are viruses that exclusively use bacteria as their hosts for replication. Coliphages use *Escherichia coli* and closely related coliform bacteria as hosts and can be released by these bacterial hosts into the faeces of humans and other warm-blooded animals.

Coliphages used in drinking-water quality assessment are divided into two major groups: somatic coliphages and F-RNA coliphages. Differences between the two groups include the route of attachment and infection of bacterial cells.

Somatic coliphages initiate infection by attaching to receptors permanently located on the cell wall of host bacteria. The somatic coliphages replicate more frequently in the gastrointestinal tract of warm-blooded animals and could potentially replicate more readily in water environments than F-RNA coliphages. Somatic coliphages have DNA genomes that can be single or double-stranded. They comprise a wide range of phage families (Myoviridae, Siphoviridae, Podoviridae and Microviridae) with a spectrum of morphological types (Grabow 2001).

F-RNA coliphages initiate infection by attaching to fertility (F-) fimbriae on *E. coli* hosts. These F-fimbriae are produced only by bacteria carrying the fertility (F-) plasmid. Since F-fimbriae are produced only in the logarithmic growth phase at warmer temperatures, typically above 30°C, F-RNA coliphages are unlikely to replicate in environments other than the gastrointestinal tract of warm-blooded animals. F-RNA coliphages comprise a restricted group of closely related phages, which belong to the family Leviviridae and consist of a single-stranded RNA genome and an icosahedral capsid that is morphologically similar to that of picornaviruses (Grabow 2001).

SOURCE AND OCCURRENCE

Both somatic coliphages and F-RNA coliphages are routinely found in sewage. Somatic coliphages are found more commonly than F-RNA coliphages in the gastrointestinal tracts of humans and are typically found at higher numbers in sewage. This can be advantage in source water assessment. On the other hand, because F-RNA coliphages are unlikely to grow in the environment, they can be used as specific indicators of faecal contamination.

METHOD OF IDENTIFICATION AND DETECTION

There are standard methods for detection of somatic and F-RNA coliphages (ISO 1995, 2000) using plaque assays. The assays are based on growing lawns of specific host bacteria on agar-based media. Plaques represent holes within the lawn where coliphage infection has lysed the bacterial cells.

Somatic coliphages are detectable by relatively simple and inexpensive plaque assays, which yield results within 24 hours. Plaque assays for F-RNA coliphages are not quite as simple, as the culture of host bacteria has to be in the logarithmic growth phase at a temperature above 30°C to ensure that F-fimbriae are present.

NOTE: Important general information is contained in PART II, Chapter 5

INDICATOR VALUE AND APPLICATION

Phages share many properties with human viruses, notably composition, morphology, structure and mode of replication. As a result, coliphages are useful models or surrogates to assess the behaviour of enteric viruses in water environments. In this regard, they are superior to faecal bacteria. However, there is no direct correlation between numbers of coliphages and numbers of enteric viruses. Coliphages are sensitive to disinfectants such as chlorine (Grabow 2001, WHO 2004)

The presence of coliphages is indicative of the likely presence of faecal contamination, and they are more robust than bacterial indicators. Coliphages can be used in source water quality assessment to help detect the presence of possible viral contamination. They are particularly suitable for groundwater assessment, where the larger bacterial indicators might not be found due to natural filtration and adsorption processes. Coliphages are less useful for assessing surface waters, where concentrations tend to be low.

Coliphages are widely used in validation of treatment processes. The physical similarities between coliphages and viruses are particularly useful in validating efficacy of filtration (e.g., USEPA 2005) and they can also be used to validate disinfection processes (e.g., USEPA 2006).

Although testing is more costly than for bacterial indicators, coliphages can be valuable components of verification monitoring. The presence of coliphages in drinking water indicates shortcomings in treatment or in the protection of distribution systems, and detection should always lead to further investigations.

REFERENCES

Grabow WOK (2001). Bacteriophages: Update on application as models for viruses in water. *Water SA*, 27:251-268.

ISO 10705-1 (1995). Water quality – Detection and enumeration of bacteriophages – Part 1 Enumeration of F-specific RNA bacteriophages. International Organization for Standardization, Geneva, Switzerland.

ISO 10705-2 (2000). Water quality – Detection and enumeration of bacteriophages – Part 2 Enumeration of somatic coliphages. International Organization for Standardization, Geneva, Switzerland.

USEPA (United States Environmental Protection Agency) (2005). *Membrane Filtration Guidance Manual.* EPA 815-R-06-009, USEPA, Cincinatti, USA.

USEPA (United States Environmental Protection Agency) (2006). *Ultraviolet Disinfection Guidance Manual.* EPA 815-D-03-007, USEPA, Cincinatti, USA.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Section 11.6.6, Coliphages. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Clostridium perfringens

(endorsed 2011)

GUIDELINE

No guideline value has been set for* Clostridium perfringens *in drinking water. If used as an indicator and detected in drinking water, immediate action should be taken, including investigation of potential sources of faecal contamination.

GENERAL DESCRIPTION

Clostridium spp are anaerobic, sulfite-reducing, spore-forming bacilli. There are a number of species, of which *C. perfringens* is uniquely of faecal origin. Largely because it is anaerobic, *C. perfringens* rarely multiplies in water environments.

The spores are smaller than protozoan cysts and oocysts. They are exceptionally resistant to unfavourable conditions in water environments, including temperature and pH extremes, and are also resistant to disinfection processes such as chlorination.

SOURCE AND OCCURRENCE

C. perfringens is a member of the normal intestinal flora of 13-35% of humans, and is relatively common in dogs but less so in other warm-blooded animals (Leeming *et al*. 1998). The numbers excreted in faeces are normally substantially lower than those of *Escherichia coli*. *C. perfringens* and its spores are commonly present in sewage.

METHOD OF IDENTIFICATION AND DETECTION

There are standard methods for the detection and enumeration of *C. perfringens* using membrane filtration and multiple tube dilution methods (AS/NZS 4276.17.1 2000, AS/NZS 4276.17.2 2000). The assays involve incubation in selective media under anaerobic conditions. While assays for *C. perfringens* are more complicated than assays for other bacterial indicators such as *E. coli* or heterotrophic plate counts, they can be undertaken by standard microbiology laboratories.

INDICATOR VALUE AND APPLICATION

Due to their small size and exceptional resistance to disinfection processes and other unfavourable environmental conditions, *C. perfringens* spores have been proposed as potential indicators for enteric viruses and protozoa in drinking-water supplies (Payment and Franco 1993). However, usefulness in routine monitoring of source water quality is limited by the fact that concentrations in faeces and sewage are far lower than *E. coli*. In addition, the survival of *C. perfringens* is much longer than that of enteric viruses and protozoa; hence detection in treated drinking water needs to be treated with caution, as the spores could be present long after faecal pollution and after death of other enteric pathogens (WHO 2004).

Monitoring of *C. perfringens* in drinking water could be undertaken to assess the likelihood of intermittent faecal contamination in distribution systems. If *C. perfringens* is detected, potential sources of contamination should be immediately investigated.

C. perfringens could be a useful indicator for validating removal of protozoa by filtration (WHO 2004).

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

AS/NZS 4276.17.1 (2000). Water Microbiology Method 17.1: Spores of sulfite-reducing anaerobes (clostridia) including *Clostridium perfringens* – Membrane filtration method. Standards Australia, Standards New Zealand.

AS/NZS 4276.17.2 (2000). Water Microbiology Method 17.2: Spores of sulfite-reducing anaerobes (clostridia) including *Clostridium perfringens* – Estimation of most probable number (MPN) using the multiple tube dilution technique. Standards Australia, Standards New Zealand.

Leeming R, Nichols PD, Ashbolt NJ (1998). *Distinguishing sources of faecal pollution in Australian inland and coastal waters using sterol biomarkers and microbial faecal indicators.* Research Report 204, Water Services Association of Australia.

Payment P, Franco E (1993). *Clostridium perfringens* and somatic coliphages as indicators of the efficiency of drinking-water treatment for viruses and protozoan cysts. *Applied and Environmental Microbiology*, 59:2418-2424.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Section 11.6.5, *Clostridium perfringens*. Geneva: World Health Organization.

NOTE: Important general information is contained in PART II, Chapter 5

Escherichia coli

(endorsed 2011)

GUIDELINE

Escherichia coli *should not be detected in any 100 mL sample of drinking water. If detected in drinking water, immediate action should be taken including investigation of potential sources of faecal contamination.*

GENERAL DESCRIPTION

Coliforms are Gram-negative, non-spore-forming, rod-shaped bacteria that are capable of aerobic and facultative anaerobic growth in the presence of bile salts or other surface active agents with similar growth-inhibiting properties. They are found in large numbers in the faeces of humans and other warm-blooded animals, but many species also occur in the environment.

Thermotolerant coliforms are a sub-group of coliforms that are able to grow at 44.5 ± 0.2°C. *E. coli* is the most common thermotolerant coliform present in faeces and is regarded as the most specific indicator of recent faecal contamination because generally it is not capable of growth in the environment. In contrast, some other thermotolerant coliforms (including strains of *Klebsiella, Citrobacter* and *Enterobacter)* are able to grow in the environment and their presence is not necessarily related to faecal contamination. While tests for thermotolerant coliforms can be simpler than for *E. coli*, *E. coli* is considered a superior indicator for detecting faecal contamination.

Thermotolerant coliforms, including *E. coli,* can ferment lactose (or mannitol) at 44.5 ± 0.2°C with the production of acid within 24 hours. Thermotolerant coliforms that produce indole from tryptophan at 44.5 ± 0.2°C are regarded as being *E. coli*. *E. coli* also gives a positive result in the methyl-red test and a negative Voges-Proskauer test, and it cannot use citrate as the sole source of carbon. Most *E. coli* produce the enzyme ß-glucuronidase.

SOURCE AND OCCURRENCE

E. coli is a normal inhabitant of the intestine, generally present in high numbers in human and animal faeces, and it generally does not grow in natural waters, although there have been reports that it can multiply in tropical waters (Fujioka *et al*. 1999) and three atypical strains were reported as being able to grow in two Australian lakes (Power et al. 2005). Two of the three atypical strains were ß-glucuronidase negative. While most *E. coli* are non-pathogenic, there are some pathogenic subtypes that can cause enteric illness, including enteropathogenic, enteroinvasive, enterotoxigenic and enterohaemorrhagic strains (Bopp 1999). These are described in the fact sheet on Pathogenic *Escherichia coli*.

METHOD OF IDENTIFICATION AND DETECTION

The presence of *E. coli* in water samples can be determined using a number of methods. A common method involves membrane filtration (MF) for concentration of the organisms from water, followed by growth in enrichment/selective media or multiple tube dilution (most probable number – MPN) procedures (AS/NZS 4276.6 2007, AS/NZS 4276.7 2007). Specific secondary tests are used with both MF and MPN procedures to confirm the identification of *E. coli*.

Alternatively, *E. coli* can be detected by testing for the production of the enzyme ß-glucuronidase (AS 4276.21 2005). Test media include enzyme substrates such as 4-methylumbelliferyl-ß-D-

NOTE: Important general information is contained in PART II, Chapter 5

glucuronide (MUG) which is hydrolysed by ß-glucuronidase to produce the fluorogenic metabolite 4-methylumbelliferyl. Both enumeration and presence/absence tests are available from a number of commercial suppliers.

INDICATOR VALUE AND APPLICATION IN PRACTICE

E. coli is used as a specific indicator of recent faecal contamination. It can be used to assess:

- source water quality and potential impacts of human and animal waste;
- inadequate treatment;
- post-treatment ingress of human and animal waste into distribution systems;
- the effectiveness of risk management plans in assuring delivery of safe drinking water at consumers' taps.

E. coli is not an effective indicator for the presence of enteric protozoa or viruses.

E. coli should not be present in any 100 mL sample of drinking water. Risk management plans should incorporate corrective actions in the event of the detection of *E. coli* in drinking water. The presence of these organisms can indicate faecal contamination of the water supply, and if they are detected in drinking water, the cause should always be investigated. Possible causes include inadequate treatment or ingress of contamination. Investigation will generally require further testing.

REFERENCES

AS/NZS 4276.6 (2007). Water Microbiology. Method 6: Coliforms, *Escherichia coli* and thermotolerant coliforms – Determination of most probable number (MPN). Standards Australia, Sydney, NSW; Standards New Zealand.

AS 4276.7 (2007). Water Microbiology. Method 7: *Escherichia coli* and thermotolerant coliforms – Membrane filtration method. Standards Australia, Sydney, NSW; Standards New Zealand.

AS 4276.21 (2005). Water Microbiology. Method 21. Examination for coliforms and *Escherichia coli*- Determination of most probable number (MPN) using enzyme hydrolysable substrates. Standards Association of Australia, Sydney, NSW.

Bopp CA, Brenner FW, Wells JG, Strockbine NA (1999). Escherichia, Shigella and Salmonella. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Volken RH (eds), *Manual of Clinical Microbiology*, 7th edition. ASM Press, Washington DC, pp 459-474.

Fujioka R, Sian-Denton C, Borja M, Castro J, Morphew K (1999). Soil: the environmental source of *Escherichia coli* and Enterococci in Guam's streams. *Journal of Applied Microbiology Symposium Supplement,* 85:83S-89S.

Power ML, Littlefield-Wyer J, Gordon DM, Veal DA and Slade MB (2005) Phenotypic and genotypic characterization of encapsulated *Escherichia coli* isolated from blooms in two Australian lakes. Environmental Microbiology 7(5), 631-640.

NOTE: Important general information is contained in PART II, Chapter 5

Heterotrophic plate counts

(endorsed 2011)

GUIDELINE

No guideline value has been set for heterotrophic plate counts in drinking water. Immediately after disinfection, numbers would be expected to be low. If used as an indicator of distribution system cleanliness, numbers should be established on a system-specific basis. Marked increases in numbers after disinfection or within distribution systems should be investigated.

GENERAL DESCRIPTION

Use of heterotrophic plate counts (HPC) has a long history. In the 19th century Robert Koch referred to use of total bacterial counts in assessing performance of sand filtration (Bartram *et al.* 2003).

Heterotrophs are broadly defined as microorganisms that require organic carbon for growth. They include bacteria and fungi. HPC refer to numbers of organisms grown on non-specific culture media without inhibitory or selective agents. Even though the test is non-selective, only a small proportion of the microorganisms present in water will be recovered. The types of organisms detected by HPC tests vary widely with location and time of year. Tests detect microorganisms that grow over a specified incubation period and at a defined temperature. Incubation periods can range from one day to weeks, and temperatures from 20°C to 40°C.

Microorganisms detected within HPC include:

- vegetative bacteria such as coliforms and other Enterobacteriacae that are sensitive to disinfection processes;
- fungi and bacteria such as *Bacillus* spp that form disinfectant-resistant spores; and
- bacteria and fungi that grow in water.

Although HPC can include enteric pathogens and opportunistic pathogens such as *Aeromonas* spp and *Pseudomonas* spp, the vast majority are non-pathogenic.

HPC are one of the simplest tests that can be performed in monitoring of water quality. Other names used for HPC include total plate counts or colony counts.

SOURCE AND OCCURRENCE

Heterotrophic microorganisms include the naturally occurring microbial flora of water and soil environments (typically non-hazardous) and organisms present in a range of pollution sources. They occur in large numbers in raw water sources. Some drinking-water treatment processes, such as coagulation and sedimentation, reduce the number of HPC organisms in water but growth can occur in other processes such as sand filtration. Numbers of HPC organisms are reduced significantly by disinfection processes; however, they can grow rapidly in drinking water once disinfection residuals have dissipated. Heterotrophs can also grow on surfaces in contact with water as biofilms. The principal determinants of growth or "regrowth" are temperature; availability of nutrients, including assimilable organic carbon; lack of disinfectant residual; and stagnation.

METHOD OF IDENTIFICATION AND DETECTION

No sophisticated laboratory facilities or highly trained staff are required. HPC are determined using aerobic incubation of non-selective nutrient agar.

NOTE: Important general information is contained in PART II, Chapter 5

Various nutrient media are available to determine HPC, including tryptone glucose yeast agar, R2A agar, and yeast extract agar (APHA *et al.* 2005, AS/NZS 4276.3.1 2007). It is standard practice to perform two different tests, one at 20-22°C for 3-5 days and a second at 35-37°C for 1-2 days.

INDICATOR VALUE AND APPLICATION IN PRACTICE

The use of HPC as an indicator of safety declined with the adoption of testing for faecal indicators such as *E. coli*. There is no evidence that HPC alone can be used as an index of pathogen presence or directly relate to health risk through normal uses of drinking water by the general population (Bartram *et al.* 2003).

They can, however, be a useful component of operational monitoring. HPC can be used in conjunction with measuring disinfectant residual or dose for operational monitoring of disinfection processes. In this case, the objective is to keep HPC numbers as low as possible. They can also be used to monitor the integrity, cleanliness and maintenance of distribution systems and the presence of biofilms. For this type of monitoring, absolute numbers are less important than changes in numbers and the objective is to keep HPC numbers within defined limits. Marked increases, measured in orders of magnitude, provide evidence of deteriorating conditions that should be investigated. The causes could include inadequate treatment, loss of disinfection residual, and stagnant water.

REFERENCES

APHA, AWWA, WEF (American Public Health Association, American Water Works Association, Water Environment Federation (2005). *Standard methods for the examination of water and wastewater, 21st edition*. American Public Health Association, Washington DC.

AS/NZS 4276.3.1 (2007). Water Microbiology Method 3.1: Heterotrophic colony count methods – pour plate method using yeast extract agar. Standards Australia, Standards New Zealand.

Bartram J, Cotruvo J, Exner M, Fricker M, Glasmacher A (2003). *Heterotrophic Plate Counts and Drinking-water Safety: the significance of HPCs for water quality and human health*. WHO Emerging Issues in Water and Infectious Disease Series. IWA Publishing, London.

NOTE: Important general information is contained in PART II, Chapter 5

Intestinal enterococci

(endorsed 2011)

GUIDELINE

Intestinal enterococci should not be present in drinking water. If used as an indicator and detected in drinking water, immediate action should be taken, including investigation of potential sources of faecal contamination.

GENERAL DESCRIPTION

Intestinal enterococci are a functional group of organisms from the *Enterococcus* and *Streptococcus* genera that are excreted in human and animal waste. Species include *Enterococcus faecalis*, *E. faecium, E. durans*, *E. hirae*, *E. cecorum*, *E. columbae, E. avium* and *E. gallinarum* together with *Streptococcus bovis* and *S. equinus* (ISO 1998, Ashbolt *et al*. 2001, WHO 2003).

Use of the terms intestinal enterococci, faecal streptococci, enterococci and streptococci has been a source of some confusion. This has been exacerbated by revisions of taxonomy. The older taxonomy described faecal streptococci as a subgroup of the genus *Streptococcus*. The group including *S. faecalis*, *S. faecium*, *S. bovis*, *S. equinus*, *S. avium*, and *S. gallinarum* all possess the Lancefield group D antigen. The enterococci *S. faecalis*, *S. faecium, S .avium* and *S. gallinarum* represented a smaller subgroup of the faecal streptococci, which was differentiated by an ability to grow in 6.5% sodium chloride, at pH 9.6 and at both 10ºC and 45ºC (APHA *et al*. 2005). The nomenclature of this subgroup has been changed and they are now identified as *Enterococcus* spp: *E. faecalis*, *E. faecium, E. avium* and *E. gallinarum* (LeClerc *et al*. 1996). These species are principal members of the intestinal enterococci, together with other enterococci of faecal origin.

The similar membership of the various groups means that tests for intestinal enterococci, faecal streptococci, enterococci and streptococci often provide the same results.

SOURCE AND OCCURRENCE

Intestinal enterococci are excreted in the faeces of humans and other warm-blooded animals, including livestock, domestic animals and birds (Ashbolt *et al*. 2001). Most species do not grow in water but the standard test for intestinal enterococci can detect environmental species such as *E. casseliflavus* and *E. mundtii* (ISO 1998).

Intestinal enterococci are present in large numbers in sewage and can be present in water environments polluted by sewage or wastes from humans and animals.

METHOD OF IDENTIFICATION AND DETECTION

Intestinal enterococci can be detected by a liquid- or agar-based culture method requiring standard microbiology laboratory facilities.

A standardised test for intestinal enterococci in water has been described by the International Organization for Standardization (ISO 1998). The test is based on the capability of growing at 44ºC and of hydrolysing 4-methylumbelliferyl- -D-glucoside (MUD) in the presence of thallium acetate, naladixic acid and 2,3,5-triphenyltetrazolium chloride (TTC). The test is performed in a liquid medium. It detects enterococci of faecal origin and may occasionally detect strains of *S. bovis* and *S. equinius*. It also detects non-faecal species such as *E. casselifalvus* and *E. mundtii*.

A membrane filtration method is also available (AS/NZS 4276.9, 2007) using initial isolation on m-Enterococcus agar (36 ± 2ºC for 44 ± 4 hrs) followed by confirmation on Bile Aesulin Azide Agar (44 ± 0.5ºC for 2 hrs).

NOTE: Important general information is contained in PART II, Chapter 5

INDICATOR VALUE AND APPLICATION IN PRACTICE

Numbers of intestinal enterococci in sewage are generally about an order of magnitude lower than those of *E. coli*. An important advantage of this group is that they tend to survive longer in water environments than *E. coli* (Ashbolt *et al*. 2001, WHO 2004), and they have been used as an index of faecal pathogens that survive longer in water than *E. coli*. While they are more resistant to disinfection than *E. coli*, they are readily removed by disinfectants used to treat drinking water.

It has been suggested that intestinal enterococci (faecal streptococci) to *E. coli* ratios could be used to indicate whether faecal waste is of human or animal origin, but this is confounded by a number of factors including different rates of die-off for the various species in water environments, and it is not considered reliable (Sinton and Donnison 1994, Ashbolt *et al*. 2001).

The presence of intestinal enterococci provides evidence of recent faecal contamination. They can be used to assess:

- source water quality and potential impacts of human and animal waste;
- the adequacy of treatment;
- whether there is post-treatment ingress of human and animal waste into distribution systems; and
- the effectiveness of risk management plans in assuring delivery of safe drinking water at consumer taps (verification).

Although the intestinal enterococci as a group survive longer than *E. coli* they are not effective indicators for the presence of enteric protozoa or viruses.

The detection of intestinal enterococci in drinking water should always lead to investigation of the cause, which could include inadequate treatment or ingress of contamination. Investigation will generally require further testing.

REFERENCES

APHA, AWWA, WEF (American Public Health Association, American Water Works Association, Water Environment Federation (2005). *Standard methods for the examination of water and wastewater, 21st edition*. American Public Health Association, Washington DC.

AS/NZS 4276.9 (2007). *Water Microbiology Method 9: Enterococci – membrane filtration method* (ISO 7899-2:2000, MOD). Standards Australia, Standards New Zealand.

Ashbolt NJ, Grabow WOK, Snozzi M (2001). Indicators of microbial water quality. In: Fewtrell L, Bartram J, eds. *Water quality: Guidelines, standards and health — Assessment of risk and risk management for water-related infectious disease*. WHO Water Series. London, IWA Publishing, pp. 289-315.

ISO 7899-1:1998 (1998). *Water Quality – Detection and Enumeration of Intestinal Enterococci in Surface and Wastewater – Part 1 Minituarized method (most probable number) by inoculation in liquid medium*. International Organization for Standardization, Geneva, Switzerland.

LeClerc H, Devries LA, Mossel DAA (1996). Taxonomical changes in intestinal (faecal) enterococci and streptococci: consequences on their use as indicators of faecal contamination in drinking water. *Journal of Applied Bacteriology,* 81:459-466.

Sinton LW, Donnison AM (1994). Characterisation of faecal streptococci from some New Zealand effluents and receiving waters. *New Zealand Journal of Marine and Freshwater Research*, 28:145-158.

WHO (World Health Organization) (2003). *Guidelines for Safe Recreational Water Environments. Volume 1 Coastal and fresh waters*. WHO, Geneva, Switzerland.

WHO (World Health Organization) (2004). *Guidelines for Drinking-Water Quality.* WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Thermotolerant coliforms

(endorsed 2011)

GUIDELINE

Thermotolerant coliforms should not be detected in any 100 mL sample of drinking water. If detected in drinking water, immediate action should be taken including investigation of potential sources of faecal contamination.

GENERAL DESCRIPTION

Coliforms are Gram-negative, non-spore-forming, rod-shaped bacteria that are capable of aerobic and facultative anaerobic growth in the presence of bile salts or other surface active agents with similar growth-inhibiting properties. They are found in large numbers in the faeces of humans and other warm-blooded animals, but many species also occur in the environment.

Thermotolerant coliforms are a sub-group of coliforms that are able to grow at 44.5 ± 0.2°C. *E. coli* is the most common thermotolerant coliform present in faeces and is regarded as the most specific indicator of recent faecal contamination because generally it is not capable of growth in the environment. In contrast, some other thermotolerant coliforms (including strains of *Klebsiella, Citrobacter* and *Enterobacter)* are able to grow in the environment and their presence is not necessarily related to faecal contamination.

Thermotolerant coliforms, including *E. coli,* can ferment lactose (or mannitol) at 44.5 ± 0.2°C with the production of acid within 24 hours.

SOURCE AND OCCURRENCE

Thermotolerant coliforms are normal inhabitants of the intestine, generally present in high numbers in human and animal faeces;. However, environmental thermotolerant coliforms, can occur in natural waters. These organisms are of lesser significance.

METHOD OF IDENTIFICATION AND DETECTION

The presence of thermotolerant coliforms in water samples can be determined using a number of methods. A common method involves membrane filtration (MF) for concentration of the organisms from water, followed by growth in enrichment/selective media or multiple tube dilution (most probable number – MPN) procedures (AS/NZS 4276.6 2007, AS/NZS 4276.7 2007). Specific secondary tests are used with both MF and MPN procedures to confirm the identification of thermotolerant coliforms.

INDICATOR VALUE AND APPLICATION IN PRACTICE

Thermotolerant coliforms can be used as an indicator of faecal contamination but they are not as specific as *E. coli*, which is the preferred indicator. The group includes types that can grow in the environment and be present in the absence of faecal contamination.

The presence of thermotolerant coliforms may provide evidence of recent faecal contamination. Thermotolerant coliforms can be used to assess:

- source water quality and potential impacts of human and animal waste;
- inadequate treatment;
- post-treatment ingress of human and animal waste into distribution systems;

NOTE: Important general information is contained in PART II, Chapter 5

- the effectiveness of risk management plans in assuring delivery of safe drinking water at consumers' taps.

Thermotolerant coliforms are not an effective indicator for the presence of enteric protozoa or viruses.

Thermotolerant coliforms should not be present in any 100 mL sample of drinking water. Risk management plans should incorporate corrective actions in the event of the detection of thermotolerant coliforms in drinking water. The presence of these organisms may indicate faecal contamination of the water supply, and if they are detected in drinking water, the cause should always be investigated. Possible causes include inadequate treatment or ingress of contamination. Investigation will generally require further testing.

REFERENCES

AS/NZS 4276.6 (2007). Water Microbiology. Method 6: Coliforms, *Escherichia coli* and thermotolerant coliforms – Determination of most probable number (MPN). Standards Australia, Sydney, NSW; Standards New Zealand.

AS 4276.7 (2007). Water Microbiology. Method 7: *Escherichia coli* and thermotolerant coliforms – Membrane filtration method. Standards Australia, Sydney, NSW; Standards New Zealand.

NOTE: Important general information is contained in PART II, Chapter 5

Total coliforms

(endorsed 2011)

GUIDELINE

No guideline value has been set for total coliforms in drinking water. If used as an indicator, numbers should be established on a system-specific basis. Increased concentrations should be investigated.

GENERAL DESCRIPTION

Coliforms are Gram-negative, non-spore-forming, rod-shaped bacteria that are capable of aerobic and facultative anaerobic growth in the presence of bile salts or other surface active agents with similar growth-inhibiting properties. They are able to ferment lactose with the production of acid within 48 hours at 35-37°C. Fermentation by these organisms begins with the cleavage of lactose into galactose and glucose by the enzyme ß-galactosidase. Coliforms are oxidase-negative. These characteristics are not taxonomic criteria, but practical working definitions used for water examination purposes.

Coliforms are a diverse group of bacteria including *Escherichia coli* and other thermotolerant coliforms (see also Fact Sheets on *Escherichia coli* and Thermotolerant Coliforms, under Microbial Indicators, and Pathogenic *Escherichia coli*, under Bacteria). Human and animal faeces contains large numbers of coliform bacteria, but there are many species that occur naturally in the environment. For this reason, coliforms can be present and grow in biofilms in drinking-water distribution systems. Coliforms that have been recovered from distribution systems include the non-thermotolerant genera *Serratia, Hafnia* and *Pantoea* as well as thermotolerant genera including *Klebsiella and Enterobacter*. Their presence in water, in the absence of *E. coli*, does not necessarily indicate faecal contamination.

SOURCE AND OCCURRENCE

Total coliform bacteria (excluding *E. coli*) occur in both sewage and natural waters. Some of these bacteria are excreted in the faeces of humans and animals, but many coliforms are heterotrophic and able to multiply in water and soil environments. Total coliforms can also survive and grow in water distribution systems, particularly in the presence of biofilms.

METHOD OF IDENTIFICATION AND DETECTION

Total coliforms can be quantified in water by a number of techniques. Membrane filtration (MF) can be used for concentration of the organisms from water, followed by growth in enrichment/selective media or multiple tube dilution (most probable number – MPN) procedures (AS/NZS 4276.5. 2007, AS/NZS 4276.6. 2007). Specific secondary tests are used with both MF and MPN procedures to confirm the identification of coliform organisms.

Alternatively, the presence of coliform bacteria can be detected by testing for the production of the enzyme ß-galactosidase (AS 4276.21 2005). Enzyme substrate tests incorporate chromogenic substrates such as ortho-nitrophenyl-ß-D-galactopyranoside (ONPG) or chlorophenol red-ß-Dgalactopyranoside (CPRG). When the substrates are hydrolysed, a colour change is produced.

It has been reported that more coliform bacteria may be detected using enzyme substrate-based methodology than with MF-based methodology (Adcock and Saint 1997).

NOTE: Important general information is contained in PART II, Chapter 5

INDICATOR VALUE AND APPLICATION IN PRACTICE

Total coliforms (excluding *E. coli)* are not considered useful as indicators of the presence of faecal contamination and enteric pathogens, as there are many environmental coliforms that are not of faecal origin. The presence of these coliforms may represent release from pipe or sediment biofilms, and may be part of the normal flora of the drinking-water distribution system.

No guideline value has been set for total coliforms in drinking water. If used as an indicator, numbers should be established on a system-specific basis, taking into consideration relevant historical data and an understanding of the characteristics of the system. While coliforms can be used in operational monitoring to indicate inadequate treatment, breakdowns in system integrity, or the presence of biofilms, there are better indicators for these purposes. As a disinfection indicator, the test for total coliforms is far slower and less reliable than direct measurement of disinfectant residual. Heterotrophic plate count tests detect a wider range of microorganisms and are generally considered a better indicator of distribution system integrity and cleanliness.

REFERENCES

Adcock PW, Saint C (1997). Trials of Colilert System. *Water,* 24(2):22-25.

AS/NZS 4276.5 (2007). Water Microbiology. Method 5 Coliforms – Membrane filtration method. Standards Australia, Sydney, NSW; Standards New Zealand.

AS/NZS 4276.6 (2007). Water Microbiology. Method 6: Coliforms, *Escherichia coli* and thermotolerant coliforms – Determination of most probable number (MPN). Standards Australia, Sydney, NSW; Standards New Zealand.

AS 4276.21 (2005). Water Microbiology. Method 21. Examination for coliforms and Escherichia coli-Determination of most probable number (MPN) using enzyme hydrolysable substrates. Standards Association of Australia, Sydney, NSW.

NOTE: Important general information is contained in PART II, Chapter 5

MICROORGANISMS BACTERIA

Aeromonas

(endorsed 1996)

GUIDELINE

No guideline value has been set for* Aeromonas *in drinking water. The absence of* Escherichia coli *(or alternatively thermotolerant coliforms) does not indicate the absence of Aeromonas.

GENERAL DESCRIPTION

Aeromonas spp are Gram-negative, rod-shaped, nonsporing bacteria which are presently classified in the family Vibrionaceae, although they also bear many similarities to the Enterobacteriaceae. They are isolated from certain patients with diarrhoea and may cause septicaemia.

The genus *Aeromonas* is divided into two groups. The group of psychrophilic nonmotile aeromonads consists of one species *A. salmonicida*, an obligate fish pathogen that will not be considered further here. The group of mesophilic motile aeromonads consists of three biochemically distinguishable groups: *A. hydrophila, A. sobria* and *A. caviae.* Each of these three species consists of at least three different DNA-hybridisation groups.

Aeromonas is a normal inhabitant of fresh water, and occurs in water, soil and food, particularly meat, fish and milk.

AUSTRALIAN SIGNIFICANCE

Aeromonas spp have been isolated from several drinking waters in Australia but the relationship between the isolates and clinical disease is not clear.

TREATMENT OF DRINKING WATER

Free available chlorine residuals of 0.2–0.5 mg/L are generally sufficient to control Aeromonas in distribution systems.

METHOD OF IDENTIFICATION AND DETECTION

The numbers of *Aeromonas* in drinking water can be quantified using membrane filtration and anaerobic incubation (Cunliffe and Adcock 1989).

HEALTH CONSIDERATIONS

Mesophilic aeromonads have long been known to be pathogenic for cold-blooded animals such as fish and amphibians. In humans, three types of infections are described: systemic infections, usually in people who are seriously immunocompromised; wound infections (mainly surface contact); and diarrhoea (Jana *et al.* 1988). They have given rise to serious cases of septicaemia, often in people with underlying disease; and they have been linked with gastroenteritis in children (Gracey *et al.* 1982), although no causative role has been established, and their significance as an enteropathogenic organism is not clear. In animal test models, such as the suckling mouse test and the rabbit ileal loop test, pure cultures of *Aeromonas* have been found to cause marked fluid accumulation. This can partially be ascribed to the production of extracellular cytotoxins; however, despite the strong toxin production by *Aeromonas* strains *in vitro*, it has not been possible to induce diarrhoea in test animals or human volunteers.

NOTE: Important general information is contained in PART II, Chapter 5

It is assumed that *Aeromonas* strains are only poorly able to colonise the gastrointestinal tract. Little information is available on adhesion factors of *Aeromonas* or their interaction with receptors in the gastrointestinal tract.

Epidemiological investigations on the significance of *Aeromonas* as an enteropathogenic organism have been contradictory. In some studies the occurrence of *Aeromonas* in faeces of patients with diarrhoea was higher than in control groups, whereas other studies showed no difference. Sometimes the bacterium was even found more often in control groups. Aeromonads have sometimes been associated with acute self-limiting gastroenteritis.

DERIVATION OF GUIDELINE

No specific guideline value can be established for *Aeromonas* because of difficulties in determining the pathogenicity of an isolate and its relevance to human health. Further work in the area is currently under way in Australia. Water must be tested directly for *Aeromonas* if their presence is suspected.

REFERENCES

Cunliffe DA and Adcock P (1989). Isolation of *Aeromonas* spp. from water by using anaerobic incubation. *Applied and Environmental Microbiology*, 55, 2138–2140.

Gracey M, Burke V and Robinson J (1982). Aeromonas-associated gastroenteritis. *Lancet*, ii, 1304–1306.

Jana JM and Duffy PS (1988). Mesophilic aeromonads in human disease: current taxonomy, laboratory identification, and infectious disease spectrum. *Reviews of Infectious Diseases*, 10, 980.

Burkholderia pseudomallei

(endorsed 2001)

GUIDELINE

No guideline value has been established for* Burkholderia pseudomallei *in drinking water.

GENERAL DESCRIPTION

Burkholderia pseudomallei, which causes the disease melioidosis, is a motile Gram-negative bacillus commonly found in soil and muddy water in tropical regions. *B. pseudomallei* can survive in water for prolonged periods in the absence of nutrients, and is acid tolerant (Wuthiekanun *et al.* 1995).

Melioidosis is most common in northern Australia and South-East Asia. Infection usually results from contact with soil or surface-accumulated water (muddy water). Exposure to environmental *B. pseudomallei* after heavy rainfall presents the greatest risk. Most infection appears to be through skin cuts or abrasions; however, infection may also occur via other routes, particularly through inhalation or ingestion. The relative importance of these routes of infection is not known.

AUSTRALIAN SIGNIFICANCE

Melioidosis is an endemic disease in northern Australia and although generally a tropical illness it has been detected in the southwest of Western Australia (Golledge *et al.* 1992). The first human case was diagnosed in Australia in 1950. Melioidosis is reported with increasing frequency in the Top End of the Northern Territory and *B. pseudomallei* is the most common organism isolated in fatal community acquired pneumonia. Cases appear throughout the year but peak during the rainy season (Currie 2000).

Two outbreaks of melioidosis have been reported in Australia: in 1990–91 in the Northern Territory and in 1997 in Western Australia (Inglis *et al.* 1999). In the latter outbreak, indistinguishable isolates of ***B. pseudomallei*** were cultured from cases and the potable water supply (Inglis *et al.* 1999, 2000).

MANAGEMENT

Standard disinfection procedures should be sufficient to eliminate *B. pseudomallei* from water supplies.

METHOD OF IDENTIFICATION AND DETECTION

Selective culture techniques have been described (Brook *et al.* 1997). Confirmation of identity as traditionally been done by biochemical tests (Inglis *et al.* 1998) but polymerase chain reaction (PCR) based methods may be more accurate. Genetic typing can be performed by several methods, including ribotyping and pulsed fi eld gel electrophoresis (Haase *et al.* 1995; Inglis *et al.* 2000).

HEALTH CONSIDERATIONS

Melioidosis is a potentially fatal disease. Pneumonia is the most common presentation. Many patients present with milder forms of pneumonia, which respond well to appropriate antibiotics, but some may present with a severe septicaemic pneumonia. Other symptoms include skin abscesses or ulcers, abscesses in internal organs and unusual neurological illnesses such as brainstem encephalitis and acute paraplegia. Individuals without symptoms or known history of disease may also be positive on serological testing. Late onset disease, including acute septicaemia, can occur months or years after initial exposure.

NOTE: Important general information is contained in PART II, Chapter 5

Although melioidosis can occur in healthy children and adults, it mainly occurs in people whose defences against infection are impaired, due either to an underlying condition (e.g. diabetes, chronic renal or lung disease, or alcoholic liver disease), or to poor general health associated with poor nutrition or living conditions.

DERIVATION OF GUIDELINE

No guideline is proposed for *B. pseudomallei* because there is limited evidence for the involvement of drinking water in its transmission in Australia. The numbers of organisms that would be significant for human health are unknown.

If a water supply is implicated as a possible source of melioidosis, investigations should be undertaken to assess whether the supply has been well managed and continually disinfected. The supply should be tested for the presence of the organisms.

REFERENCES

Brook MD, Currie B and Desmarchelier PM (1997). Isolation and identification of *Burkholderia pseudomallei* from soil using selective culture techniques and the polymerase chain reaction. *Journal of Applied Microbiology,* 82, 589–596.

Currie BJ (2000). The epidemiology of melioidosis in Australia and Papua New Guinea. *Acta Tropica*, 74, 121–127.

Haase A, Smith-Vaughan H, Melder A, Wood Y, Janmaat A, Gilfedder J, Kemp D and Currie B (1995). Subdivision of *Burkholderia pseudomallei* ribotypes into multiple types by RAPD analysis provides new insights into its epidemiology. *Journal of Clinical Microbiology*, 33, 1687–1690.

Inglis TJJ, Garrow SC, Adams C, Henderson M, Mayo M and Currie BJ (1999). Acute melioidosis outbreak in Western Australia. *Epidemiology and Infection,* 123, 437–443.

Inglis TJJ, Garrow SC, Henderson M, Clair A, Sampson J, O'Reilly L and Cameron B (2000). Outbreak strain of *Burkholderia pseudomallei* traced to water treatment plant. *Emerging Infectious Diseases*, 6, 56–59.

Inglis TJJ, Chiang D, Lee GSH and Lim CK (1998). Potential misidentification of *Burkholderia pseudomallei* by API 20NE. *Pathology,* 30, 62–64.

Golledge CL, Chin WS, Tribe AE, Condon RJ and Ashdown LR (1992), A case of human melioidosis originating in south west Western Australia. *Medical Journal of Australia*, 157, 332–334.

Wuthiekanun V, Smith MD and White NJ (1995). Survival of *Burkholderia pseudomallei* in the absence of nutrients. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 89, 491.

NOTE: Important general information is contained in PART II, Chapter 5

Campylobacter

(endorsed 1996)

GUIDELINE

***Escherichia coli* (or alternatively thermotolerant coliforms) can be used to indicate the possible presence of pathogenic Campylobacter. If explicitly sought, Campylobacter spp should not be detected. If detected, advice should be sought from the relevant health authority.**

GENERAL DESCRIPTION

Thermophilic *Campylobacter* spp are transmitted by the oral route, and cause gastrointestinal illness. Wild birds and poultry are the most important reservoirs of *Campylobacter*. Other domestic animals, such as pigs, cattle, dogs and cats, are also reservoirs of thermophilic *Campylobacter* organisms, and so meat, and particularly poultry products and unpasteurised milk, are important sources of *Campylobacter* infection. Milk may be contaminated with faeces or by secretion of organisms into the milk of cows with mastitis. Recent studies have shown that raw sewage frequently contains from 10 to 105 thermophilic *Campylobacter* organisms per 100 mL; high counts can be reduced by wastewater treatment processes. Thermophilic *campylobacters* have been found in crude sewage sludge, but were not detectable in digested conditioned sludge or filter effluent. Their occurrence in surface waters is dependent on rainfall, water temperature and the presence of water fowl.

Several waterborne outbreaks caused by *Campylobacter* spp have been reported in the past decade worldwide. The number of people involved ranged from a few to several thousand. Water was implicated in the only two of these outbreaks where *Campylobacter* was isolated from patients the main sources were found to be unchlorinated surface water and faecal contamination of water storage reservoirs by wild birds. Communities are at risk of outbreaks of campylobacteriosis from the consumption of unchlorinated or inadequately chlorinated surface waters. Contamination of drinking water reservoirs by excrement of water fowl should be controlled, particularly if *Campylobacter* contamination is suspected. Hygienic precautions should be improved in case the water is distributed without disinfection, or disinfection is interrupted.

Campylobacter spp, like other bacterial pathogens, survive well at low temperatures, and can survive for several weeks in cold groundwater or unchlorinated tap water.

The presence of thermophilic *Campylobacter* organisms in piped water supplies, whether treated or untreated, suggests a serious fault in the design or management of the system.

Two closely related genera, *Helicobacter* and *Archobactor,* include species previously identified in the *Campylobacter* genus. *Helicobacter pylori* may be differentiated from *Campylobacter* spp by a strong urease activity. It is a cause of gastritis in humans.

AUSTRALIAN SIGNIFICANCE

Campylobacter have been identified in some Australian water supplies, but there have been no reports of infections from drinking water in Australia. No information is available on *Helicobacter* spp in Australian water supplies.

NOTE: Important general information is contained in PART II, Chapter 5

TREATMENT OF DRINKING WATER

Provided the water has low turbidity, standard disinfection procedures are sufficient to prevent the spread of *Campylobacter* in distribution systems.

METHOD OF IDENTIFICATION AND DETECTION

Campylobacter are Gram-negative, slender, comma-shaped rods which show a characteristic corkscrew-like motion which can be easily seen by phase contrast microscopy. They also appear S-shaped and gullwinged when in pairs. They are microaerophilic, requiring a low oxygen tension (3–6%) for growth.

The numbers of thermophilic *Campylobacter* spp in water can be determined by concentration, followed by enrichment, isolation and confirmation (AS4276.19 2014).

HEALTH CONSIDERATIONS

Some of the 14 described species are pathogens for humans and animals (for example *C. jejuni, C. coli, C. fetus)*, while others are considered to be nonpathogenic (for example *C. sputorum, C. concisus)* (Penner 1988). Most the members of the thermophilic group (growing at 42°C) of campylobacters cause enteritis in humans. In Australia, *Campylobacter* are very important bacterial causes of acute gastroenteritis.

Several major outbreaks of *Campylobacter* enteritis have been linked to the ingestion of contaminated food, milk or water.

DERIVATION OF GUIDELINE

Campylobacter in drinking water can cause acute gastroenteritis and should be absent from drinking water supplies.

REFERENCES

AS4276.19 (2014). Australian Standard. Water microbiology: Examination for thermophilic *Campylobacter* spp. – Membrane filtration. Standards Australia, Sydney, NSW.

Penner JL (1988). The genus *Campylobacter*: A decade of progress. *Clinical Reviews in Microbiology,*1, 157–172.

NOTE: Important general information is contained in PART II, Chapter 5

Escherichia coli (E. coli) (pathogenic)

(endorsed 2011)

GUIDELINE

No guideline value has been set for pathogenic* Escherichia coli *and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Escherichia coli (*E. coli*) is used as the primary indicator of faecal contamination of drinking-water supplies, due to its prevalence in the gut of warm-blooded animals. Most *E. coli* are non-pathogenic, normal inhabitants of the gut, but there are several types that are pathogens and have been responsible for waterborne disease outbreaks. Pathogenic *E. coli* are classed into six groups: enterotoxigenic *E. coli* (ETEC), enterohaemorrhagic *E. coli* (EHEC) [also known as Shig-toxin producing *E. coli* STEC], enteroinvasive *E. coli* (EIEC), enterpathogenic *E. coli* (EPEC), enteroadherent-aggregative *E. coli* (EA-AggEC), and diffuse adherent *E. coli* (DAEC) (Nataro and Kaper 1998, Rice and Degnan 2006).

All strains of pathogenic *E. coli* other than EHEC have the human gastrointestinal tract as a primary reservoir; EHEC is predominately found in ruminants. Toxigenic *E. coli* (including O157 and other related strains) are carried by 10-15% of healthy ruminants, including cattle, sheep, goats and deer. In cattle, toxigenic EHEC strains have been found at colonisation rates as high as 60% and typically in the range 10-25% (Nataro and Kaper 1998).

The bacteria may be transmitted to humans by eating raw or undercooked meats, or via foodstuffs or water supplies contaminated with faeces from infected humans or animals. Outbreaks have been attributed to drinking-water supplies as well as recreational water bodies and direct contact with animals. For EHEC, the infectious dose may be as low as 10 to 100 organisms (Teunis *et al*. 2004).

AUSTRALIAN SIGNIFICANCE

There have been no reported outbreaks of waterborne disease associated with pathogenic *E. coli* in Australia. A significant food-borne outbreak of *E. coli* O111:NM occurred in Adelaide in 1995, associated with the consumption of uncooked semi-dry fermented sausages (Cameron *et al*. 1995). Twenty-three cases of Haemolytic Uraemic Syndrome (HUS) and a further 30 cases of bloody diarrhoea were reported.

PREVENTING CONTAMINATION OF DRINKING WATER

Protecting source waters from contamination by human and livestock waste will reduce the potential presence of pathogenic *E. coli*. Like other *E. coli* strains, they are highly sensitive to disinfection. Distribution systems should be protected from ingress of faecal contamination.

NOTE: Important general information is contained in PART II, Chapter 5

METHOD OF IDENTIFICATION AND DETECTION

There is no standard method for detection of pathogenic *E. coli*. They can differ from non-pathogenic *E. coli* in a number of ways that make traditional detection methods unsuitable. For example, some pathogenic groups, such as EIEC, do not ferment lactose; and EHEC do not ferment sorbitol or rhaminose, or contain beta-glucuronidase and they grow poorly at 44.5ºC. Hence defined substrate technologies that rely on beta-glucuronidase activity to detect *E. coli* will not detect all pathogenic strains.

Standard Methods for the Examination of Water and Wastewater (APHA *et al*. 2005) describes different methods for EHEC, EPEC, ETEC and EIEC strains. Each of the methods involves initial isolation using standard liquid or agar-based media used for total coliforms, thermotolerant coliforms or *Salmonella*. Confirmation requires biochemical testing and serotyping. Commercial kits are available for detecting some toxins, including the shiga toxins produced by EHEC.

HEALTH CONSIDERATIONS

The most significant pathogenic *E. coli* for the water industry are the enterohaemorrhagic *E. coli* (EHEC). EHEC comprise more than 100 different serotypes, including O157:H7, which has been responsible for a number of waterborne disease outbreaks.

EHEC, including serogroups such as O111 and O157, are relatively rare strains which produce large quantities of shiga-like (or vero) toxins that can cause illness ranging from mild diarrhoea to haemorrhagic colitis. Haemorrhagic colitis is characterised by blood-stained diarrhoea accompanied by abdominal pain. In addition, EHEC strains can cause HUS, which is characterised by acute renal failure and haemolytic anaemia. The infectious dose may be very low (Teunis *et al*. 2004) and the incubation period ranges from 2 to 8 days.

Several waterborne disease outbreaks have been caused by *E. coli* O157:H7, including the groundwater outbreak in Walkerton, Canada, in May 2000. This outbreak resulted in an estimated 2300 individuals becoming ill, with 65 hospital admissions and 7 deaths. The causal agents were *E. coli* O157:H7 and *Camplyobacter*, attributed to manure contamination of the groundwater supply (Hrudey and Hrudey 2004).

Enteropathogenic *E. coli* (EPEC) have been primarily associated with outbreaks of infantile gastroenteritis, but investigations have shown that they also cause disease in adults (Nataro and Kaper 1998). The pathogenic mechanisms employed by these organisms are not fully understood.

Enteroinvasive *E. coli* (EIEC) produce dysentery by a mechanism similar to *Shigella* spp. These organisms invade the colonic mucosa and cause bloody diarrhoea. This property seems to be restricted to a few O serogroups.

Epidemiological evidence suggests that enterotoxigenic *E. coli* (ETEC) are responsible for most episodes of *E. coli* diarrhoea, particularly in developing countries. ETEC strains can cause a cholera-like syndrome in infants, children and adults, producing a heat-labile enterotoxin (LT) related to cholera enterotoxin, and/or a heat-stable enterotoxin (ST). The action of LT is the same as the cholera toxin. The ability of ETEC to cause disease depends not only on the production of enterotoxin but also upon the organisms' ability to colonise the small intestine. Various colonising factors or adhesins have been described.

DERIVATION OF GUIDELINE

No guideline value is proposed for pathogenic *E. coli* and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures for pathogenic *E. coli,* including prevention of source water contamination by human and animal waste, effective disinfection, and protection of distribution systems from ingress of faecal material. *Escherichia coli* can be used as a reliable indicator for the presence/absence of pathogenic *E. coli*.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

APHA, AWWA, WEF (American Public Health Association, American Water Works Association, Water Environment Federation) (2005). *Standard Methods for the Examination of Water and Wastewater, 21st edition*. Method 9260F. Detection of pathogenic bacteria: Pathogenic *Escherichia coli*. American Public Health Association, Washington DC.

Cameron A, Beers M, Walker C (1995). Community outbreak of hemolytic uremic syndrome attributable to *Escherichia coli* 0111:NM-South Australia. *Morbidity and Mortality Weekly Report,* 44:550-558.

Hrudey SE, Hrudey EJ (2004). *Safe Drinking Water. Lessons From Recent Outbreaks in Affluent Nations*. IWA Publishing, London.

Nataro JP, Kaper JB (1998). Diarrheagenic *Escherichia coli. Clinical Microbiology Reviews,* 11:142-201.

Rice EW, Johnson CH, Reasoner DJ (1996). Detection of *Escherichia coli* O157:H7 in water from coliform enrichment cultures. *Letters in Applied Microbiology,* 23:179-182.

Teunis P, Takumi K, Shinagawa K (2004). Dose response for infection by *Escherichia coli* O157:H7 from outbreak data. *Risk Analysis,* 24:401-407.

NOTE: Important general information is contained in PART II, Chapter 5

Helicobacter pylori

(endorsed 2011)

GUIDELINE

No guideline value has been set for* Helicobacter pylori *in drinking water and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protection of water supplies from human waste is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Helicobacter pylori, originally classified as *Campylobacter pylori*, is a Gram-negative, micro-aerophilic, spiral-shaped, motile bacterium that is able to colonise the stomach. Although not an acidophile, it is able to tolerate the acidic conditions in the stomach. There are at least 14 species of *Helicobacter*, but only *H. pylori* has been identified as a human pathogen. It has a narrow host range and is found primarily in humans and some other primates.

H. pylori has been detected in water. Although it is unlikely to grow in the environment, it has been found to survive for more than 4 days in water (Azevedo *et al*. 2008) and there is evidence that it can be present in biofilms (Park *et al*. 2001). In a US study, *H. pylori* was found in a majority of surface water and shallow groundwater samples; its presence was not correlated with the presence of *Escherichia coli* (Hegarty *et al*. 1999).

The community prevalence of *H. pylori* varies: in developing countries it can exceed 80%, while in developed countries, prevalence is typically below 40% and is decreasing. In developing countries infections have been associated with poor and overcrowded living conditions. Interfamilial clustering is common (Kusters *et al*. 2006).

H. pylori has been detected in saliva, vomit and faeces, but there is evidence that it is sensitive to bile salts, and this will reduce faecal excretion. Person-to-person contact within families has been identified as the most likely source of infection through oral-oral transmission. Faecal-oral transmission is considered possible and consumption of contaminated drinking water has been suggested as a potential source of infection; however, evidence to date is limited to developing countries and further investigation is required to determine whether waterborne transmission occurs (Percival *et al*. 2004).

AUSTRALIAN SIGNIFICANCE

Surveys of seroprevalence have indicated a pattern similar to that found in other developed countries. Antibodies to *H. pylori* have been detected in about 30% of Victorian adults, with seropositivity increasing with age (Robertson *et al*. 2003).

PREVENTING CONTAMINATION OF DRINKING WATER

H. pylori has been detected in human vomit and faeces but the organism does not grow or survive indefinitely in water. Hence protection of drinking water supplies from human waste will minimise the likelihood of contamination. *H. pylori* is sensitive to chlorination (Johnson *et al*. 1997).

NOTE: Important general information is contained in PART II, Chapter 5

METHOD OF IDENTIFICATION AND DETECTION

There is no standard method for isolating and culturing *H. pylori* from water; however, it can be grown on culture media.

HEALTH CONSIDERATIONS

H. pylori is found in the stomach; although most infections are asymptomatic, the organism is associated with chronic gastritis, which may lead to complications such as peptic and duodenal ulcer disease and gastric cancer. The majority of *H. pylori* infections are initiated in childhood and, unless treated, are chronic.

DERIVATION OF GUIDELINE

No guideline value is proposed for *H. pylori* and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures for *H. pylori,* including prevention of contamination by human waste, and effective disinfection. *E. coli* is not a reliable indicator for the presence/absence of *H. pylori*.

REFERENCES

Azevedo NF, Almeida C, Fernandes I, Cerqueira L, Dias S, Keevil CW, Vieira MJ (2008). Survival of gastric and enterohepatic *Helicobacter* spp in water: implications for transmission. *Applied and Environmental Microbiology,* 74:1805-1811.

Hegarty JP, Dowd MT, Baker KH (1999). Occurrence of *Helicobacter pylori* in surface water in the United States. *Journal of Applied Microbiology*, 87:697-701.

Johnson CH, Rice EW, Reasoner DJ (1997). Inactivation of *Helicobacter pylori* by chlorination. *Applied and Environmental Microbiology,* 63:4969-4970.

Kusters JG, van Vliet AHM, Kuipers, EJ (2006). Pathogenesis of *Helicobacter pylori* infection. *Clinical Microbiology Reviews,* 19:449-490.

Park SR, Mackay WG, Reid DC (2001). *Helicobacter* sp. recovered from drinking water biofilm sampled from a water distribution system. *Water Research*, 35:1624-1626.

Percival S, Chalmers R, Embrey M, Hunter P, Sellwood J, Wyn-Jones P (2004). *Microbiology of Waterborne diseases*. Elsevier Academic Press, London UK.

Robertson MS, Cade JF, Savoia HF, Clancy RL (2003). *Helicobacter pylori* infection in the Australian community: current prevalence and lack of association with ABO blood groups. *Internal Medicine Journal,* 33:63-167.

NOTE: Important general information is contained in PART II, Chapter 5

Klebsiella

(endorsed 1996)

GUIDELINE

Coliforms detected in a water supply may include* Klebsiella *spp.

GENERAL DESCRIPTION

Klebsiella spp are inherently environmental organisms that survive and sometimes multiply in suitable waters. They are associated with roots of plants and can grow to high levels on the leaves of vegetables. They are frequently present in raw waters, and can increase to high levels in waters containing pulp mill wastes.

They are also found in the faeces of a significant proportion of healthy people.

K. pneumoniae and *K. oxytoca* are significant opportunistic pathogens in hospitals, but the relationship between infections and drinking water is at best dubious, given the wide distribution of members of this genus in the environment.

The genus is heterogenous and has been difficult to classify. Four species are now included: *K. pneumoniae, K. oxytoca, K. planticola*, and *K. terrigena*. A fifth species, *K. mobilis*, has been proposed, but it remains controversial whether this should be classified in this genus or that of *Enterobacter* (Grimont *et al.* 1991).

As the organisms have similar sensitivity to disinfection to *E. coli* and some bacterial enteric pathogens, their presence in drinking water indicates that disinfection has been inadequate.

AUSTRALIAN SIGNIFICANCE

Klebsiella spp have been detected in Australian drinking water, but there is no evidence that they have caused disease.

METHOD OF DETECTION AND IDENTIFICATION

Klebsiella spp are Gram-negative nonsporing oxidase-negative rod-shaped bacteria, capable of aerobic and facultatively anaerobic growth in the presence of bile salts or other surface active agents with similar growth-inhibiting properties. They are able to ferment lactose, with the production of acid and gas within 48 hours at 35–37°C.

Most *Klebsiella* spp can be quantified in water by either multiple tube dilution, or membrane filtration methods (AS1095.4.1), which are followed by suitable tests for identification of the genus.

HEALTH CONSIDERATIONS

Klebsiella may colonise patients in hospital, being spread mainly by the frequent handling which occurs in intensive care units. Those most at risk are people with impaired defence mechanisms, such as the elderly or the very young, people with burns or excessive wounding, those undergoing immunosuppressive therapy, or those with acquired immune deficiency syndrome (AIDS). From colonisation, invasive infections may occur. On rare occasions *Klebsiella* may cause infections, including destructive pneumonia, in apparently healthy people. These problems appear to be associated with *K. pneumoniae* and *K. oxytoca*.

NOTE: Important general information is contained in PART II, Chapter 5

DERIVATION OF GUIDELINE

No guideline value is established, if used for operational monitoring, numbers should be established on a system specific basis, taking into consideration relevant historical data and an understanding of the characteristics of the system. *Klebsiella* spp form a significant proportion of the organisms identified as coliforms in standard tests for indicator bacteria, and these organisms are thus covered by the guideline for coliforms.

REFERENCES

AS1095.4.1 (1981). Microbiological examination of water for dairy purposes. Microbiological methods for the dairy industry. Australian Standard, Standards Association of Australia, Sydney, NSW.

Grimont F, Grimont PAD and Richard C (1991). The genus *Klebsiella*. In: Balows A, Truper HG, Dworkin

M, Harder W and Schleifer KH, *The prokaryotes*. Springer-Verlag Publishers, 2nd edition, New York, United States, pp 1217–1224 and 1249–1261.

NOTE: Important general information is contained in PART II, Chapter 5

Legionella

(endorsed 1996)

GUIDELINE

No guideline value has been set for* Legionella *in drinking water.

GENERAL DESCRIPTION

The family Legionellaceae contains a single genus, *Legionella*, with 26 currently reported species, of which *L. pneumophila* serogroup 1 is most frequently associated with human disease. Other serogroups of *L. pneumophila* and occasionally other Legionellae have also been reported to cause disease.

Legionella organisms are widespread in natural sources of freshwater and may also be found in soils. They occur commonly in man-made water systems, particularly in hot water and cooling water systems.

Legionella spp appear to infect humans by inhalation, and their presence in drinking water per se seems irrelevant until they are amplified by growing in specific sites under specific conditions (usually thermal enrichment), from which infective aerosols, and droplet nuclei, may be created.

Conditions in cooling towers, spas, warm water systems in buildings, hot water systems operated below 60°C, or 'dead legs' of hot water systems operated at higher temperatures, may favour the growth of *Legionella* organisms. Spraying water in cooling towers or water agitated in spas may then produce aerosols; water from hot water systems can also form aerosols in showers, through nozzle heads, or by splashing in sinks, baths etc.

Legionella organisms can be ingested by the trophozoites of certain amoebae *(Acanthamoeba, Hartmanella, Valkampfi a and Naegleria)* and then grow intracellularly and become incorporated in their cysts. This may explain the difficulty in eradicating *Legionella* organisms from water systems, and it could be a factor in the aetiology of Pontiac fever.

AUSTRALIAN SIGNIFICANCE

Legionella spp have been found in cooling tower waters in many parts of Australia. However, very few *Legionella* organisms have been isolated from drinking waters. No published reports are available on the presence of *L. pneumophila* in drinking waters.

TREATMENT OF DRINKING WATER

Treatment of water with chlorine or chloramines will eliminate these organisms.

METHOD OF IDENTIFICATION AND DETECTION

Legionella spp are Gram-negative, rod-shaped, nonsporing bacteria that require L-cysteine for growth and primary isolation. Cellular fatty acids in *Legionella* organisms are unique for Gram-negative bacilli in that they contain primarily branched chains.

An Australian Standard has been developed for the detection of *Legionella* organisms in water (AS3896 1991).

Isolation of *Legionella* spp from environmental samples may require pre-concentration if numbers are low. Immunofluorescence techniques may also be used to detect *Legionella* spp in the environment.

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

Legionella spp are not known to cause disease by the ingestion of drinking water.

Legionella infections can lead to two types of disease: legionellosis and Pontiac fever. The epidemic form of legionellosis associated with a common infection source is also known as Legionnaires' disease. This is a form of pneumonia with an incubation period usually of 3 to 6 days. Males are more frequently affected than females, and most cases occur in the 40 to 70 year age group. Risk factors include smoking, alcoholism, cancer, diabetes, chronic respiratory or kidney disease, and severe immunosuppression, as in transplant recipients. Ten per cent or more of cases are fatal, even though Legionnaires' disease can be treated effectively by antibiotics such as erythromycin and rifampicin.

Pontiac fever is a milder disease with a high attack rate. The incubation period is 5 hours to 3 days, and symptoms are similar to those of influenza: fever, headache, nausea, vomiting, aching muscles and coughing. No fatal cases have been reported and few outbreaks have been recognised, possibly because the nonspecific nature of the symptoms of the disease hinders its detection.

Infection through human-made water systems such as cooling towers and hot water supplies proceeds through inhalation of aerosols which are small enough to penetrate lungs and be retained by the alveoli – the degree of risk depending on four factors: the density of the bacteria in their source, the extent of aerosol generation, the number of inhaled bacteria, and the susceptibility of the exposed individual.

The number of inhaled bacteria depends on the size of the aerosol generated (<5 µm being most dangerous), the dispersal of the aerosol in the air, and the duration of the exposure. Host defence is important in determining whether exposure to *Legionella* organisms will lead to clinical disease, and differences in susceptibility largely explain the fact that in some cases, high counts of *L. pneumophila* in water systems have been reported in the absence of disease, whereas in other cases similar or lower counts have been associated with epidemics. It is also likely, although not yet adequately proven, that differences in virulence between strains account partly for these observations.

ADVICE ON DISINFECTION

It is not necessary to monitor water systems for *Legionella* spp routinely or to disinfect all environmental sites where Legionellae are detected. The following are generally accepted indications for disinfection:

- sites which are implicated in an outbreak of Legionnaires' disease or Pontiac fever
- hospital wards housing high-risk patients, such as organ transplant units
- buildings in which the water system has not been used for some time and where high numbers are likely to be found.

Vulnerable systems should be designed and maintained in such a way that colonisation by *Legionella* spp is prevented or minimised. The main points to consider are:

- preventing the accumulation of sludge, scale, rust, algae and slime and removing such deposits regularly
- maintaining hot water temperatures permanently above 60°C or at intervals above 70°C, and keeping cold water supplies below 20°C
- selecting materials in contact with water which do not release nutrients that support the growth of *Legionella* spp.

These measures are preferable to, and more effective than, the use of biocides to control *Legionella* organisms in water supplies within buildings; however, biocides are essential to prevent the build-up of microbial slimes in airconditioning systems that use wet evaporative cooling towers. Such systems should be kept clean and well maintained. They should be inspected weekly for fouling and accumulated slime, scale and corrosion, and thoroughly cleaned and disinfected twice yearly. Biocides are best used intermittently in clean systems.

NOTE: Important general information is contained in PART II, Chapter 5

DERIVATION OF GUIDELINE

No specific guideline value can be established for *Legionella* spp. The absence of test mechanisms does not guarantee the total absence of the organism. Warm-water handling systems should always be regarded as being at risk of contamination by *Legionella* spp.

REFERENCE

AS 3896, (1991). Waters – Examination for legionellae. Australian Standard, Standards Association of Australia, Sydney, NSW.

NOTE: Important general information is contained in PART II, Chapter 5

Mycobacterium

(endorsed 2011)

GUIDELINE

No guideline value has been set for* Mycobacterium spp *in drinking water and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Minimising biofilm growth and maintaining cleanliness of distribution systems is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Mycobacteria are saprophytic, aerobic, rod-shaped and acid-fast bacteria. The "typical" species *M. tuberculosis*, *M. bovis*, *M. africanum* and *M. leprae*, which are associated with diseases such as tuberculosis and leprosy, have only human or animal reservoirs and are not transmitted by water. In contrast, the non-tuberculous or "atypical" species of *Mycobacterium* grow slowly in a variety of water environments including biofilms. One of the most commonly occurring atypical species, *M. gordonae*, is known as the "tap water bacillus". Other species associated with water include *M. avium, M. intracellulare, M. kansasii*, *M. marinum*, *M. scrofulaceum*, *M. xenopi* and the (relatively) more rapid growers *M. chelonae* and *M. fortuitum*. The term "*M. avium* complex" has been used to describe a group of pathogenic species including *M. avium* and *M. intracellulare*. However, other atypical mycobacteria are also pathogenic.

Principal routes of infection are inhalation, contact, and ingestion of contaminated water. Infection by various species has been associated with their presence in drinking water supplies. Aerosols generated from showerheads were linked to *M. kansasii* infections in the Czech Republic (Chobot *et al.* 1997). Infection by other species has been associated with contaminated water in spas and ice machines (Lumb *et al.* 2004, Pedley *et al.* 2004).

The ecology of environmental *Mycobacterium* spp is poorly understood; however, there is increasing evidence that they can survive and grow in water distribution systems. In a similar fashion to *Legionella*, resistance to disinfection is enhanced by the ability of mycobacteria to survive intracellularly within amoebae and to grow in biofilms (Pedley *et al.* 2004). In water distribution systems, the presence of mycobacteria has been associated with increased turbidity, dissolved organic carbon and biofilms (Falkinham *et al.* 2001, Pedley *et al.* 2004).

AUSTRALIAN SIGNIFICANCE

An Australian survey in 2000 provided a conservative estimate of 1.8 infections with atypical mycobacteria per 100,000 people (Haverkort 2003). Many of the infections were asymptomatic, with the most common sites of disease being the respiratory tract, soft tissue and the lymphatic system. It is considered that most infections arise from environmental exposure, although sources are typically not identified. Water was a possible source of *M. kansasii* infection in Portland, Victoria (Huang *et al.* 1991) and infections were linked to spa pools in Adelaide (Lumb *et al.* 2004).

PREVENTING CONTAMINATION OF DRINKING WATER

Mycobacterium spp are relatively resistant to treatment and disinfection (LeChevallier 2004) and this is exacerbated by growth in biofilms. Control measures that are designed to minimize biofilm growth, including removal of organic carbon, restriction of residence times of water in distribution systems and maintenance of disinfectant residuals, should reduce growth of these organisms.

NOTE: Important general information is contained in PART II, Chapter 5

METHOD OF IDENTIFICATION AND DETECTION

Mycobacterium spp have a high lipid content in cell walls, which enables them to retain specific dyes in staining procedures that employ an acid wash (i.e., acid-fast). Most of the *Mycobacterium* spp including the "*M. avium* complex" are characterised by slow growth, with optimum generation times ranging from 2 to 48 hours. The rapid-growing species (e.g. *M. chelonae* and *M. fortuitum*) can be detected after 5 days. Concentration and culture methods are available, but the slow growth of mycobacteria adds to the difficulty of growth and identification. DNA and antibody-based methods are also available (Stinear *et al.* 2004).

HEALTH CONSIDERATIONS

Atypical *Mycobacterium* spp can cause a range of diseases involving the skeleton, lymph nodes, skin and soft tissues, as well as the respiratory, gastrointestinal and genitourinary tracts. Manifestations include pulmonary disease, skin ulcers (e.g., Buruli and Bairnsdale ulcers), osteomyelitis and septic arthritis in people with no known predisposing factors. Mycobacteria are a major cause of disseminated infections in people who are immunocompromised and are a common cause of death in people who are HIV-positive.

DERIVATION OF GUIDELINE

No guideline value is proposed for *Mycobacterium* spp and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures designed to minimize biofilm growth, and monitoring the cleanliness of distribution systems.

REFERENCES

Chobot S, Malis J, Sebakova H, Pelikan M, Zatloukal O, Palicka P, Kocurova D (1997). Endemic incidence of infections caused by *Mycobacterium kansasii* in the Karvina district in 1968-1995. *Central European Journal of Public Health*, 5(4):164-173.

Falkinham JO, Norton CD, LeChevallier MW (2001). Factors influencing numbers of *Mycobacterium avium*, *Mycobacterium intracellulare* and other mycobacteria in drinking water distribution systems. *Applied and Environmental Microbiology*, 66:1225-1231.

Havekort F (2003). National atypical mycobacteria survey, 2000. *Communicable Disease Intelligence*, 27(2):180-189.

Huang ZH, Ross BC, Dwyer B (1991). Identification of *Mycobacterium kansasii* by DNA hybridization. *Journal of Clinical Microbiology*, 29:2125-2129.

LeChevallier MW (2004). Control, treatment and disinfection of *Mycobacterium avium* complex in drinking water. In: Pedley S, Bartram J, Rees G, Dufour A, Cutruvo JA (eds), *Pathogenic Mycobacteria in Water: A guide to public health consequences, monitoring and management*. World Health Organization, Geneva, pp 143-168.

Lumb R, Stapledon R, Scroop A, Bond P, Cunliffe D, Goodwin A, Doyle R, Bastian I (2004). Investigation of spa pools associated with lung disorders caused by *Mycobacterium avium* complex in immunocompetent adults. *Applied and Environmental Microbiology*, 70:4906-4910.

Pedley S, Bartram J, Rees G, Dufour A, Cutruvo JA (eds) (2004). *Pathogenic Mycobacteria in Water: A guide to public health consequences, monitoring and management*. World Health Organization, Geneva, Switzerland.

Stinear T, Ford T, Vincent V (2004). Analytical methods for the detection of waterborne and environmental pathogenic mycobacteria. In: Pedley S, Bartram J, Rees G, Dufour A, Cutruvo JA (eds), ***Pathogenic mycobacteria in water:A guide to public health consequences, monitoring and management.*** Geneva, World Health Organization, pp 55-73.

NOTE: Important general information is contained in PART II, Chapter 5

Pseudomonas aeruginosa

(endorsed 1996)

GUIDELINE

No guideline value has been established for* Pseudomonas aeruginosa *in drinking water.

GENERAL DESCRIPTION

Pseudomonas aeruginosa is commonly found in faeces, soil, water and sewage. It cannot be used as an indicator of faecal contamination, as it is not universally present in faeces and sewage, and it may also multiply in an enriched aquatic environment and on the surface of suitable organic materials in contact with water. Its presence, however, can be used to assess the general cleanliness of water distribution systems and the quality of bottled waters.

P. aeruginosa has also been found in various foods.

AUSTRALIAN SIGNIFICANCE

Though *P. aeruginosa* occurs in Australian drinking water supplies, it has only been associated with cases of folliculitis (inflammation of the hair follicles) in health-spa whirlpools.

TREATMENT OF DRINKING WATER

Free available chlorine residuals of 0.2–0.5 mg/L are generally sufficient to control *P. aeruginosa* in water.

METHOD OF IDENTIFICATION AND DETECTION

P. aeruginosa is a member of the family Pseudomonadaceae and is a polarly-flagellated, Gram-negative rod. When grown in suitable media it is capable of producing pigments, the most significant of which are the nonfluorescent phenazine pigments, pyocyanin and fluorescin. Pigment may not be produced by strains of *P. aeruginosa* recovered from clinical specimens and the ability to produce pigment may be lost on subculture. Like other fluorescent pseudomonads in natural waters, *P. aeruginosa* strains produce catalase and oxidase, produce ammonia from arginine, use citrate as the sole source of carbon, and areaerobic.

P. aeruginosa can grow at 41–42°C (AS 1095.4.1.13 1981). The blue-green pigment produced differs from the fluorescent pale green pigment (fluorescin) produced by other species of fluorescent pseudomonads found in water. The organism can also grow anaerobically in stab cultures of nitrate agar.

HEALTH CONSIDERATIONS

P. aeruginosa is a classical opportunistic pathogen. It rarely becomes established in, and even more rarely infects, the intact host but colonises damaged systems, for example burn wounds, the respiratory tract of people with underlying disease, physically damaged eyes etc. From these it may invade the body, causing destructive lesions or septicaemia. Immunosuppressed people, particularly those with low polymorph counts, are at risk. Contaminated 'irrigation' fluids or pharmaceutical agents (e.g. eye drops) delivered to damaged areas have caused severe infection.

NOTE: Important general information is contained in PART II, Chapter 5

While it is clearly undesirable for water supplies to hospitals to have high counts of this organism (or other opportunistic pathogens), a direct association of hospital infections with drinking water sources is yet to be established. High counts of this organism in spa and swimming pool water have been linked with rashes and superficial infections of the outer ear canal (Calderon and Mood 1982, Jones and Bartlett 1985).

DERIVATION OF GUIDELINE

Owing to the widespread occurrence of the organism and its opportunistic pathogenicity, it is difficult to set a guideline for drinking water. However, the presence of the organism in drinking water may indicate a serious deterioration in bacteriological quality, often accompanied by taste, odour and turbidity complaints associated with low rates of flow and increased water temperatures.

REFERENCES

AS 1095.4.1.13, (1981). Examination of water for *Pseudomonas aeruginosa* by membrane filtration. Australian Standard, Microbiological methods for the dairy industry. Standards Association of Australia, Sydney, NSW.

Calderon R and Mood EW (1982). An epidemiological assessment of water quality and 'swimmer's ear'. *Archives of Environmental Health*, 73, 300–305.

Jones F and Bartlett CLR (1985). Infections associated with whirlpools and spas. *Journal of Applied Microbiology* (symposium supplement, microbial aspects of water management), 61s–66s.

NOTE: Important general information is contained in PART II, Chapter 5

Salmonella

(endorsed 1996)

GUIDELINE

Escherichia coli *(or alternatively thermotolerant coliforms) are used to indicate the possible presence of* Salmonella *spp. If explicitly sought,* Salmonella *spp should not be detected. If detected, advice should be sought from the relevant health authority.*

GENERAL DESCRIPTION

Salmonella spp are widely distributed in the environment and gain entry into water systems though faecal contamination from livestock, native animals, drainage waters and incompletely treated waste discharges.

Faecal contamination of water which is inadequately treated or inadequately disinfected is the main cause of waterborne outbreaks of salmonellosis.

AUSTRALIAN SIGNIFICANCE

Salmonella has been isolated from a number of source waters in Australia and occasionally from reticulated waters. However, published associations between the isolation of *Salmonella* from drinking water and health effects in the community are mainly anecdotal.

Most illnesses resulting from *Salmonella* infection are derived from contaminated foodstuffs, e.g. poultry and livestock. Waterborne *Salmonella* spp play only a minor role in causing disease.

TREATMENT OF DRINKING WATER

Treatment by disinfection using chlorine is usually effective against *Salmonella* spp, provided the water has low turbidity.

METHOD OF IDENTIFICATION AND DETECTION

The numbers of Salmonella in water can be determined by concentration followed by enrichment, isolation and confirmation (AS4276.14 2014).

HEALTH CONSIDERATIONS

Salmonella spp, with the exception of those that cause enteric fever in humans (Lloyd 1983), are pathogens of animals, which provide important reservoirs for the infection of humans.

Salmonella Typhi, however, is a specific human pathogen. In particular, *S*. Typhi, *S*. Paratyphi A, and *S*. Paratyphi B are able to invade tissues and cause a septicaemia with high temperature rather than diarrhoea. This is known as enteric fever. In humans, most of the other serovars cause a transient intestinal infection which results in acute gastroenteritis with diarrhoea. Certain serovars are highly pathogenic for humans, while others appear nonpathogenic. Many *Salmonella* infections are symptomless.

Epidemiological and volunteer studies show that the infective dose of *Salmonella* varies considerably. Method of intake, individual host susceptibility, and virulence of the particular strain are important in determining the dose required to produce an infection.

NOTE: Important general information is contained in PART II, Chapter 5

Waterborne outbreaks due to substantial contamination are usually characterised by rapid onset. The majority of cases develop over a period of a few days, and these may be followed by secondary cases. The spatial distribution of infections in major outbreaks is often strongly correlated with the water supply system.

DERIVATION OF GUIDELINE

The presence of faecal indicator bacteria is useful to determine the possible presence of *Salmonella* spp. However, as with many other pathogens, *Salmonella* spp may occasionally be present when indicators are absent, particularly where a supply may have been subject to faecal contamination by amphibians (frogs) and reptiles. It is also important, therefore, to test directly for Salmonellae if contamination is suspected.

The direct effect on the community of noncompliance with the guideline will depend on the *Salmonella* species involved. The numbers of *Salmonella* may be amplified through contamination of foodstuffs.

REFERENCES

AS4276.14, (2014). Australian Standard. Water microbiology: Detection of *Salmonella* spp. Standards Australia, Sydney, NSW.

Lloyd B (1983). *Salmonella*, enteric fever and salmonelloses. In: Feachem RG, Bradley DJ *et al.* (editors). *Health aspects of excreta and wastewater management*. Chichester, John Wiley and Sons, pp 251–286.

NOTE: Important general information is contained in PART II, Chapter 5

Shigella

(endorsed 1996)

GUIDELINE

Escherichia coli *(or alternatively thermotolerant coliforms) are used to indicate the presence of pathogenic* Shigella *spp. If explicitly sought, pathogenic* Shigella *spp should not be detected. If detected, advice should be sought from the relevant health authority.*

GENERAL DESCRIPTION

Bacteria of the genus *Shigella* cause bacillary dysentery. Although shigella infection is not often waterborne, major outbreaks resulting from waterborne transmission have been described. The isolation of *Shigella* spp from drinking water indicates recent human faecal contamination, but this occurs only rarely. This possibly indicates the limitations of the method rather than absence of the organisms, as there is no useful enrichment or selective medium for isolation of these bacteria. Techniques used have been designed for isolation of *Salmonella* spp and are not optimal for *Shigella* spp.

AUSTRALIAN SIGNIFICANCE

No conclusive evidence for the transmission of shigellosis through water supplies in Australia has been reported. The incidence of infection by *Shigella* in Australia is low except in central Australia, and among travellers returning from abroad.

TREATMENT OF DRINKING WATER

Standard disinfection procedures eliminate *Shigella* spp from water, provided that turbidity is low.

METHOD OF IDENTIFICATION AND DETECTION

Shigella spp are Gram-negative, nonsporing, nonmotile rods, growing both aerobically and anaerobically. Metabolism is both respiratory and fermentative; acid, but usually not gas, is produced from glucose but lactose is seldom fermented. Catalase is usually produced, except by *Shigella dysenteriae* type 1, while oxidase is produced by one serotype only. Nitrates are reduced to nitrites (APHA method 9260 E 1992).

Shigella spp are serotyped on the basis of their somatic O antigens. Both group and type antigens are distinguished, group antigenic determinants being common to a number of related types. Serological typing is adequate for all species except *S. sonnei*.

HEALTH CONSIDERATIONS

Shigella spp have a low infective dose and are highly pathogenic for humans. Characteristic bloody diarrhoea results from the invasion of the colonic mucosa by the bacterium; the process is probably highly species-specific. *Shigella* spp have no natural hosts other than the higher primates, and effectively, humans are the only source of infection in the community. Among the enteric bacterial pathogens, Shigellae seem to be the best adapted to cause human disease. Transmission occurs directly between susceptible individuals, and the infectious dose is lower than for other bacteria.

NOTE: Important general information is contained in PART II, Chapter 5

DERIVATION OF GUIDELINE

The isolation of *Shigella* spp from drinking water indicates recent human faecal contamination and, in view of the extreme virulence of the organisms, is of crucial public health significance.

The effect on the community of noncompliance with the guideline will depend on the *Shigella* strain involved, the numbers, and the susceptibility of the population. Cases of shigellosis will almost certainly result.

REFERENCE

APHA Method 9260E, (1992). Detection of pathogenic bacteria: *Shigella*. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington DC.

NOTE: Important general information is contained in PART II, Chapter 5

Vibrio

(endorsed 1996)

GUIDELINE

Escherichia coli *(or alternatively thermotolerant coliforms) are used to indicate the presence of pathogenic* Vibrio *spp. If explicitly sought, pathogenic* Vibrio *spp should not be detected. If detected, advice should be sought from the relevant health authority.*

GENERAL DESCRIPTION

Vibrio species may be waterborne. Cholera (*V. cholerae* O1 and O139), for example, is usually a water-associated disease and numerous such outbreaks have been documented. Food-borne outbreaks, however, are also common and person-to-person transmission may occur under conditions of extreme crowding and poor hygiene. The transmission of cholera has been extensively reviewed, and although water is undoubtedly an important vehicle for transmission, many aspects of the epidemiology of cholera remain open to debate (Miller *et al*. 1985). There is evidence to suggest that in some circumstances, *V. cholerae*, including serotype O1 and O139, may occur naturally in some surface waters.

AUSTRALIAN SIGNIFICANCE

Vibrio spp have been isolated from a number of source waters in Queensland, but not from reticulated waters. There are no published associations between the isolation of Vibrios from source water and health effects in the community.

TREATMENT OF DRINKING WATER

Standard disinfection procedures eliminate *V. cholerae* O1 and O139 (the source of the classic cholera epidemics) from reticulated water, provided turbidity is low.

METHOD OF IDENTIFICATION AND DETECTION

Vibrio spp are nonsporing, slightly curved Gram-negative rods, motile by a single polar flagellum. Their metabolism is both respiratory and fermentative without the production of gas, while their growth is aerobic and facultatively anaerobic. Both catalase and oxidase are formed and nitrates are reduced to nitrites (APHA method 9260H 1992, AS4276.15 2014).

HEALTH CONSIDERATIONS

Vibrio cholerae is a well-defined species frequently found in source waters. While cases of diarrhoea are caused by other types, only the serovars O1 and O139 are associated with the classical cholera symptoms in which a proportion of cases suffer fulminating and severe watery diarrhoea. The O1 serovar has been further divided into 'classical' and 'El Tor' biotypes, the latter distinguished by (*inter alia*) the ability to produce a dialysable, heat-labile haemolysin, active against sheep and goat red blood cells.

When present in large numbers in the intestinal mucosa, *V. cholerae* O1 produces an enterotoxin (cholera toxin) that alters the ionic fluxes across the mucosa with resulting catastrophic loss of water and electrolytes in liquid stools.

NOTE: Important general information is contained in PART II, Chapter 5

Almost all the organisms that are known to cause epidemic cholera are members of the serogroups O1 and O139, though the very similar *V. mimicus* (sucrose nonfermenter) has been isolated from cases of clinical cholera.

DERIVATION OF GUIDELINE

The isolation of *V. cholerae* O1 and O139 from water used for drinking is of major public health importance. However, other serogroups of *V. cholerae* are part of the normal flora of some waters. *V. cholerae* and other pathogenic *Vibrio* spp should be absent from drinking water supplies.

REFERENCES

APHA Method 9260H, (1992). Detection of pathogenic bacteria: *Vibrio cholerae*. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington DC.

AS4276.15 (2014). Australian Standard. Water microbiology: Examination for *Vibrio cholera*. Standards Australia, Sydney, NSW.

Miller CJ, Drasar BJ and Feachem RG (1985). Cholera epidemiology in developed and developing countries: New thoughts on transmission, seasonality and control. *Lancet*, i, 261–263.

NOTE: Important general information is contained in PART II, Chapter 5

Yersinia

(endorsed 1996)

GUIDELINE

Escherichia coli *(or alternatively thermotolerant coliforms) are used to indicate the presence of* Yersinia*. If explicitly sought, pathogenic* Yersinia *spp should not be detected. If they are detected, advice should be sought from the relevant health authority.*

GENERAL DESCRIPTION

The genus *Yersinia* is currently placed in the family Enterobacteriaceae and comprises seven species. Strains of *Y. enterocolitica* can cause gastrointestinal disease if ingested.

A special feature of *Y. enterocolitica* and *Y. enterocolitica*-like organisms is their ability to grow at temperatures as low as 4°C. Accordingly, long survival of these organisms in water habitats can be demonstrated. For example, *Y. enterocolitica* was detected in distilled water for over 18 months at 4°C. Such long survival makes it difficult to find the origin of contamination.

Many domestic and wild animals are considered to be possible reservoirs of *Y. enterocolitica*, due to the high isolation rates of the organism from such sources. Wild animals, particularly hares and foxes, are probably a source of the bacteria, and swine have been implicated as a source of serotypes involved in human infections. The major vehicle of transmission is probably food, especially meat and meat products, milk and dairy products (Lloyd 1983). While *Y. enterocolitica* has also been isolated from a variety of environmental samples, especially from water, the isolated serotypes differ from those associated with human disease.

Ingestion of contaminated food and water is probably the most likely route of transmission of *Y. enterocolitica*. Direct transmission from person to person and from animals to people also occurs, but its relative importance has not been clarified. Further research is needed to define the epidemiological importance of 'environmental' strains of *Y. enterocolitica*.

AUSTRALIAN SIGNIFICANCE

The prevalence of notified cases of *Yersinia* infection varies between states. There has been a marked increase in the number of cases recorded in South Australia in recent years.

TREATMENT OF DRINKING WATER

Standard disinfection procedures are sufficient to avoid transmission of *Yersinia*, provided the water has a low turbidity when treated. Free chlorine in the range required for water disinfection (0.2–0.5 mg/L) for 10 minutes at pH 7 completely eradicates the bacterium. Ozone eradicates the organism after contact with 0.05 mg/L for 1 minute, regardless of pH.

METHOD OF IDENTIFICATION AND DETECTION

Y. enterocolitica is a Gram-negative rod, motile at 25°C but nonmotile in cultures grown at 37°C (APHA Method 9260K 1992).

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

Some serovars of *Y. enterocolitica* are human pathogens. Atypical strains within *Y. enterocolitica,* isolated most frequently from environmental samples, are separated as *Y. enterocolitica*-like organisms. They are not pathogenic for humans and can be subdivided into *Y. intermedia, Y. frederekseni, Y. kristensenii,* and *Y. aldovae* by biochemical means.

Yersiniosis generally presents as an acute gastroenteritis with diarrhoea, but other human diseases caused by *Y. enterocolitica* are also known. *Y. enterocolitica* may be waterborne.

DERIVATION OF GUIDELINE

Water samples yielding *Y. enterocolitica* often show only light coliform contamination. One study indicated that 25% of *Y. enterocolitica*-positive samples were negative for both total and thermotolerant coliforms. Other studies showed a close relation between faecal pollution and *Y. enterocolitica* isolation rates. As it is not possible, at this stage, to determine an infectious dose, *Y. enterocolitica* should be absent from drinking water supplies.

REFERENCES

APHA Method 9260K, (1992). Detection of pathogenic bacteria: *Yersinia enterocolitica*. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington DC.

Lloyd B (1983). *Yersinia* and yersiniosis. In: Feachem RG, Bradley DJ *et al*. (eds). *Sanitation and disease: Health aspects of wastewater management*. Chichester, John Wiley and Sons, pp 327–330.

MICROORGANISMS Protozoa

Acanthamoeba

(endorsed 1996)

GUIDELINE

No guideline value is set for* Acanthamoeba *species in drinking water.

GENERAL DESCRIPTION

Acanthamoeba spp are small free-living amoebae that are common in aquatic environments and are among the predominant protozoa in soil. Several of approximately 20 known species are virulent, causing the cerebral infection granulomatous amoebic encephalitis (GAE), or the corneal infection amoebic keratitis, or both. One or both diseases have occurred in most temperate and tropical regions of the world. *Acanthamoeba* spp may also be significant as host cells for the proliferation and dispersal of *Legionella* species.

The relative importance of water as a source of infection is unknown. The wide distribution of *Acanthamoeba* in the natural environment makes soil, airborne dust and water all likely sources. Delays in the diagnosis of GAE and keratitis cases have made it difficult to investigate possible sources of infection, while the lack of a stable classification of *Acanthamoeba* inhibits identification of individual isolates, including the matching of amoebae from infections with organisms from the environment.

Regular monitoring for *Acanthamoeba* is not appropriate, but these organisms need to be considered when planning the maintenance of eyewash stations that use mains water.

AUSTRALIAN SIGNIFICANCE

Amoebic keratitis has been recorded in New South Wales, Queensland, South Australia, Victoria and Western Australia (e.g. Roussel *et al.* 1985). Currently, four cases of GAE have been diagnosed in Australia (Victoria and Western Australia, e.g. Harwood *et al.* 1988). Data have also been collected on the diversity and density of *Acanthamoeba* species in water and sediments, mainly in South Australia; the organisms are likely to proliferate over a wide temperature range in water where organic carbon levels promote significant bacterial production. Contamination of environments that may become sources of infection (swimming and spa pools, cooling towers etc) cannot be assumed to originate with organisms from the water supply, given the wide distribution of *Acanthamoeba* in the natural environment.

TREATMENT OF DRINKING WATER

Acanthamoeba species are usually less numerous in surface source waters than *Naegleria* species, but often contaminate piped water supplies at a low level, even when chlorine is present. Their cysts are among the most resistant of protozoan cells to oxidative disinfectants, making removal difficult at the levels of disinfectant generally used for drinking water. In any case, control of *Acanthamoeba* may be most important in specialised uses of water: distribution in hospitals, renal dialysis or industrial eye-wash stations.

METHOD OF IDENTIFICATION AND DETECTION

Detection of amoebae, concentrated from water samples, requires relatively simple growth media and standard laboratory incubation facilities. Identification of *Acanthamoeba* species is more specialised. These amoebae are most likely to be significant in specific investigations of sources of infection, when comparison with reference strains would be essential to their identification.

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

Acanthamoeba species are opportunistic pathogens. GAE usually occurs in immunocompromised patients, secondary to infection of another organ (often lungs or subcutaneous tissue). Most cases have been recognised at post-mortem after protracted illness, making any investigation of the circumstances of infection difficult. Amoebic keratitis occurs in two groups of people: those who sustain a corneal lesion before or at the time of infection and who often have outdoor occupations (Roussel *et al.* 1985); and people who wear contact lenses. A specific source of infection has rarely been confirmed, but circumstances suggest that the first group are often infected by cysts from airborne dust or soil, while tap water, used incorrectly to wash lenses, may often be the source for the second group.

DERIVATION OF GUIDELINE

No guideline value is proposed for *Acanthamoeba* species, given the uncertainty about sources of infection, but water authorities should be aware of the direct health significance of these organisms and their possible role in the ecology of *Legionella*.

REFERENCES

Harwood CR, Rich GE, McAleer R and Cherian G (1988). Isolation of *Acanthamoeba* from a cerebral abscess. *Medical Journal of Australia,* 148, 47–49.

Roussel TJ, Badenoch PR, Chandraratnam E and Coster DJ (1985). Acanthamoebic keratitis in a healthy Australian man. *Medical Journal of Australia*, 143, 615–617.

NOTE: Important general information is contained in PART II, Chapter 5

Blastocystis

(endorsed 2011)

GUIDELINE

No guideline value has been set for* Blastocystis *in drinking water and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Although *Blastocystis* was first described in the early 1900s, its pathogenicity and taxonomy remains uncertain. *Blastocystis* has been detected in a range of animal hosts but speciation has not been established. Isolates from humans are generally referred to as *B. hominis,* while isolates from other animals are referred to as *Blastocystis* spp. However, there is some evidence that *Blastocystis* spp may not be host-specific and that animal-to-human transmission is possible. A survey in Malaysia showed that animal handlers and abattoir workers had an increased risk of infection (Rajah *et al.* 1999).

Blastocystis hominis is probably the most common protozoan detected in human faecal samples worldwide. Reported prevalence ranges from 2% to 50%, with the highest rates reported for developing countries with poor environmental hygiene (Stenzel and Boreham 1996). Infection appears to be more common in adults than in children.

While prevalent, the pathogenicity of *B. hominis* is controversial because of the non-specific symptoms. There have been contradictory reports on clinical significance: some reports suggest pathogenicity, but the frequency of asymptomatic infections is very high (Stenzel and Boreham 1996).

Although not confirmed experimentally, faecal-oral transmission is considered to be the main mode of infection. *Blastocystis* cysts have been detected in sewage (Suresh *et al.* 2005). These cysts could be environmentally persistent in a similar fashion to other protozoan cysts, but there are no data on its survival in the environment. The role of drinking water as a source of *Blastocystis* infections has been suggested but not established (Leelayoova *et al.* 2004).

AUSTRALIAN SIGNIFICANCE

An Australian study found no correlation between clinical symptoms and infection with *Blastocystis hominis* (Leder *et al.* 2005). There is no evidence of waterborne transmission in Australia.

PREVENTING CONTAMINATION OF DRINKING WATER

Control measures applied to other infectious protozoa will also reduce risks associated with *Blastocystis.* The likely source of *Blastocystis* is faecal waste, and prevention of contamination of water sources by human and animal waste is a priority. There is little information on the removal and/or inactivation of *Blastocystis* by water and wastewater treatment processes. The morphology of *Blastocystis* varies over a broad range, but faecal cysts can be as small as 3-10 µm in diameter. These should be removed by filtration in a similar manner to *Cryptosporidium* oocysts (4-6 µm in diameter). It has been reported that *Blastocystis* cysts are relatively resistant to chlorine (Suresh *et al.* 2005).

NOTE: Important general information is contained in PART II, Chapter 5

METHOD OF IDENTIFICATION AND DETECTION

A method for concentrating cysts has been reported together with an *in vitro* culture method (Suresh *et al*. 2005). Clinical samples are examined by light microscopy.

HEALTH CONSIDERATIONS

The pathogenicity of *B. hominis* has not been established. A broad range of symptoms has been attributed to *B. hominis*, including watery or loose stools, diarrhoea, abdominal pain, cramps and nausea; however, as mentioned above, the frequency of asymptomatic infections is very high (Stenzel and Boreham 1996).

DERIVATION OF GUIDELINE

No guideline value is proposed for *Blastocystis* and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures, including prevention of contamination by human and animal waste, and (where used) filtration. *Escherichia coli* is not a reliable indicator for the presence/absence of *Blastocystis*.

REFERENCES

Leder K, Hellard ME, Sinclair MI, Fairley CK, Wolfe R (2005). No correlation between clinical symptoms and *Blastocystis hominis* in immunocompetent individuals. *Journal of Gastroenterology and Hepatology,* 20:1390-1394.

Leelayoova S, Ramsin R, Taamasri P, Naaglor T, Thathaisong U, Mungthin M (2004). Evidence of waterborne transmission of *Blastocystis hominis*. *American Journal of Tropical Medicine and Hygiene*, 70:658-662.

Rajah Salim H, Suresh Kumar G, Vellayan S, Mak JW, Khairul Anuar A, Init I, Vennila GD, Saminathan R, Ramakrishnan K (1999). *Blastocystis* in animal handlers. *Parasitology Research*, 85:1032-1033.

Stenzel DJ, Boreham PFL (1996). *Blastocystis hominis* revisited. *Clinical Microbiology Reviews*, 9(4): 563-584.

Suresh K, Smith HV, Tan TC (2005). Viable Blastocystis cysts in Scottish and Malaysian sewage samples. *Applied and Environmental Microbiology*, 71:5619-5620.

NOTE: Important general information is contained in PART II, Chapter 5

Cryptosporidium

(endorsed 2011)

GUIDELINE

No guideline value is set for* Cryptosporidium *due to the lack of a routine method to identify human infectious strains in drinking water. If such a guideline were established, it would be well below 1 organism per litre and would involve testing of impractically large volumes of water.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of* Cryptosporidium *contamination. Protection of catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

Although routine monitoring for* Cryptosporidium *is not recommended, investigative testing may be required in response to events that could increase the risk of contamination (e.g., heavy rainfall, increased turbidity, treatment failure). If* Cryptosporidium *is detected in drinking water, the relevant health authority should be notified immediately. All necessary measures to assess and minimise public health risks should be implemented as soon as possible.

GENERAL DESCRIPTION

In recent years, *Cryptosporidium* has come to be regarded as one of the most important waterborne human pathogens in developed countries. Over 30 outbreaks associated with drinking water have been reported in North America and Britain, with the largest infecting an estimated 403,000 people (Mackenzie *et al.* 1994). Recent research has led to improved methods for testing water for the presence of human infectious species, although such tests remain technically demanding and relatively expensive.

Cryptosporidium is an obligate parasite with a complex life cycle that involves intracellular development in the gut wall, with sexual and asexual reproduction. Thick-walled oocysts, shed in faeces are responsible for transmission. Concentrations of oocysts as high as 14,000 per litre in raw sewage and 5,800 per litre in surface water have been reported (Madore *et al.* 1987). Oocysts are robust and can survive for weeks to months in fresh water under cold conditions (King and Monis 2007).

There are a number of species of *Cryptosporidium,* with *C. hominis* and *C. parvum* identified as the main causes of disease (cryptosporidiosis) in humans. *C. hominis* appears to be confined to human hosts, while the *C. parvum* strains that infect humans also occur in cattle and sheep. *C. parvum* infections are particularly common in young animals, and it has been reported that infected calves can excrete up to 10 billion oocysts in one day. Waterborne outbreaks of cryptosporidiosis have been attributed to inadequate or faulty treatment and contamination by human or livestock (particularly cattle) waste. *C. hominis* and *C. parvum* can be distinguished from one another and from other *Cryptosporidium* species by a number of genotyping methods. Infectivity tests using cell culture techniques have also been developed.

Consumption of contaminated drinking water is only one of several mechanisms by which transmission (faecal-oral) can occur. Recreational waters, including swimming pools, are an important source of cryptosporidiosis and direct contact with a human carrier is also a common route of transmission. Transmission of *Cryptosporidium* can also occur by contact with infected farm animals, and occasionally through contaminated food.

NOTE: Important general information is contained in PART II, Chapter 5

AUSTRALIAN SIGNIFICANCE

The most publicised incident of drinking-water contamination in Australia occurred in July-September 1998 in Sydney. High numbers of *Cryptosporidium* and *Giardia* (see Fact Sheet) were reported in treated water, and boil-water notices were issued for three million residents. No increase in illness was detected in association with the contamination, despite increased epidemiological surveillance. The incident highlighted the lack of a method (at that time) to determine whether detected organisms were infective for humans.

Cryptosporidiosis is a notifiable condition in all Australian states and territories. A case-control study of sporadic cases in Adelaide and Melbourne from 1998 to 2001 indicated that person-to-person contact and public swimming pools were the most common risk factors for infection (Robertson *et al.* 2002). Outbreaks associated with contaminated swimming pools have occurred in several Australian states. In South Australia, a relatively large number of illnesses were recorded in 1990-91 but no source was identified (Weinstein *et al.* 1993). The only known outbreak of illness associated with drinking water occurred in Victoria, when a mixture of infections due to *Cryptosporidium* and *Giardia* followed contamination of a private water supply by overflow from a septic tank (Lester 1992).

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be implemented to minimise the risk of contamination by *Cryptosporidium*. Protection of water catchments from contamination by human and animal wastes is a priority. Water from unprotected catchments is likely to be subject to contamination by *Cryptosporidium* and treatment, including effective filtration, will be required to remove these organisms and ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

Water catchments should be surveyed for potential sources of contamination, and source water should be subject to investigative and event-based testing for *Cryptosporidium*, to:

- assess risk factors for contamination;
- provide a basis for catchment management to reduce these risks; and
- determine the level of water treatment required.

It has been reported that increases in turbidity associated with rainfall events may signal increased numbers of *Cryptosporidium* (Atherholt *et al.* 1998), although Australian data indicate that there is no uniform relationship that is applicable across different catchments (CRC 2007).

Groundwater from confined aquifers or from depth should be free from contamination by *Cryptosporidium*. However, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Cryptosporidium oocysts are extremely resistant to chlorine and will not be killed by concentrations that can be practically used in drinking water. Other chemical disinfectants such as ozone are more effective (Bouchier 1998). More recently it has been shown that ultraviolet (UV) light disinfection is effective against *Cryptosporidium*, and this technology is being increasingly adopted for drinking-water treatment. The United States Environmental Protection Agency (USEPA) has developed detailed guidance on the application of UV for inactivation of *Cryptosporidium,* including the relationship between dose and log reduction as well as aspects of plant design, process validation and operational issues (USEPA 2006). Ensuring that UV light doses are at all times greater than specified values provides a practical means of ensuring that *Cryptosporidium* oocysts are inactivated and are not a threat to public health.

NOTE: Important general information is contained in PART II, Chapter 5

Due to their small size (4-6 µm), *Cryptosporidium* oocysts can challenge removal by granular media-based filtration processes; however, well designed and operated systems can provide a high level of removal. Membrane filtration processes that provide a direct physical barrier can represent a viable alternative for the effective removal of *Cryptosporidium*. Where *Cryptosporidium* oocysts are suspected or known to be present in the raw water, the design and operation of water treatment plants should be carefully examined to ensure that required performance is achieved and maintained. For granular media-based systems, there should be particular attention to ensuring that coagulation/flocculation is optimal, turbidity is monitored from all filters, backwash water is handled appropriately, and increases in turbidity are minimised during filter start-up and operation, to avoid sudden flow surges (see Badenoch 1995, Bouchier 1998). A turbidity limit of 0.2 NTU or less for effluent from individual filters has been shown to provide optimal removal of *Cryptosporidium* and other classes of enteric pathogens (Xagoraraki *et al*. 2004).

The performance of filtration plants should be monitored continuously and treated water of a constant quality should be produced, irrespective of the quality of the raw water.

Filtration plants should be operated by trained and skilled personnel. Failure of water treatment processes, including failure to meet specified targets for turbidity (or particle counts), represents a potential risk of oocyst contamination of the drinking-water supply.

The integrity of distribution systems should be maintained. Storages for treated water should be roofed, backflow prevention measures should be applied, and faults and burst mains should be repaired in a way that will prevent contamination.

METHOD OF IDENTIFICATION AND DETECTION

There are a number of different methods for isolating *Cryptosporidium* oocysts from water. Most quantitative methods involve concentration of relatively large volumes of water and fluorescent staining of the concentrated material. Many methods are adapted from USEPA Method 1623 (USEPA 2005), which uses immunomagnetic beads to separate oocysts from contaminating debris. The use of any method should include exacting quality control procedures and determination of recovery efficiencies. The *Cryptosporidium* Proficiency Testing program operated by the National Association of Testing Authorities, Australia sets out performance requirements for laboratories undertaking *Cryptosporidium* testing, but does not mandate a specific test methodology.

Different species and strains of *Cryptosporidium* may be distinguished using a range of genotyping techniques, which have been developed by different laboratories. An international trial has been undertaken to compare the utility of six techniques to characterise *C. hominis* and *C. parvum* isolates (Chalmers *et al*. 2005).

In the past, techniques such as excystation and vital dye staining have been used to assess oocyst viability or infectivity; however it is now recognised that these methods are unreliable and may overestimate human infection risks. Cell culture techniques provide more accurate measures of human infectivity although correlation can be variable (Rochelle et al. 2002; Schets et al. 2005). Cell culture may be coupled with a number of molecular techniques using the polymerase chain reaction to amplify genetic markers. This provides more rapid and sensitive tests for infectivity (Di Giovanni *et al*. 1999, Keegan *et al*. 2003).

Although there have been considerable advances in methodology, identification of human infectious *Cryptosporidium* oocysts in water remains a technically demanding and relatively expensive process.

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

Infection of normally healthy people by *Cryptosporidium* can result in self-limiting diarrhoea that usually resolves within a week but can last for a month or more. Illness varies according to age and immune status. Chronic infections may occur, and can be life threatening, in some severe immunodeficiency conditions (advanced stages of AIDS, severe combined immune deficiency, specific T-cell deficiency) (Bouchier 1998, Chief Medical Officer 1999). People with severe immunodeficiency conditions may also be more susceptible to infection by species other than *C. hominis* and *C. parvum*.

DERIVATION OF GUIDELINE

No guideline value is proposed for *Cryptosporidium* and routine monitoring of distribution systems, including outlets from water treatment plants, is not recommended because of the lack of a reliable and efficient method to identify human infectious *C. parvum*. In addition, current risk assessment models suggest that impractically large volumes of water would need to be tested to provide a meaningful indication of health risk (Haas *et al.* 1996). Human dose response trials have indicated that infectivity for different isolates of *Cryptosporidium* varies widely (Chappell *et al.* 2006).

Investigative testing of drinking water may be required if *Cryptosporidium* contamination is suspected. This could occur in association with a major rainfall event, which could lead to a marked decrease in water quality and a marked increase in the numbers of *Cryptosporidium* in source water, sub-optimal operation of treatment processes, a breakdown in treatment plant operations, or a fault within the distribution system. Monitoring may also be required in response to suspected waterborne cryptosporidiosis.

When an incident of concern leads to the testing of distribution systems for *Cryptosporidium*, the relevant health authority should be notified immediately. If *Cryptosporidium* is detected in finished water, the relevant health authority should again be notified immediately.

Comprehensive protocols should be developed by water and health agencies to deal with detections of *Cryptosporidium* in drinking water and should describe approaches for interpreting the health and operational significance. In responding to incidents or detection, the health authority may choose to do so in consultation with the water authority and/or an expert panel. Credible public communication is essential. Responses could include: further sampling to confirm the presence and source of the organisms; testing for the presence of infectious organisms and the specific presence of *C. hominis* or *C. parvum*; issuing advice, including boil-water notices, to the public; and enhanced surveillance to detect possible increases in community cryptosporidiosis.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

Atherholt TB, LeChevallier MW, Norton WD, Rosen JS (1998). Effect of rainfall on *Giardia* and Crypto. *Journal of the American Waterworks Association*, 90(9):66-80.

Badenoch J (1995). *Cryptosporidium* in water supplies. Second Report of the Group of Experts. Department of the Environment, Department of Health, HMSO.

Bouchier I (1998). *Cryptosporidium* in water supplies. Third Report of the Group of Experts. Department of the Environment Transport and the Regions, Department of Health, HMSO.

Chalmers RM, Ferguson C, Cacciò S, Gasser RB, Abs EL, Osta YG, Heijnen L, Xiao L, Elwin K, Hadfield S, Sinclair M, Stevens M (2005). Direct comparison of selected methods for genetic categorisation of *Cryptosporidium parvum* and *Cryptosporidium hominis* species. *International Journal for Parasitology* 35(4):397-410.

Chappell CL, Okhuysen PC, Langer-Curry R, Widmer G, Akiyoshi DE, Tanriverdi S, Tzipori S (2006). *Cryptosporidium hominis*: Experimental challenge in healthy adults. *American Journal of Tropical Medicine and Hygiene,* 75(5):851-857.

Chief Medical Officer's Update 23 August (1999). *Cryptosporidium* in water: clarification of the advice to the immunocompromised. Department of Health, London.

CRC (2007). *Source Water Quality Assessment and the Management of Pathogens in Surface Catchments and Aquifers*. Cooperative Research Centre for Water Quality and Treatment Research Report 29.

Di Giovanni GD, Hashemi FH, Shaw NJ, Abrams FA, LeChevallier MW, Abbaszadegan M (1999). Detection of infectious *Cryptosporidium parvum* oocysts in surface and filter backwash water samples by immunomagnetic separation and integrated cell culture-PCR. *Applied and Environmental Microbiology,* 65(8):3427-32.

Haas CN, Crockett C, Rose JB, Gerba CP, Fazil A (1996). Risk assessment of *Cryptosporidium parvum* oocysts in drinking water. *Journal of the American Waterworks Association*, 88(9):131-136.

Keegan AR, Fanok S, Monis PT, Saint CP (2003). Cell culture-Taqman PCR assay for evaluation of *Cryptosporidium parvum* disinfection. *Applied and Environmental Microbiology,* 69(5):2025-11.

King BJ, Monis PT (2007). Critical processes affecting *Cryptosporidium* oocyst survival in the environment. *Parasitology,* 134(3):309-23.

Lester R (1992). A mixed outbreak of cryptosporidiosis and giardiasis. Update. *Quarterly Bulletin of Infectious Diseases*, Health Department Victoria, 1:14-15.

MacKenzie WR, Hoxie NJ, Proctor ME, Gradus MS, Blair KA, Paterson DE, Kazmicrczak JJ, Addiss DG, Fox KR, Rose JB, Davis JP (1994). A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public water supply. *New England Journal of Medicine*, 331:161-167.

Madore MS, Rose JB, Gerba CP, Arrowood MJ, Sterling CR (1987). Occurrence of *Cryptosporidium* oocysts in sewage effluents and selected surface waters. *Journal of Parasitology*, 73: 702-5.

Robertson B, Sinclair MI, Forbes AB, Veitch M, Kirk M, Cunliffe D, Willis J, Fairley CK (2002). Case-control studies of sporadic cryptosporidiosis in Melbourne and Adelaide, Australia. *Epidemiology and Infection*, 128:419-431.

Rochelle PA, Marshall MM, Mead JR, Johnson AM, Korich DG, Rosen JS and De Leon R (2002) Comparison of in vitro cell culture and a mouse assay for measuring infectivity of *Cryptosporidium parvum*. *Applied and Environmental Microbiology,* 68:3809-3817.

NOTE: Important general information is contained in PART II, Chapter 5

Schets FM, Engels GB, During M and Roda Husman AM (2005) Detection of infectious Cryptosporidium oocysts by cell culture immunofluorescence assay: applicability to environmental samples. *Applied and Environmental Microbiology,* 71:6793-6798.

USEPA (United States Environmental Protection Agency) (2005). Method 1623: *Cryptosporidium* and *Giardia* in Water by Filtration/IMS/FA..

USEPA (United States Environmental Protection Agency) (2006). *Ultraviolet Disinfection Guidance Manual.* EPA 815-D-03-007, USEPA, Cincinatti, USA.

Weinstein P, Macaitis M, Walker C, Cameron S (1993). Cryptosporidial diarrhoea in South Australia: an exploratory case-control study of risk factors for transmission. *Medical Journal of Australia*, 158:117-119.

Xagoraraki, I., Harrington GW, Assavasilavasukul P. and Standridge JH. (2004). Removal of emerging waterborne pathogens and pathogen indicators by pilot-scale conventional treatment. *Journal of the American Water Works Association*, 96(5):102-113.

Cyclospora

(endorsed 2011)

GUIDELINE

No guideline value has been set for* Cyclospora *in drinking water and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Cyclospora cayetanensis is a single-cell, obligate, intracellular, coccidian protozoan parasite. It produces thick-walled oocysts of 8-10 µm in diameter that are excreted in the faeces of infected individuals. Humans are the only host identified. The unsporulated oocysts are excreted in faecal material and undergo sporulation, which is complete in 7-12 days, depending on environmental conditions. Only the sporulated oocysts are infectious.

C. cayetanensis is an emerging pathogen, having first been identified as a human pathogen in the 1970s and 1980s (Herwaldt 2000). The organism increased in profile following several food-borne outbreaks in the United States in the 1990s. It has also been associated with waterborne outbreaks (Herwaldt 2000).

C. cayetanensis is transmitted by the faecal-oral route. Person-to-person transmission is virtually impossible, because the oocysts must sporulate outside the host to become infectious. The primary sources of infection are contaminated water and food.

The origin of organisms in food-borne outbreaks has generally not been established, but contaminated water has been implicated in several cases. Drinking water has also been implicated as a cause of outbreaks. The first report was among staff of a hospital in Chicago, USA, in 1990 (Herwaldt 2000, WHO 2002). The infections were associated with drinking tap water that had possibly been contaminated with stagnant water from a rooftop storage reservoir. In Nepal, infection of 12 of 14 soldiers was linked to drinking water consisting of a mixture of river and municipal water (Herwaldt 2000, WHO 2002). In 2005 a waterborne outbreak of cryptosporidiosis and cyclosporiasis was reported in Turkey (Aksoy *et al.* 2007).

There is limited information on the prevalence of *Cyclospora* in water environments. The cysts could be environmentally persistent in a similar fashion to other protozoan cysts but there are no data on survival in the environment. *Cyclospora* has been detected in sewage and water sources (Herwaldt 2000, WHO 2002, Dowd *et al.* 2003).

AUSTRALIAN SIGNIFICANCE

There have been no outbreaks recorded in Australia. Sporadic cases in Australia are typically associated with travellers' diarrhoea (Pinge-Suttor *et al.* 2004). This is consistent with the cause of sporadic cases in regions such as North America and Europe (Sterling and Ortega 1999, WHO 2002)

PREVENTING CONTAMINATION OF DRINKING WATER

Control measures applied to other infectious protozoa will also reduce risks associated with *Cyclospora*. The likely source of *Cyclospora* is human faecal waste, and prevention of contamination of water sources is a priority. There is little information on the removal and/or inactivation of *Cyclospora* by water and wastewater treatment processes. Oocysts are intermediate in size between *Cryptosporidium* oocysts

NOTE: Important general information is contained in PART II, Chapter 5

and *Giardia* cysts, and removal by physical processes such as filtration should be similar. There is little information on sensitivity to disinfection.

METHOD OF IDENTIFICATION AND DETECTION

No specific method has been developed for concentration of *Cyclospora* from water. However, methods developed for *Cryptosporidium* and *Giardia* should be effective. Identification is based on light microscopy and acid-fast staining of smears. Autofluoresence of *Cyclospora* has also been used to facilitate detection by microscopy (Herwaldt 2000, WHO 2002).

HEALTH CONSIDERATIONS

Sporozoites are released from the oocysts when ingested and they penetrate epithelial cells in the small intestine of susceptible individuals. Clinical symptoms of cyclosporiasis include watery diarrhoea, abdominal cramping, weight loss, anorexia, myalgia and occasionally vomiting and/or fever. Relapsing illness often occurs.

DERIVATION OF GUIDELINE

No guideline value is proposed for *Cyclospora*. The focus should be on monitoring of control measures, including prevention of contamination by human waste and (where used) filtration. *Escherichia coli* is not a reliable indicator for the presence/absence of *Cyclospora*.

REFERENCES

Aksoy U, Akisu C, Sahin S, Usluca S, Yalcin G, Kuralay F (2007). First reported waterborne outbreak of cryptosporidiosis with *Cyclospora* co-infection in Turkey. *Eurosurveillance* 12(7). (www.eurosurveillance.org/ViewArticle.aspx?ArticleId=3142, accessed August 2008).

Dowd SE, John D, Eliopolus J, Gerba CP, Naranjo J, Klein R, López B, de Mejía M, Mendoza CE, Pepper IL (2003). Confirmed detection of *Cyclospora cayetanensis*, *Encephalitozoon intestinalis* and *Cryptosporidium parvum* in water used for drinking. *Journal of Water and Health*, 1:117-123.

Herwaldt BL (2000). *Cyclospora cayetanensis*: A review, focusing on the outbreaks of cyclosporiasis in the 1990s. *Clinical Infectious Diseases*, 31:1040-1057.

Pinge-Suttor V, Douglas C, Wettstein A (2004). Cyclospra infection masquerading as coeliac disease. *Medical Journal of Australia*, 180(6):295-296.

Sterling CR, Ortega YR (1999). *Cyclospora*: an enigma worth unravelling. *Emerging Infectious Diseases,* 5(1):48-53.

WHO (World Health Organization) (2002). Protozoan parasites (*Cryptosporidium*, *Giardia*, *Cyclospora*). In: *Guidelines for Drinking-Water Quality,* 2nd edition, Addendum: Microbiological agents in drinking water. World Health Organization, Geneva, pp. 70-118.

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NOTE: Important general information is contained in PART II, Chapter 5

Giardia

(endorsed 2011)

GUIDELINE

No guideline value is set for* Giardia *due to the lack of a routine method to identify human infectious strains in drinking water. If such a guideline were established, it would be well below 1 organism per litre and would involve testing of impractically large volumes of water.

A multiple barrier approach operating from catchment to tap is recommended to minimise the risk of* Giardia *contamination. Protection of catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

Although routine monitoring for* Giardia *is not recommended, investigative testing may be required in response to events that could increase the risk of contamination (e.g., heavy rainfall, increased turbidity, treatment failure). If* Giardia *is detected in drinking water, the relevant health authority should be notified immediately. All necessary measures to assess and minimise public health risks should be implemented as soon as possible.

GENERAL DESCRIPTION

Although known as a human parasite for 200 years, *Giardia* has been regarded seriously as an agent of disease only since the 1960s. It has been identified as an important waterborne pathogen, and linked to many outbreaks of illness associated with drinking water, particularly in North America. Although the importance of this organism has been established, there are large gaps in knowledge about it, and there are no tests for identifying the presence of human infectious species in water.

Giardia has a relatively simple life cycle involving two stages: a flagellate that multiplies in the intestine, and an infective thick-walled cyst that is shed intermittently but in large numbers in faeces. Concentrations of cysts as high as 88,000 per litre in raw sewage and 240 per litre in surface water have been reported (Wallis *et al.* 1996). *Giardia* is typically present in larger numbers in Australian sewage than *Cryptsoporidium*. Cysts are robust and can survive for weeks to months in fresh water.

There are a number of species of *Giardia,* but human infections (giardiasis) are usually assigned to one, *G. intestinalis* (= *G. lamblia* and *G. duodenalis*). *G. intestinalis* infections have been reported from domestic and wild animals, but the host range of human infectious species is uncertain. Although substantial advances have been made in the sampling and counting of cysts, there are currently no established methods to identify human infectious organisms in water. Waterborne outbreaks of giardiasis have generally been linked to consumption of untreated or unfiltered surface water and contamination with human waste.

Consumption of contaminated drinking water is only one of several mechanisms by which transmission (faecal-oral) can occur. Recreational waters, including swimming pools, are also emerging as an important source of giardiasis. However, excluding outbreaks, by far the most likely route of transmission is by direct contact with a human carrier. Transmission of *Giardia* can also occur by contact with infected animals and occasionally through contaminated food.

NOTE: Important general information is contained in PART II, Chapter 5

AUSTRALIAN SIGNIFICANCE

Outbreaks of giardiasis in Australia often involve close communal groups. In day-care centres, for instance, as many as 20% of children may carry *Giardia* without symptoms (Grimmond *et al*. 1988). Infection is endemic and is significant among children and adults in the wider community, and sources of this infection are difficult to identify. Giardiasis is notifiable in some states and territories.

The most publicised incident of drinking-water contamination in Australia occurred from July to September 1998 in Sydney. High numbers of *Cryptosporidium* (see Fact Sheet) and *Giardia* were reported for treated water, and boil-water notices were issued for three million residents. No increase in illness was detected in association with the contamination despite increased epidemiological surveillance. An epidemiological study in Queensland showed no correlation between infection and source of drinking water, point-of-use treatment (boiling or filtration) or recreational contact with water (Boreham and Phillips 1986). Another study identified contact with septic tank waste or contaminated soil as a possible mechanism of infection (Boreham *et al*. 1981). An outbreak of illness associated with drinking water was reported in Victoria when mixed infections due to *Cryptosporidium* and *Giardia* followed contamination of a private water supply by overflow from a septic tank (Lester 1992).

PREVENTING CONTAMINATION OF DRINKING WATER

Approaches applied to *Cryptosporidium* will usually be as or more effective against *Giardia*.

A multiple barrier approach operating from catchment to tap should be implemented to minimise the risk of contamination by *Giardia*. Protection of water catchments from contamination by human and animal wastes is a priority. Water from unprotected catchments is likely to be subject to contamination by *Giardia* and treatment, including effective filtration or enhanced disinfection, will be required to remove these organisms and ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

Water catchments should be surveyed for potential sources of contamination, and source water should be subject to investigative and event-based testing for *Giardia*, to:

- assess risk factors for contamination;
- provide a basis for catchment management to reduce these risks; and
- determine the level of water treatment required.

It has been reported that increases in turbidity associated with rainfall events may signal increased numbers of *Giardia* (Atherholt *et al*. 1998), although Australian data indicate that there is no uniform relationship that is applicable across different catchments (CRC 2007).

Groundwater from confined aquifers or from depth should be free from contamination by *Giardia*. However, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Giardia cysts are more resistant than enteric bacteria to chlorine, but not as resistant as *Cryptosporidium*. The time required for a 90% (1 log 10) kill at 1 mg/L free chlorine is of the order of 25-35 minutes. Other chemical disinfectants such as ozone are more effective. The United States Environmental Protection Agency (USEPA) has published C.*t* tables specifying chlorine concentrations (C) and contact times (*t*) required to inactivate *Giardia* cysts over a range of conditions (see USEPA 1999). In addition, C.*t* tables have been provided for chloramines, ozone and chlorine dioxide. More recently it has been shown that ultraviolet (UV) light disinfection is effective against *Giardia* and this technology is being increasingly adopted for drinking-water treatment. The USEPA has developed detailed guidance on the application of UV for inactivation of *Giardia*, including the relationship between dose and log reduction as well as

NOTE: Important general information is contained in PART II, Chapter 5

aspects of plant design, process validation and operational issues (USEPA 2006). Ensuring that UV light doses or chemical disinfectant concentrations and contact times are at all times greater than specified values provides a practical means of ensuring that *Giardia* cysts are inactivated and are not a threat to public health.

The USEPA National Primary Drinking Water Standards prescribe comprehensive treatment (including filtration and disinfection) of most surface waters to protect drinking water from contamination by *Giardia*. Operational procedures in water treatment plants should be carefully examined where *Giardia* cysts are suspected or known to be present in the raw water, to ensure that optimum removal is achieved and maintained. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Storages for treated water should be roofed, backflow prevention measures should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

METHOD OF IDENTIFICATION AND DETECTION

USEPA has published a standard method for *Giardia* applying the same principles as those used for isolating *Cryptosporidium* oocysts from water (USEPA 2005). Testing for *Giardia* should include exacting quality control procedures and determination of recovery efficiencies.

In the past, techniques such as excystation and vital dye staining have been used to assess cyst viability or infectivity; however it is now recognised that these methods are unreliable and may overestimate human infection risks. There are currently no established methods to identify human infectious organisms in water.

HEALTH CONSIDERATIONS

Infection by *Giardia* may reduce absorption of nutrients and cause diarrhoea. In most cases, illness is self limiting, but in some cases chronic infection with intermittent diarrhoea can occur. Specific treatments are available.

DERIVATION OF GUIDELINE

No guideline value is proposed for *Giardia*, and routine monitoring of distribution systems, including outlets from water treatment plants, is not recommended because of the lack of a reliable and efficient method to identify human infectious organisms. In addition, current risk assessment models suggest that impractically large volumes of water would need to be tested to provide a meaningful indication of health risk.

Investigative testing of drinking water may be required if *Giardia* contamination is suspected. This could occur in association with a major rainfall event, which could lead to a marked decrease in water quality and a marked increase in the numbers of *Giardia* in source water, suboptimal operation of treatment processes, a breakdown in treatment plant operations, or a fault within the distribution system. Monitoring may also be required in response to suspected waterborne giardiasis.

When an incident of concern leads to the testing of distribution systems for *Giardia,* the relevant health authority should be notified immediately. If *Giardia* is detected in finished water, the relevant health authority should again be notified immediately.

Comprehensive protocols should be developed by water and health agencies to deal with detections of *Giardia* in drinking water and should describe approaches for interpreting the health and operational significance. In responding to incidents or detection, the health authority may choose to do so in consultation with the water authority and/or an expert panel. Credible public communication is essential.

NOTE: Important general information is contained in PART II, Chapter 5

Responses could include further sampling to confirm the presence and source of the organisms; testing for the presence of viable organisms; increased disinfection; the issuing of advice, including boil-water notices, to the public; and enhanced surveillance to detect possible increases in community giardiasis.

REFERENCES

Atherholt TB, LeChevallier MW, Norton WD, Rosen JS (1998). Effect of rainfall on *Giardia* and *Crypto*. *Journal of the American Waterworks Association*, 90(9):66-80.

Boreham PFL, Phillips RE (1986). Giardiasis in Mount Isa, north west Queensland. *Medical Journal of Australia*, 144:524-528.

Boreham PFL, Dondey J, Walker R (1981). Giardiasis among children in the city of Logan, South East Queensland. *Australian Paediatric Journal*, 17:209-212.

CRC (2007). *Source Water Quality Assessment and the Management of Pathogens in Surface Catchments and Aquifers*. Cooperative Research Centre for Water Quality and Treatment Research Report 29.

Grimmond TR, Radford AJ, Brownridge T, Farshid A, Harris C, Turton P, Wordsworth K (1988). *Giardia* carriage in aboriginal and non-aboriginal children attending urban day-care centres in South Australia. *Australian Paediatric Journal*, 24:304-305.

Lester R (1992). A mixed outbreak of cryptosporidiosis and giardiasis. Update. *Quarterly Bulletin of Infectious Diseases*, Health Department Victoria, 1:14-15.

USEPA (United States Environmental Protection Agency) (1999). *Disinfection Profiling and Benchmarking Guidance Manual.* EPA 815-R-99-013, USEPA, Cincinatti, USA.

USEPA (United States Environmental Protection Agency) (2005) Method 1623: *Cryptosporidium* and *Giardia* in Water by Filtration/IMS/FA.

USEPA (United States Environmental Protection Agency) (2006). *Ultraviolet Disinfection Guidance Manual.* EPA 815-D-03-007, USEPA, Cincinatti, USA.

Wallis PM, Erlandsen SL, Isaac-Renton JL, Olson ME, Robertson WJ, van Keulen H (1996). Prevalence of *Giardia* cysts and *Cryptosporidium* oocysts and characterization of Giardia spp isolated from drinking water in Canada. *Applied and Environmental Microbiology*, 62:2789-2.

NOTE: Important general information is contained in PART II, Chapter 5

Naegleria fowleri

(endorsed 1996)

GUIDELINE

No guideline value is set for* Naegleria fowleri *in drinking water, but an 'action level' is recommended for water supplies likely to be contaminated. If the organism is detected, advice should be sought from the relevant health authority.

GENERAL DESCRIPTION

Naegleria fowleri is a free-living, thermophilic amoeboflagellate which causes the waterborne disease primary amoebic meningoencephalitis (PAM). This rare but fatal condition has followed use of water for swimming, or domestic bathing. The organism occurs naturally in freshwater of suitable temperature, feeding on bacteria. Its occurrence is only indirectly related to human activity, inasmuch as such activity may modify temperatures or promote bacterial production. PAM has been reported from many countries, usually associated with thermally polluted environments, geothermal water or heated swimming pools. *N. fowleri* is almost exclusively aquatic, and water is the only known source of infection. Numerous nonvirulent *Naegleria* species are known in Australia.

AUSTRALIAN SIGNIFICANCE

PAM cases have been recorded from South Australia, Western Australia, Queensland and New South Wales; *Naegleria fowleri* has been detected in water in each of these states and in the Northern Territory. Australia is the only country where *N. fowleri* has been detected in public water supplies (Dorsch *et al.* 1983). Most of the available data on the density of *N. fowleri* in water relates to water supplies in South Australia (including the highest reported densities). In temperate Australia, significant seasonal cycles of density occur, from below one organism per litre to hundreds or thousands per litre in poorly disinfected water (Robinson and Christy 1984). *N. fowleri* detected at water temperatures below 18°C is likely to be present as cysts, which are not infectious, but which may seed a suitable environment.

TREATMENT OF DRINKING WATER

Free chlorine or chloramines at 0.5 mg/L or higher will control *N. fowleri*, provided that the disinfectant persists throughout the water supply system. Chloramination is the preferred process in extensive rural water supplies, owing to its stability (Robinson and Christy 1984).

METHOD OF IDENTIFICATION AND DETECTION

Detection of amoebae, concentrated from water samples, requires relatively simple growth media and standard laboratory incubation facilities. Identification of *Naegleria* species, particularly recognition of *N. fowleri,* is more specialised. In routine or investigative analyses, presence of any thermophilic amoebae (able to grow at 42°C or above) is evidence that conditions are suitable for *N. fowleri* should it be introduced. If samples include any *Naegleria*, remedial action should be taken immediately without waiting for specific identification.

Prospective studies directed at water supplies that are susceptible to colonisation by *N. fowleri* can be valuable since the mortality rate of infection is so high, but universal monitoring is not appropriate.

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

N. fowleri is apparently an accidental pathogen. Its unusual route of infection (intranasal) means that PAM is associated with bathing rather than with ingesting water. Treatment is rarely effective, even in cases diagnosed early, and PAM is almost invariably fatal. Most Australian victims have been children (Dorsch *et al* 1983).

Recreational bathing presents the greatest risk of infection by *N. fowleri*, owing to the nature and duration of exposure, but domestic bathing can also lead to infection (Dorsch *et al.* 1983). Public water supplies can therefore be important as sources of contamination of public or private swimming pools, or as direct sources of infection. The infectious dose is unknown, but the frequency of infections has been low, even in populations that seem to have been widely and repeatedly exposed. A density of around 100 organisms per litre may present an immediate risk of infection but rapid density changes of this free-living organism can occur (Robinson and Christy 1984).

DERIVATION OF GUIDELINE

No guideline value is proposed for *N. fowleri*, given its irregular distribution in Australia and its dependence on relatively high water temperatures. However, any water supply that seasonally exceeds 30°C or that continually exceeds 25°C can support the growth of *N. fowleri*. In such cases, a periodic prospective study would be valuable, but regular monitoring is not warranted unless *N. fowleri* is detected. A density of 2 organisms per litre (or detection in a 500 mL sample) is an appropriate threshold for action, given the rapid density changes that can occur. Other thermophilic *Naegleria* can be useful 'proxy' organisms for *N. fowleri*, allowing early remedial action.

REFERENCES

Dorsch MM, Cameron AS and Robinson BS (1983). The epidemiology and control of primary amoebic meningoencephalitis with particular reference to South Australia. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 77, 372–377.

Robinson BS and Christy PE (1984). Disinfection of water for control of amoebae. *Water*, 11, 21–24.

NOTE: Important general information is contained in PART II, Chapter 5

MICROORGANISMS Cyanobacteria and their toxins

Cyanobacteria and their toxins

(endorsed 2011)

GENERAL DESCRIPTION

Cyanobacteria are Gram-negative bacteria that contain chlorophyll. The presence of chlorophyll allows them to undertaken photosynthesis, hence their historical identification as blue-green algae. Their primary health significance is that many species of cyanobacteria produce toxins, which can be either contained intracellularly, or expressed extracellularly and therefore present in the surrounding water.

Monitoring programs developed by water suppliers are typically based in the first instance on detecting the presence of cyanobacteria. These tests are generally more sensitive and less expensive than tests for toxins. Testing for toxicity is generally not implemented until cell numbers are in the 1000s per mL.

The two main types of toxin are:

- **Cyclic peptides (microcystins and nodularin).** Microcystins cause damage to the liver and are possibly carcinogenic. Nodularin has an identical mode of action to microcystin in animals and is considered to present at least the same risk to human health as microcystin.
- **Alkaloids (neurotoxins and cylindrospermopsin).** Neurotoxins produced by cyanobacteria include anatoxin a, anatoxin a-s and the saxitoxins. Only saxitoxins have been detected in Australian waters.

Cylindrospermopsin is a general cytotoxin that blocks protein synthesis. The major pathological effects are damage to the liver, kidneys, lungs, heart, stomach, adrenal glands, the vascular system, and the lymphatic system. Acute clinical symptoms are kidney and liver failure.

The table below lists potentially toxin-producing cyanobacteria found in freshwater.

Cyanobacteria	**Toxin(s) produced**
Cylindrospermopsis raciborskii, *Aphanizomenon ovalisporum*, *Aphanizomenon flos-aquae*, *Raphidiopsis curvata*, and *Umezakia natans*.	Cylindrospermopsins *Cylindrospermopsis raciborskii* is the most common producer of cylindrospermopsins in Australian water sources.
Microcystis, Anabaena, Planktothrix (*Oscillatoria*), *Nostoc, Anabaenopsis* and *Radiocystis*	Microcystins *Microcystis sp.* and *M. aeruginosa* in particular is the most common producer of microcystins in Australian water sources.
Nodularia spumigena	Nodularins
Anabaena, *Lyngbya, Oscillatoria*, *Cylindrospermopsis*, *Cylindrospermum*, and *Aphanizomenon*	Saxitoxins, anatoxin-a and anatoxin-a(s) In Australia neurotoxin production appears to be limited to saxitoxins from *Anabaena circinalis* (Velzeboer *et al.* 2000).

Being Gram-negative bacteria, all cyanobacteria contain lipopolysaccharides in their cell walls. The lipopolysaccharides can have inflammatory and irritative effects if contact exposure occurs, and have also been proposed as a causative agent for allergic reactions observed in sensitive individuals (skin rashes, eye irritations etc). A study in Australia indicated that about 12% of the population could be sensitive (NHMRC 2008).

The rest of this section contains detailed Fact Sheets on the specific toxins produced by cyanobacteria in Australian freshwaters, arranged in alphabetical order by toxin.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

National Health and Medical Research Council (2008). *Guidelines for Managing Risks in Recreational Water.* Commonwealth of Australia.

Velzeboer RMA, Baker PD, Rositano J (1998). *Characterisation of Saxitoxins Produced by the Cyanobacterial Genus* Anabaena *in Australia*. UWRAA Research Report No. 135. Urban Water Research Association of Australia, Melbourne.

NOTE: Important general information is contained in PART II, Chapter 5

Cylindrospermopsin

(endorsed 2011)

GUIDELINE

Due to the lack of adequate data, no guideline value is set for concentrations of cylindrospermopsin. However given the known toxicity, the relevant health authority should be notified immediately if blooms of* Cylindrospermopsis raciborskii *or other producers of cylindrospermopsin are detected in sources of drinking water.

GENERAL DESCRIPTION

Cylindrospermopsin is tricyclic guanidine alkaloid cytotoxin with a molecular weight of 415, produced by the freshwater cyanobacteria *Cylindrospermopsis raciborskii*, *Aphanizomenon ovalisporum*, *Aphanizomenon flos-aquae*, *Raphidiopsis curvata*, and *Umezakia natans*. There are two structural variants identified in addition to the most common form. It was first characterised and named from an Australian isolate of *C. raciborskii* (Ohtani *et al.* 1992). Subsequently cylindrospermopsin has been detected in two other cyanobacteria: *Umezakia natans* in Japan (Harada *et al.* 1994, Terao *et al.* 1994), and *Aphanizomenon ovalisporum* in Israel (Banker *et al.* 1997) and Australia (Shaw *et al.* 1999). In pure form, cylindrospermopsin is predominantly a hepatotoxin, although extracts of *C. raciborskii* administered to mice induced pathological symptoms in the kidneys, spleen, thymus, heart and eye. Two other structural variants of cylindrospermopsin have been identified (Banker *et al.* 2000, Norris *et al.* 1999).

The production of toxins and therefore the presence of toxicity in individual populations of some cyanobacterial species is known to be variable (Chorus and Bartram 1999 Chapter 3). In the case of *C. raciborskii*, however, the majority of the strains tested so far in Australia appear to produce cylindrospermopsin. It is therefore likely that most blooms of *C. raciborskii* will have some degree of toxicity. The natural breakdown of cylindrospermopsin in natural waters is influenced by a number of factors including previous occurrence. Degradation occurs within a few weeks in surface water subject to repeated occurrence, but is far slower in waters with no recorded history of occurrence (Chiswell *et al.* 1999, Smith *et al.* 2007).

AUSTRALIAN SIGNIFICANCE

Cylindrospermopsin is believed to have been the causative agent in the Palm Island "mystery disease" poisoning incident in Queensland in 1979, in which 148 people were hospitalised (Byth 1980). It was subsequently shown that water from Solomon Dam on Palm Island contained blooms of toxic *C. raciborskii* (Hawkins *et al.* 1985). *C. raciborskii* has been found in many water supply reservoirs in northern, central and southern Queensland. Although *C. raciborskii* and *A. ovalisporum* are both considered to be predominantly tropical/sub-tropical in terms of habitat, with most Australian blooms occurring in Queensland, *C. raciborskii* also occurs in the Murray-Darling River system (Baker and Humpage 1994). In recent years there has been increasing evidence of detection in the River Murray and *C. raciborskii* was detected in the major blooms that affected several hundred kilometres of the River Murray on the border between New South Wales and Victoria in 2009 and 2010 (NSW Office of Water 2009, MDBA 2010). *C. raciborskii* is not a scum-forming organism, but forms dense bands below the water surface in stratified lakes, while *A. ovalisporum* may form thick brown surface scums (Shaw *et al.* 1999). Although no reports of human poisoning attributable to cylindrospermopsin have appeared since the Palm Island incident, recent cattle deaths in Queensland are attributed to this toxin (Saker *et al.* 1999).

NOTE: Important general information is contained in PART II, Chapter 5

TREATMENT OF DRINKING WATER

The first line of defence against cyanobacteria is catchment management to minimise nutrient inputs to source waters. Source water management techniques to control cyanobacterial growth include maintaining flow in regulated rivers; water mixing techniques, both to eliminate stratification and reduce nutrient release from sediments in reservoirs; and the use of algicides in dedicated water supply storages. Destratification has been used to attempt to reduce bloom intensities of *C. raciborskii* in reservoirs in Queensland, however it has not yet been possible to determine the efficacy of this treatment method. Caution is necessary in using algicides if a bloom has developed because these agents will disrupt cells and liberate intracellular cylindrospermopsin that could otherwise be removed by cell removal, as noted below. Once these intracellular toxins are released they are more difficult to manage. The extracellular release of cylindrospermopsin will increase when a developed bloom declines and algal cells lyse, reinforcing the need to prevent blooms as far as possible. Algicide use should be in accordance with local environment and chemical registration regulations. Where multiple intakes are available, withdrawing water selectively from different depths can minimise the intake of high accumulations of cyanobacterial cells at the surface.

The right combination of water treatment processes can be highly effective in removing both cyanobacterial cells and cylindrospermopsin. In contrast to other cyanotoxins, a high proportion of cylindrospermopsin in actively growing *C. raciborskii* blooms may be found free in the water, i.e. non cell-bound (Chiswell *et al*. 1999). Only the proportion of cylindrospermopsin that is cell-bound can be removed by coagulation and filtration in a conventional treatment plant (Chorus and Bartram 1999 Chapter 9). It should be noted that using oxidants such as chlorine or ozone to treat water containing cyanobacterial cells, while killing the cells, will also result in the release of free toxin; therefore pre-chlorination or pre-ozonation are not recommended without a subsequent step to remove dissolved toxins.

Cylindrospermopsin is readily oxidised by a range of oxidants including ozone and chlorine. Adequate contact time and pH control are needed to ensure optimum removal of these compounds, and this will be more difficult to achieve in the presence of whole cells (Chorus and Bartram 1999 Chapter 9). Cylindrospermopsin is also adsorbed from solution by both granular activated carbon and powdered activated carbon. Because powdered activated carbon may be a more practical option for intermittent or emergency use, it is important to seek advice and carefully select the most appropriate type for toxin removal, as carbons vary significantly in performance for different compounds. Boiling is not effective for the destruction of cylindrospermopsin. Based on current knowledge, the recommended best-practice treatment scheme for removal of cylindrospermopsin would include conventional treatment (coagulation/filtration) followed by an adsorption or oxidation step.

METHOD OF IDENTIFICATION AND DETECTION

Animal bioassays (mouse tests) have been used to determine the toxicity of *C. raciborskii* (Falconer *et al*. 1999, Seawright *et al*. 1999). These tests provide a definitive indication of toxicity, although they cannot be used for precise quantification of compounds in water. Instrumental analytical techniques are available for determining the presence of cylindrospermopsin in water, including high performance liquid chromatography (HPLC) with UV detection (Harada *et al*. 1994) and HPLC-Mass Spectrometry (Eaglesham *et al*. 1999).

Cyanobacteria are detected by light microscopy, identified using morphological characteristics, and counted per standard volume of water (Hotzel and Croome 1999). Practical keys for the identification are provided in Baker and Fabbro (2002).

NOTE: Important general information is contained in PART II, Chapter 5

HEALTH CONSIDERATIONS

The major pathological effects of cylindrospermopsin are damage to the liver, kidneys, lungs, heart, stomach, adrenal glands, the vascular system, and the lymphatic system (Falconer and Humpage 2006). Liver damage is likely to be severe and dose dependent. Cylindrospermopsin is a slow-acting toxin, commonly requiring between 5 and 7 days to produce maximum toxic effect in experimental animals. It has been shown that the LD_{50} for cylindrospermopsin decreases greatly between 24 hours and 5 days. The 24-hour LD_{50} for mice (i.p.) is 2 mg/kg, while the 5-6 day i.p. LD_{50} is 0.2 mg/kg (Ohtani *et al.* 1992, Terao 1994). The 5-day LD_{50} for mice by oral administration is approximately 6 mg/kg (Seawright *et al.* 1999).

A range of sub-chronic oral toxicity studies have demonstrated that the most sensitive responses in mice are in increased liver, kidney, and testis weights, together with a decrease in urine protein content. These studies can be used to derive the maximum no observed adverse effect level (NOAEL) for oral cell extracts of *C. raciborskii* or purified cylindrospermopsin.

The most detailed sub-chronic oral dosing study was undertaken by Humpage and Falconer (2003). In two trials, mice were exposed to various doses of cylindrospermopsin for 10-11 weeks. Body weights were significantly increased at low doses (30 and 60 µg kg^{-1} d^{-1}) and decreased at high doses (432 and 657 µg kg^{-1} d^{-1}). Liver and kidney weights were significantly increased at doses of 240 µg kg^{-1} d^{-1} and 60 µg kg^{-1} d^{-1}, respectively. Serum bilirubin levels were significantly increased and bile acids significantly decreased at doses of 216 µg kg^{-1} d^{-1} and greater. Serum cholesterol levels were significantly increased at 30 and 60 µg kg^{-1} d^{-1}. Urine total protein was significantly decreased at doses above 60 µg kg^{-1} d^{-1}. In contrast to previous findings from studies using higher doses and/or shorter exposure times, the kidney rather than the liver appeared to be the more sensitive organ in this trial, although both were clearly affected.

Shaw *et al.* (2000) calculated a NOAEL of 50 µg kg^{-1} d^{-1} based on fatty infiltration of the liver. Reisner *et al.* (2004) reported increased serum cholesterol, changes in red blood cell membrane cholesterol, distortion of cell morphology and increased hematocrits in a 21- day oral exposure trial with male mice drinking water containing 600 µg L^{-1} cylindrospermopsin (estimated daily cylindrospermopsin intake of 66 µg kg^{-1}). Sukenik *et al.* (2006) reported impacts from giving mice increasing concentrations of cylindrospermopsin in the drinking water over 42 weeks, ranging from initial doses of approximately 10 µg kg^{-1} d^{-1} to 58 µg kg^{-1} d^{-1} Relative kidney weights were significantly increased at 20 weeks whereas liver weights were significantly increased only at 42 weeks. Effects on cholesterol, red cell morphology and hematocrit were observed.

The variations in experimental design of these studies makes an interpretation of dose-response difficult, but overall these findings are in agreement with the 11 week trial described above, both in terms of adverse effects and dose levels producing them.

A NOAEL based on these studies above is estimated to be around 30 µg/kg body weight per day.

NOTE: Important general information is contained in PART II, Chapter 5

DERIVATION OF HEALTH ALERT

The strength of data is insufficient to establish a guideline value. However, an initial health alert can be estimated using the results described above.

$$0.945\ \mu g/L \text{ rounded to } 1\ \mu g/L = \frac{30\ \mu g/kg \text{ bodyweight per day} \times 70\ kg \times 0.9}{2\ L/day \times 1000}$$

where:

- 30 μg/kg body weight per day is the No Observed Adverse Effect Level (NOAEL) from the 10 and 11 week ingestion studies with cylindrospermopsin in mice based on liver histopathology, body organ weight and serum enzyme level changes (Humpage and Falconer 2003);
- 70kg is the average weight of an adult;
- 0.9 is the proportion of total daily intake attributed to the consumption of water;
- 2 L/day is the average amount of water consumed by an adult;
- 1000 is the safety factor derived from extrapolation of an animal study to humans (10 for interspecies variability, 10 for intraspecies variability and 10 for limitations in the database, related particularly to the lack of data on chronic toxicity, genotoxicity and carcinogenicity).

In situations where *C. raciborskii* occurs in drinking water supplies and toxin monitoring data are unavailable, cell numbers may be used to provide a preliminary orientation to the potential hazard to public health. This type of assessment has been used for *Microcystis aeruginosa*. However, this is slightly more problematic for *C. raciborskii,* as, at any time, a significant proportion of cylindrospermopsin toxin may be extracellular and free in solution, and this cannot be accounted for in the assessment of cell counts from the raw water.

Nevertheless, in the case of *C. raciborskii*, local knowledge and experience can allow the development of local thresholds. For example, in Queensland both water and health authorities have extensive monitoring data and experience for a range of populations of toxic *C. raciborskii* (G McGregor, personal communication). Data from 23 reservoirs indicated that most of the cylindrospermopsin was found in the cell-bound fraction and that concentrations of approximately 1 μg/L were associated with cell concentrations in the range of 15,000-20,000 cells/mL, which is equivalent to a biovolume of 0.6-0.8 mm^3/L (based on a mean cell volume of *C. raciborskii* 42 μm^3). These numbers are indicative only and for health risk assessment, total toxin determination, including both intracellular and extracellular concentrations, is required.

NOTIFICATION PROCEDURE

It is recommended that a notification procedure be developed by water and health authorities. A tiered framework should be considered. Initial notification to health authorities could be provided when numbers of *C. raciborskii* reach 30% of the density equivalent to 1 μg/L cylindrospermopsin (4,500 cells/mL; biovolume 0.2 mm^3/L), while an alert could be provided when cell numbers are equivalent to 1 μg/L cylindrospermopsin (15,000 cells/mL; biovolume 0.6 mm^3/L). For cylindrospermopsin-producing species other than *C. raciborskii*, notifications and alerts should be based on biovolumes.

In all cases, cell numbers should only be used as preliminary signals and as triggers for toxin testing to enable assessment of potential health risks.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

Baker PD, Fabbro LD (2002). *A Guide to the Identification of Common Blue-Green Algae (Cyanoprokaryotes) in Australian Freshwaters*. CRCFE Identification Guide No. 25. Cooperative Research Centre for Freshwater Ecology, Albury.

Baker P, Humpage AR (1994). Toxicity associated with commonly occurring cyanobacteria in surface waters of the Murray-Darling Basin, Australia. *Australian Journal of Marine and Freshwater Research*, 45:773-786.

Banker R, Teltsch B, Sukenik A, Carmeli S (2000). 7-epicylindrospermopsin, a toxic minor metabolite of the cyanobacterium Aphanizomenon ovalisporum from Lake Kinneret, Israel. *Journal of Natural Products* 63(3):387-389.

Banker R, Carmeli S, Hadas O, Teltsch B, Porat R, Sukenik A (1997). Identification of cylindrospermopsin in the cyanobacterium *Aphanizomenon ovalisporum* (Cyanophyceae) isolated from Lake Kinneret, Israel. *Journal of Phycology*, 33:613-616.

Byth S (1980). Palm Island mystery disease. *Medical Journal of Australia*, 2:40-42.

Chiswell RK, Shaw GR, Eaglesham GK, Smith MJ, Norris RL, Seawright AA, Moore MR (1999). Stability of cylindrospermopsin, the toxin from the cyanobacterium, *Cylindrospermopsis raciborskii*: Effect of pH, temperature and sunlight on decomposition. *Environmental Toxicology*, 14:155-161.

Chorus I, Bartram J (eds) (1999). *Toxic Cyanobacteria in Water. A guide to their public health consequences, monitoring and management*. E&FN Spon, London.

Eaglesham GK, Norris RL, Shaw GR, Smith MJ, Chiswell RK, Davis BD, Neville GR, Seawright AA, Moore MR (1999). Use of HPLC-MS/MS to monitor cylindrospermopsin, a blue-green algal toxin, for public health purposes. *Environmental Toxicology*, 14:151-154.

Falconer IR, Hardy SJ, Humpage AR, Froscio SM, Toizer GJ, Hawkins PR (1999). Hepatic and renal toxicity of the blue-green alga (cyanobacterium) *Cylindrospermopsis raciborskii* in male Swiss albino mice. *Environmental Toxicology*, 14:143-150.

Falconer IR, Humpage AR (2006). Cyanobacterial (blue-green algal) toxins in water supplies: Cylindrospermopsin. *Environmental Toxicology*, 21:299-304.

Harada K, Ikuko O, Iwamoto K, Suzuki M, Watanabe M, Watanabe M, Terao K (1994). Isolation of cylindrospermopsin from a cyanobacterium *Umezakia natans* and its screening method. *Toxicon*, 32:73-84.

Hawkins PR, Runnegar MTC, Jackson ARB, Falconer IR (1985). Severe hepatotoxicity caused by the tropical cyanobacterium (blue-green alga), *Cylindrospermopsis raciborskii* (Woloszynska) Seenaya and Subba Raju isolated from a domestic water supply reservoir. *Applied Environmental Microbiology*, 50:1292-1295.

Hotzel G, Croome R (1999). *A Phytoplankton Methods Manual for Australian Freshwaters*. LWRRDC Occasional Paper 22/99, Land and Water Resources Research and Development Corporation, Canberra.

Humpage AR, Falconer IR (2003). Oral toxicity of the cyanobacterial toxin cylindrospermopsin in male Swiss albino mice: Determination of no observed adverse effect level for deriving a drinking water guideline value. *Environmental Toxicology*, 18:94–103.

MDBA (Murray Darling Basin Authority) (2010) River Murray algal blooms. http://www.mdba.gov.au/media_centre/media_releases/river-murray-algal-blooms (accessed April 2010)

NOTE: Important general information is contained in PART II, Chapter 5

New South Wales Office of Water (2009).The Murray River algal bloom. New South Wales Department of Climate Change and Water, Sydney, Australia.

Norris RL, Eaglesham GK, Pierens G, Shaw GR, Smith MJ, Chiswell RK, Seawright AA, Moore MR (1999). Deoxycylindrospermopsin, an analog of cylindrospermopsin from *Cylindrospermopsis raciborskii*. *Environmental Toxicology*, 14:163-165.

Ohtani I, Moore RE, Runnegar MTC (1992). Cylindrospermopsin: A potent hepatotoxin from the blue-green alga *Cylindrospermopsis raciborskii*. *Journal of the American Chemistry Society*, 114:7941-7942.

Reisner M, Carmeli S, Werman M, Sukenik A. (2004). The cyanobacterial toxin cylindrospermopsin inhibits pyrimidine nucleotide synthesis and alters cholesterol distribution in mice. *Toxicological Sciences*, 82(2):620-627.

Saker ML, Thomas AD, Norton JH (1999). Cattle mortality attributed to the toxic cyanobacterium *Cylindrospermopsis raciborskii* in an outback region of North Queensland. *Environmental Toxicology*, 14:179-182.

Seawright AA, Nolan CC, Shaw GR, Chiswell RK, Norris RL, Moore MR, Smith MJ (1999). The oral toxicity for mice of the tropical cyanobacterium *Cylindrospermopsis raciborskii* (Woloszynska). *Environmental Toxicology*, 14:135-142.

Shaw GR, Sukenik A, Livne A, Chiswell RK, Smith MJ, Seawright AA, Norris RL, Eaglesham GK, Moore MR (1999). Blooms of the cylindrospermopsin containing cyanobacterium, *Aphanizomenon ovalisporum* (Forti) in newly constructed lakes, Queensland, Australia. *Environmental Toxicology*, 14:167-177.

Shaw GR, Seawright AA, Moore MR. (2000). Toxicology and human health implications of the cyanobacterial toxin cylindrospermopsin. In: *Mycotoxins and Phycotoxins in Perspective at the Turn of the Millennium*. Proceedings of the Xth International IUPAC symposium on Mycotoxins and Phycotoxins, 21-25 May 2000, Guaruja, Brazil. IUPAC and AOAC International, pp 435-443.

Smith MJ, Shaw GR, Eaglesham GK, Ho L, Brookes JD (2007). Elucidating the factors influencing the biodegradation of cylindrospermopsin in drinking water sources. *Environmental Toxicology* 23:413-421.

Sukenik A, Reisner M, Carmeli S, Werman M (2006). Oral toxicity of the cyanobacterial toxin cylindrospermospin in mice: Long-term exposure to low doses. *Environmental Toxicology*, 21:575-582.

Terao K, Ohmori S, Igarashi K, Ohtani I, Watanabe MG, Harada K-I, Ito E, Watanabe M (1994). Electron microscopic studies on experimental poisoning in mice induced by cylindrospermopsin isolated from blue-green alga *Umezakia natans*. *Toxicon*, 32:833-843.

NOTE: Important general information is contained in PART II, Chapter 5

Microcystins

(endorsed 2011)

GUIDELINE

Based on health considerations, the concentration of total microcystins in drinking water should not exceed 1.3 μg/L expressed as microcystin-LR toxicity equivalents (TE).

GENERAL DESCRIPTION

Microcystins are a large group of hepatotoxic peptides that are produced by a range of cyanobacteria. They were first characterised in the early 1980s and named after the cyanobacterium *Microcystis aeruginosa*, from which they were initially isolated. This group of cyanotoxins includes over 90 structural variants of cyclic heptapeptides (consisting of seven amino acids in a ring structure), with molecular weights in the range 800-1100 (Chorus and Bartram 1999 Chapter 3). Within this common structure there can be modifications in all seven amino acids, but the most frequent variations are substitution of L-amino acids at positions 2 and 4. The nomenclature of microcystins is based on the variable amino acids in position 2 and 4; for example, using the amino acid abbreviations for the L-amino acids, microcystin-LR possesses leucine (L) in position 2 and arginine (R) in position 4. Microcystin-LR is the best characterised and one of the most toxic variants of microcystin. Most of the structural variants are highly toxic within a narrow range, although some non-toxic variants have been identified (Chorus and Bartram 1999 Chapter 3).

Microcystins are most commonly produced by species of the genus *Microcystis*. They have, however, been shown to be produced by species of the planktonic genera *Anabaena (Dolichospermum)*[1], *Planktothrix* (*Oscillatoria*), *Nostoc, Anabaenopsis* and *Radiocystis* and also by a terrestrial (soil) species *Haphalosiphon hibernicus,* indicating the potential for their widespread occurrence in the environment. Within these genera and species, there can be both toxigenic (toxin-producing) and non-toxigenic genotypes. Nevertheless, the majority of human and animal microcystin-related poisonings worldwide are associated with the presence of *Microcystis*.

The toxicity of individual populations of *M. aeruginosa* is variable, and one extensive survey of the toxicity across the Murray-Darling Basin indicated that 56% of field samples tested were hepatotoxic (Baker and Humpage, 1994). A natural population may consist of a mixture of toxic and non-toxic genotypes, and this is believed to explain why population toxicity may vary over time and between samples (Chorus and Bartram 1999 Chapter 3). Environmental factors are regarded as the driving force determining these processes.

These cyanotoxins are largely water-soluble and are therefore, with a few exceptions, unable easily to penetrate biological membranes. Microcystins are thought to enter the bloodstream of mammals from the intestine, predominantly through the bile acid transport system. The absorbed toxins are then concentrated into liver cells, and cause hepatoenteritis. Microcystins are extremely stable chemically and remain potent even after boiling; however they are biodegraded by a range of aquatic bacteria found naturally in lakes and rivers. The half-lives for breakdown of microcystins in natural water have been shown to range from 5 to 20 days (Jones *et al.* 1994).

1 A change of nomenclature has been proposed for *Anabaena* to *Dolichospermum* (Wacklin et al, 2009). Both names are cited due to common usage of *Anabaena* and recognising that references cited use the name *Anabaena*

NOTE: Important general information is contained in PART II, Chapter 5

AUSTRALIAN SIGNIFICANCE

Microcystins are the most significant drinking water quality issue in relation to cyanobacterial blooms in south-eastern Australia. In Australia, they are produced predominantly by *Microcystis aeruginosa*, but can occasionally be produced by *Anabaena (Dolichospermum)* spp, although this appears to be rare.

The growth of cyanobacteria and blooms are favoured by nutrient enrichment (largely phosphorus but also nitrogen), combined with warm temperatures and calm, stable water conditions, such as those occurring in slow-flowing rivers and thermally stratified lakes.

The water supply problems associated with cyanobacteria include offensive tastes and odours and the production of toxins.

TREATMENT OF DRINKING WATER

The first line of defence against cyanobacteria is catchment management to minimise nutrient inputs to source waters. Source water management techniques to control cyanobacterial growth include maintaining flow in regulated rivers; water mixing techniques, both to eliminate stratification and reduce nutrient release from sediments in reservoirs; and the use of algicides in dedicated water supply storages. Caution is necessary in using algicides if a bloom has developed because these agents will disrupt cells and liberate microcystins which largely occur as intracellular toxins that could otherwise be removed by cell removal as noted below. Once intracellular toxins are released they are much more difficult to manage. Microcystins will eventually be released into the water phase when a developed bloom declines and algal cells lyse, reinforcing the need to prevent blooms as far as possible. Algicide use should be in accordance with local environment and chemical registration regulations. Where multiple intakes are available, withdrawing water selectively from different depths can minimise the intake of high accumulations of cyanobacterial cells at the surface.

Water treatment processes can be highly effective in removing both cyanobacterial cells and microcystins. As with other cyanotoxins, a high proportion of microcystins remain intracellular unless cells are lysed or damaged, and can therefore be removed by coagulation and filtration in a conventional treatment plant (Chorus and Bartram 1999 Chapter 9). It should be noted that using oxidants such as chlorine or ozone to treat water containing cyanobacterial cells, while killing the cells, will also result in the release of free toxin; therefore pre-chlorination or pre-ozonation are not recommended without a subsequent step to remove dissolved toxins.

Microcystins are readily oxidised by a range of oxidants, including ozone and chlorine. Adequate contact time and pH control are needed for optimal removal of these compounds, and this will be more difficult to achieve in the presence of whole cells (Drikas *et al*. 2002). Microcystins are also adsorbed from solution by both granular activated carbon and, less efficiently, by powdered activated carbon. (Drikas *et al*. 2002). Because powdered activated carbon may be a more practical option for intermittent or emergency use, it is important to seek advice and carefully select the most appropriate type for toxin removal, as carbons vary significantly in performance for different compounds. Boiling is not effective for destruction of microcystins.

If treatment is instituted in response to the presence of toxin-producing cyanobacteria, the effectiveness of the process needs to be confirmed by testing for toxin in the product water.

NOTE: Important general information is contained in PART II, Chapter 5

METHODS OF IDENTIFICATION AND DETECTION

Animal bioassays (mouse tests) have traditionally been used for detecting the entire range of cyanotoxins, including microcystins. These tests provide a definitive indication of toxicity, although they cannot be used for precise quantification of compounds in water or for determining compliance with the guideline value. A number of techniques are available for determining microcystins in water (Chorus and Bartram 1999 Chapter 13) The analytical technique selected needs to allow quantitative comparison with the guideline value in terms of toxicity equivalents. When quantitative standards are available, the most precise technique in this regard is liquid chromatography with confirmation by mass spectrometry (LC–MS/MS); although this technique still involves estimation of the concentration and therefore toxicity of some microcystins in a sample against microcystin-LR as the analytical standard, which may result in a slight overestimate of total microcystins (as microcystin-LR, toxicity equivalents).

A range of commercially available immunoassay (ELISA) kits offer a rapid technique for screening and semi-quantitative measurement of toxins in cyanobacterial cell material and in water. A potential limitation of these assays is the poor cross-reactivity of the antibodies between variants of microcystin, which can lead to underestimation of total toxicity.

Cyanobacteria are detected by light microscopy, identified using morphological characteristics, and counted per standard volume of water (Hotzel and Croome 1999). Practical keys for their identification are provided in Baker and Fabbro (2002).

HEALTH CONSIDERATIONS

Microcystins are primarily hepatotoxins. The mechanism of toxicity involves inhibition of protein phosphatase enzymes in eucaryotic cells. Acute exposure to high doses of microcystin administered either intravenously (i.v.) or by intraperitoneal (i.p.) injection causes severe liver damage and is characterised by a disruption of liver cell skeletal structure, a loss of sinusoidal structure, increases in liver weight due to intrahepatic haemorrhage, haemodynamic shock, heart failure and death.

There is a significant number of reports describing animal poisonings from ingesting water that contains *Microcystis*, with some examples confirming hepatotoxicity and the associated presence of microcystins (Ressom *et al*. 1994). Significant human illness has been strongly associated with exposure to microcystins in recreational waters (Turner *et al*. 1990). In an unfortunate incident in 1996, a large number of dialysis patients in Caruaru, Brazil, were exposed to microcystins intravenously and experienced a range of symptoms including headache, eye pain, blurred vision, nausea and vomiting (Jochimsen *et al*. 1998). This incident showed that human sensitivity is similar to that found in animal studies. The patients' livers were painfully enlarged and many experienced subcutaneous, nasal or uterine bleeding. Liver samples from 52 of the 76 people who died in this incident were examined; they showed disruption of liver plates and liver cell morphology, necrosis, apoptosis, and cholestasis; and intracellular structures were also deranged. Using various assumptions, the patients who died were exposed to an estimated raw water microcystin concentration of 19.5 µg L^{-1} by dialysis, but much higher exposure levels would be required to cause these serious health outcomes by ingestion because of the much lower uptake of microcystin across the gut (Carmichael *et al*. 2001).

Microcystins are also toxic when inhaled. A study with mice showed that intranasal introduction of microcystin-LR resulted in extensive necrosis of the epithelium of the nasal mucosa of both the olfactory and respiratory zones, progressing to destruction of large areas of tissue down to levels of deep blood vessels (Fitzgeorge *et al*. 1994). The LD_{50} by this route of administration was the same as the i.p. LD_{50}, and dose-dependent liver lesions were observed. The same authors also demonstrated cumulative liver damage after repeated dosing.

NOTE: Important general information is contained in PART II, Chapter 5

In experimental animal studies, microcystin-LR can produce extreme acute toxicity. In mice the LD_{50} is in the range of 0.025 to 0.15 mg/kg bodyweight for the i.p. route, and 5 and 10.9 mg/kg bodyweight respectively for oral administration for two different strains of mice. These differences show that much higher levels of exposure are required by ingestion compared with i.p. injection. Even higher values have been demonstrated in rats (Chorus and Bartram 1999 Chapter 4).

Microcystins promote the growth of tumours in experimental animals (Falconer 1991, Nishiwaki-Matsushima *et al.* 1992). The significance of this for humans, who may be subject to chronic exposure via drinking water, is unclear.

Microcystins have been implicated in causing liver damage in an Australian population exposed via reticulated town water supply where the source water contained blooms of *Microcystis* (Falconer *et al.* 1983).

The International Agency for Research on Cancer (IARC) convened an expert Working Group in 2006 to assess the evidence for the carcinogenicity of microcystins. Their conclusion after considering all of the evidence but emphasising the strength of the mechanistic data, was that microcystin-LR is "possibly carcinogenic to humans" (group 2B) (Grosse *et al.* 2006).

Microcystins are currently regarded as non-genotoxic.

DERIVATION OF GUIDELINE

$$1.3\ \mu g/L = \frac{40\ \mu g/kg\ bodyweight\ per\ day \times 70\ kg \times 0.9}{2\ L/day \times 1000}$$

where:

- 40 μg/kg body weight per day is the No Observed Adverse Effect Level (NOAEL) from a 13-week ingestion study with microcystin-LR in mice, based on liver histopathology and serum enzyme level changes (Fawell *et al.* 1994);
- 70kg is the average weight of an adult;
- 0.9 is the proportion of total daily intake attributed to the consumption of water;
- 2 L/day is the average amount of water consumed by an adult;
- 1000 is the safety factor derived from extrapolation of an animal study to humans (10 for interspecies variability, 10 for intraspecies variability and 10 for limitations in the database, related particularly to the lack of data on chronic toxicity and carcinogenicity).

The guideline is derived for total microcystins and expressed as microcystin-LR toxicity equivalents (TE). This is because the total microcystin concentration should be considered in relation to potential health impacts.

The World Health Organization (WHO) evaluation of the health-related information for cyanobacterial toxins (Gupta 1998, WHO 1998, Chorus and Bartram 1999 Chapter 5) concluded that there are insufficient data to allow a guideline value to be derived for any cyanobacterial toxins other than microcystin-LR. The guideline recommended by the WHO for drinking water is 1 μg/L (rounded figure) for total microcystin-LR (free plus cell-bound), based on the Fawell *et al.* (1994) sub-chronic study. This guideline value for microcystin-LR is provisional, as the database is regarded as limited (WHO 1998).

The approach being taken for guideline derivation here is essentially similar to that used by WHO (Chorus and Bartram 1999 Chapter 5). The same ingestion study in mice was used to calculate the NOAEL. The difference between the Australian guideline of 1.3 μg/L total microcystin (as microcystin-LR TE) and the WHO provisional guideline of 1 μg/L microcystin-LR is due to use of a different average body weight for an adult (70 kg versus 60 kg), and a different proportion of the daily microcystin intake attributed to drinking water (0.9 in the Australian guideline versus 0.8 selected by WHO).

NOTE: Important general information is contained in PART II, Chapter 5

The higher figure is due to lower potential exposure in Australia from other environmental sources, such as contaminated bathing water, and via dietary supplements potentially containing microcystins.

Where *M. aeruginosa* occurs in drinking water supplies and toxin monitoring data are unavailable, cell numbers can provide a preliminary indication of the potential hazard to public health. For a highly toxic population of *M. aeruginosa* (toxin cell quota of 0.2 pg total microcystins/cell; mean cell volume of 87 μm^3), a cell density of approximately 6,500 cells/mL (biovolume of 0.6 mm^3/L) would be equivalent to the guideline of 1.3 µg/L microcystin-LR (TE) if the toxin were fully released into the water. This number is indicative only; toxin determination is required for health risk assessment.

NOTIFICATION PROCEDURE

It is recommended that a notification procedure be developed by water and health authorities. A tiered framework should be considered. Initial notification to health authorities could be provided when numbers of *M. aeruginosa* reach 30% of the density equivalent to the guideline value of 1.3 µg/L microcystin (2,000 cells/mL; biovolume 0.2 mm^3/L), while an alert could be provided when cell numbers are equivalent to the guideline value (6,500 cells/mL; biovolume 0.6 mm^3/L). For microcystin-producing species other than *M. aeruginosa*, notifications and alerts should be based on biovolumes.

In all cases, cell numbers should be used only as preliminary signals and as triggers for toxin testing to enable assessment of potential health risks.

REFERENCES

Baker PD, Fabbro LD (2002). *A Guide to the Identification of Common Blue-Green Algae (Cyanoprokaryotes) in Australian Freshwaters*. CRCFE Identification Guide No. 25, Cooperative Research Centre for Freshwater Ecology, Albury.

Baker P, Humpage AR (1994). Toxicity associated with commonly occurring cyanobacteria in surface waters of the Murray-Darling Basin, Australia. *Australian Journal of Marine and Freshwater Research*, 45:773-786.

Carmichael WW, Azevedo SMFO, An JS, Molica, RJR, Jochimsen EM, Lau S, Rinehart KL, Shaw GR, Eaglesham GK (2001). Human fatalities from cyanobacteria: Chemical and biological evidence for cyanotoxins. *Environmental Health Perspectives*, 109:663-668.

Chorus I, Bartram J (eds) (1999). *Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management*. E&FN Spon, London.

Drikas M, Newcombe, G, Nicholson B (2002). Water treatment options for cyanobacteria and their toxins. In: *Blue-Green Algae: Their significance and management within water supplies*. CRC for Water Quality & Treatment Occasional Paper 4, pp 75-92.

Falconer IR (1991). Tumour promotion and liver injury caused by oral consumption of cyanobacteria. *Environmental Toxicology and Water Quality*, 6:177-184.

Falconer IR, Beresford AM, Runnegar MTC (1983). Evidence of liver damage by toxin from a bloom of the blue-green alga, *Microcystis aeruginosa*. *Medical Journal of Australia*, 1:511-514.

Fawell JK, James CP, James HA (1994). *Toxins from Blue-Green Algae: Toxicological assessment of Microcystin-LR and a method for its determination in water*. pp 1-46. Water Research Centre, Medmenham, UK.

Fitzgeorge R, Clark S, Keevil C (1994). Routes of intoxication. In: Codd GA, Jeffries TM, Keevil CW, Potter P eds. *Detection Methods for Cyanobacterial Toxins*. Royal Society of Chemistry, Cambridge, pp 69-74.

NOTE: Important general information is contained in PART II, Chapter 5

Grosse Y, Baan R, Straif K, Secretan B, El Ghissassi F and Cogliano V (2006) Carcinogenicity of nitrate, nitrite and cyanobacterial peptide toxins. *The Lancet Oncology*, 7:628-629.

Gupta S (1998). *Cyanobacterial Toxins: Microcystin-LR in Guidelines for Drinking Water Quality. 2nd Edition. Addendum to Volume 2. Health Criteria and Other Supporting Information.* World Health Organization, Geneva, pp 95-110.

Hotzel G, Croome R (1999). *A Phytoplankton Methods Manual for Australian Freshwaters.* LWRRDC Occasional Paper 22/99, Land and Water Resources Research and Development Corporation, Canberra.

Jochimsen EM, Carmichael WW, An J, Cardo DM, Cookson ST, Holmes CEM, Antunes BdeC, De Melo Filho DA, Lyra TM, Barreto VST, Azevedo SMFO, Jarvis WR (1998). Liver failure and death after exposure to microcystins at a hemodialysis center in Brazil. *The New England Journal of Medicine*, 338(13):873-878.

Jones GJ, Bourne DG, Blakeley RL, Doelle H (1994). Degradation of the cyanobacterial hepatotoxin microcystin by aquatic bacteria. *Natural Toxins*, 2:228-235.

Nishiwaki-Matsushima R, Ohta T, Nishiwaki S, Suganuma M, Kohyama K, Ishikawa T, Carmichael WW, Fujiki H (1992). Liver tumor promotion by the cyanobacterial cyclic peptide toxin microcystin-LR. *Journal of Cancer Research and Clinical Oncology*, 118:420-424.

Ressom R, Soong FS, Fitzgerald J, Turczynowicz L, El Saadi O, Roder D, Maynard T, Falconer I (1994). *Health Effects of Toxic Cyanobacteria (Blue-Green Algae)*. National Health and Medical Research Council, Canberra.

Turner PC, Gammie AJ, Hollinrake K, Codd GA (1990). Pneumonia associated with contact with cyanobacteria. *British Medical Journal*, 300:1440-1441.

Wacklin P, Hoffmann L, Komarek J (2009). Nomenclature validation of the genetically revised cyanobacterial genus *Dolichospermum* (Ralfs ex Bornet et Flahault) comb nova. *Fottea* 9:59-64

WHO (World Health Organization) *(1998). Cyanobacterial Toxins: Microcystin-LR. Guidelines for Drinking Water Quality. 2nd Edition. Addendum to Volume 1. Recommendations.* World Health Organization, Geneva, pp 13-14.

NOTE: Important general information is contained in PART II, Chapter 5

Nodularin

(endorsed 2011)

GUIDELINE

Due to the lack of adequate data, no guideline value is set for concentrations of nodularin. However given the known toxicity of nodularin, the relevant health authority should be notified immediately if blooms of* Nodularia spumigena *are detected in sources of drinking water.

GENERAL DESCRIPTION

Nodularin is a cyclic pentapeptide hepatotoxin produced by and named after the cyanobacterium *Nodularia spumigena*. Nodularin is structurally similar to microcystins and exerts similar toxicity to microcystin-LR at its main target site in the liver.

Nodularin is found only in the cyanobacterium *N. spumigena* and up to five toxic variants of the usual structure and one non-toxic variant have been found to date. These variants are not considered here, as they appear to be very rare and the majority of Nodularin found in environmental samples are all of one type. The production of toxins and therefore the presence of toxicity in individual populations of some cyanobacterial species is known to be variable (Chorus and Bartram 1999 Chapter 3). In the case on *N. spumigena,* however, the majority of the strains tested so far in Australia appear to produce nodularin. It is therefore likely that most blooms of *N. spumigena* will have some degree of toxicity.

AUSTRALIAN SIGNIFICANCE

The cyanobacterium *N. spumigena* occurs primarily in brackish water. It forms blooms in estuarine lakes in Australia, New Zealand and Europe, and can also occur in brackish inland lakes in Australia (Wood 1975). In addition to these saline environments, there are also frequent blooms of toxic *N. spumigena* in freshwater lakes of the lower River Murray, South Australia (Baker and Humpage 1994). This rare circumstance where *N. spumigena* blooms in fresh water is of particular importance as the water is used for potable supplies, irrigation and stock watering. Lake Alexandrina in South Australia was the site of the first scientifically documented animal poisoning by *N. spumigena*, and indeed by any cyanobacterium (Francis, 1878). It is likely that these poisonings and the toxic effects described by Francis were due to nodularin. Low numbers of *N. spumigena* have also been recorded in the other (freshwater) river systems of the Murray-Darling Basin. The limited geographic scope for blooms of this organism in freshwater in Australia makes the occurrence of nodularin a relatively minor public health threat with respect to drinking water.

TREATMENT OF DRINKING WATER

The first line of defence against cyanobacteria is catchment management to minimise nutrient inputs to source waters. Source water management techniques to control cyanobacterial growth include maintaining flow in regulated rivers; water mixing techniques to eliminate stratification and reduce nutrient release from sediments in reservoirs; and the use of algicides in dedicated water supply storages. Caution is necessary in using algicides if a bloom has developed because these agents will disrupt cells and liberate nodularins which are largely intracellular and can otherwise be removed by cell removal as noted below. Once intracellular toxins are released they are much more difficult to manage. Nodularins will eventually be released into the water phase when a developed bloom declines and algal cells lyse, reinforcing the need to prevent blooms as far as possible. Algicide use should be in accordance with local

NOTE: Important general information is contained in PART II, Chapter 5

environment and chemical registration regulations. Where multiple intakes are available, withdrawing water selectively from different depths can minimise the intake of high accumulations of cyanobacterial cells at the surface.

Water treatment processes can be highly effective in removing both cyanobacterial cells and nodularin. As with other cyanotoxins, a high proportion of nodularin remains intracellular unless cells are lysed or damaged, and can therefore be removed by coagulation and filtration in a conventional treatment plant (Chorus and Bartram 1999 Chapter 9). It should be noted that using oxidants such as chlorine or ozone to treat water containing cyanobacterial cells, while killing the cells, will also result in the release of free toxin; therefore pre-chlorination or pre-ozonation are not recommended without a subsequent step to remove dissolved toxins.

Nodularin is readily oxidised by chlorine, but has not been evaluated with ozone. Adequate contact time and pH control are needed to ensure optimum removal of these compounds, and this will be more difficult in the presence of whole cells (Chorus and Bartram 1999 Chapter 9). Nodularin is also adsorbed from solution by powdered activated carbon, although it is important to seek advice and carefully select the most appropriate type for toxin removal, as carbons vary significantly in performance for different compounds. Boiling is not effective for destruction of nodularin.

If treatment is instituted in response to the presence of toxin-producing cyanobacteria, the effectiveness of the process needs to be confirmed by testing for toxin in the product water.

METHOD OF IDENTIFICATION AND DETECTION

Animal bioassays (mouse tests) have traditionally been used for detecting the presence of the entire range of cyanotoxins including nodularin. These tests provide a definitive indication of toxicity, although they cannot be used for precise quantification of compounds in water. A number of techniques are available for determining nodularin in water (Chorus and Bartram 1999 Chapter 13). These include screening techniques based on enzyme-linked immunosorbent assays (ELISA), protein phosphatase inhibition assays, and quantitative techniques such as high performance liquid chromatography (HPLC). The analytical techniques based on liquid chromatography (HPLC, liquid chromatography with mass spectrometry) offer good quantitative information on toxin concentrations, especially as chemical standards for nodularin are commercially available.

Cyanobacteria are detected by light microscopy, identified using morphological characteristics, and counted per standard volume of water (Hotzel and Croome 1999). Practical keys for their identification are provided in Baker and Fabbro (2002).

HEALTH CONSIDERATIONS

There are no reports of human health effects from consumption of water containing nodularin and/or *N. spumigena*. In addition, there are no human or animal studies of toxicity by oral exposure to nodularin. Nodularin is at least as hepatotoxic as microcystin for intraperitoneal exposure in experimental animals and, given its identical mode of action, can be regarded as presenting at least the same risk to human health as microcystin if ingested in drinking water. Nodularin is also known to accumulate in mussels in estuaries, and the consumption of contaminated shellfish therefore represents a potential alternative route of human exposure (Falconer *et al.* 1992).

DERIVATION OF GUIDELINE

There are insufficient animal toxicity data to establish a guideline value for nodularin.

NOTE: Important general information is contained in PART II, Chapter 5

As there are some similarities between the toxicity of nodularin and microcystins, the guideline for microcystins (see Microcystins Fact Sheet) could be used to derive cell numbers of *N. spumigena* that provide a preliminary indication of the potential hazard. The only available monitoring data for nodularin in fresh water indicated that the upper range for cell numbers of *N. spumigena* was 50,000-80,000 cells/mL, and this correlated with nodularin levels of 1.0-1.7 µg/L (Heresztyn and Nicholson 1997). Based on these limited data, nodularin levels of around 1.3 µg/L would be associated with cell densities of 40,000-100,000 cells/mL (biovolume of 9.1 to 22.7 mm^3/L; based on a mean cell volume of 227 μm^3).

NOTIFICATION PROCEDURE

It is recommended that a notification procedure be developed by water and health authorities. A tiered framework should be considered. Initial notification to health authorities could be provided when numbers of *N. spumigena* reach 30% of the density equivalent to 1.3 µg/L nodularin (12,000 cells/mL; biovolume 2.7 mm^3/L), while an alert could be provided when cell numbers are equivalent to 1.3 µg/L nodularin (40,000 cells/mL; biovolume 9.1 mm^3/L).

In all cases, cell numbers should only be used as preliminary signals and as triggers for toxin testing to enable assessment of potential health risks.

REFERENCES

Baker PD, Fabbro LD (2002). *A Guide to the Identification of Common Blue-Green Algae (Cyanoprokaryotes) in Australian Freshwaters*. CRCFE Identification Guide No. 25, Cooperative Research Centre for Freshwater Ecology, Albury.

Baker P, Humpage AR (1994). Toxicity associated with commonly occurring cyanobacteria in surface waters of the Murray-Darling Basin, Australia. *Australian Journal of Marine and Freshwater Research*, 45:773-786.

Chorus I, Bartram J (eds) (1999). *Toxic Cyanobacteria in Water. A guide to their public health consequences, monitoring and management*. E&FN Spon, London.

Falconer IR, Choice A, Hosja W (1992). Toxicity of the edible mussel (Mytilus edulis) growing naturally in an estuary during a water-bloom of the blue-green alga Nodularia spumigena. *Environmental Toxicology and Water Quality*, 7:119-123.

Francis G (1878). Poisonous Australian lake. *Nature*, 18:11-12.

Heresztyn T, Nicholson BC (1997). Nodularin concentrations in Lakes Alexandrina and Albert, South Australia, during a bloom of the cyanobacterium (blue-green alga) *Nodularia spumigena* and degradation of the toxin. *Environmental Toxicology and Water Quality*, 12:273-282.

Hotzel G, Croome R (1999). *A Phytoplankton Methods Manual for Australian Freshwaters*. LWRRDC Occasional Paper 22/99, Land and Water Resources Research and Development Corporation, Canberra.

Wood GW (1975). *An Assessment of Eutrophication in Australian Inland Waters*. Australian Water Resources Council Technical Paper No.15. Australian Government Publishing Service, Canberra.

NOTE: Important general information is contained in PART II, Chapter 5

Saxitoxins

(endorsed 2011)

GUIDELINE

Due to the lack of adequate data, no guideline value is set for concentrations of saxitoxins. However given the known toxicity, the relevant health authority should be notified immediately if blooms of* Anabaena circinalis *(*Dolichospermum circinalis*)[1] or other producers of saxitoxins are detected in sources of drinking water.

GENERAL DESCRIPTION

There are three types of cyanobacterial neurotoxins: anatoxin a, anatoxin a-s and the saxitoxins. The saxitoxins include saxitoxin, neosaxitoxin, C-toxins and gonyautoxins (Chorus and Bartram 1999 Chapter 3). The anatoxins seem unique to cyanobacteria, while saxitoxins are also produced by various dinoflagellates under the name of paralytic shellfish poisons (PSPs). A number of cyanobacterial genera can produce neurotoxins, including *Anabaena (Dolichospermum)*, *Oscillatoria*, *Cylindrospermopsis*, *Cylindrospermum*, *Lyngbya* and *Aphanizomenon,* but to date in Australia, neurotoxin production has only been detected from *Anabaena circinalis (Dolichospermum circinalis)*, and the Australian isolates appear to produce only saxitoxins (Velzeboer *et al.* 1998). As with most toxic cyanobacteria, *A. circinalis (D. circinalis)* tends to proliferate in calm, stable waters, particularly in summer when thermal stratification reduces mixing.

The toxicity of individual populations of *A. circinalis (D. circinalis)* is variable, and one extensive survey of the toxicity across the Murray-Darling Basin indicated that 54% of field samples tested were neurotoxic (Baker and Humpage, 1994). A natural population may consist of a mixture of toxic and non-toxic strains and this is believed to explain why population toxicity may vary over time and between samples (Chorus and Bartram 1999 Chapter 3).

The saxitoxins are a group of carbamoyl and decarbamoyl alkaloids that are either non-sulfated (saxitoxins), singly-sulfated (gonyautoxins), or doubly-sulfated (C-toxins). The various types of toxins vary in potency, with saxitoxin having the highest toxicity. The prevalent toxins in Australian blooms of *A. circinalis* are the C-toxins. These can convert in the environment or by acidification or boiling to more potent toxins (Negri *et al.* 1997, Ravn *et al.* 1995). The half-lives for breakdown of a range of different saxitoxins in natural water have been shown to vary from 9 to 28 days, and gonyautoxins may persist in the environment for more than three months (Jones and Negri, 1997).

AUSTRALIAN SIGNIFICANCE

Blooms of *A. circinalis (D. circinalis)* have been recorded in many rivers, lakes, reservoirs and dams throughout Australia, and *A. circinalis (D. circinalis)* is the most common organism in riverine blooms in the Murray-Darling Basin (Baker and Humpage 1994). In temperate parts of Australia blooms typically occur from late spring to early autumn. The first reported neurotoxic bloom of *A. circinalis (D. circinalis)* in Australia occurred in 1972 (May and McBarron 1973). The most publicised blooms occurred in the Murray-Darling System in 1991, 2009 and 2010 (NSWBGATF 1992, NSW Office of Water 2009, MDBA 2010). The first bloom extended over 1,000 kilometres of the Darling-Barwon River system in New South Wales (NSWBGATF 1992). A state of emergency was declared, with a focus on providing safe drinking water to towns, communities and landholders. Stock deaths were associated with the occurrence

1 A change of nomenclature has been proposed for *Anabaena* to *Dolichospermum* (Wacklin P, Hoffmann L and Komarek J (2009). Nomenclature validation of the genetically revised cyanobacterial genus *Dolichospermum*(Ralfs ex Bornet et Flahault) comb nova. *Fottea* 9: 59-64). Both names are cited due to common usage of *Anabaena* and recognising that references cited use the name *Anabaena*

NOTE: Important general information is contained in PART II, Chapter 5

of the bloom but there was little evidence of human health impacts. The blooms in 2009 and 2010 affected several hundred kilometres of the River Murray on the border between NSW and Victoria and included *Anabaena*, *Microcystis* and *Cylindrospermopsin*. Alerts were issued about risks to recreational use, primary contact by domestic users, livestock and domestic animals. A bloom of *A. circinalis (D. circinalis)* in a dam in New South Wales was shown to have caused sheep deaths (Negri *et al.* 1995).

Relatively low numbers of *A. circinalis (D. circinalis)* (below 2,000 cells/mL) can produce offensive tastes and odours in drinking water due to the production of odorous compounds such as geosmin.

TREATMENT OF DRINKING WATER

The first line of defence against cyanobacteria is catchment management to minimise nutrient inputs to source waters. Source water management techniques to control cyanobacterial growth include maintaining flow in regulated rivers; water mixing techniques to eliminate stratification and reduce nutrient release from sediments in reservoirs; and the use of algicides in dedicated water supply storages Caution is necessary in using algicides if a bloom has developed because these agents will disrupt cells and liberate saxitoxins which are largely intracellular and can otherwise be removed by cell removal as noted below. Once intracellular toxins are released they are much more difficult to manage. Saxitoxins will eventually be released into the water phase when a developed bloom declines and algal cells lyse, reinforcing the need to prevent blooms as far as possible. Algicide use should be in accordance with local environment and chemical registration regulations. Where multiple intakes are available, withdrawing water selectively from different depths can minimise the intake of high accumulations of cyanobacterial cells at the surface.

Water treatment processes can be highly effective in removing both cyanobacterial cells and saxitoxins. As with other cyanotoxins, a high proportion of saxitoxins remain intracellular unless cells are lysed or damaged, and can therefore be removed by coagulation and filtration in a conventional treatment plant (Chorus and Bartram 1999 Chapter 9). It should be noted that using oxidants such as chlorine or ozone to treat water containing cyanobacterial cells, while killing the cells, will also result in the release of free toxin; therefore pre-chlorination or pre-ozonation are not recommended without a subsequent step to remove dissolved toxins.

Saxitoxins are adsorbed from solution by both granular activated carbon and powdered activated carbon. Because powdered activated carbon may be a more practical option for intermittent or emergency use, it is important to seek advice and carefully select the most appropriate type for toxin removal, as carbons vary significantly in performance for different compounds. Ozone and normal doses of chlorine may not be entirely effective in destroying saxitoxins. Destruction of saxitoxins by chlorine is dependent on both pH and the particular toxin, and toxin destruction only occurs at relatively high pH (Drikas *et al.* 2002). Boiling is not effective for destruction of saxitoxins.

If treatment is instituted in response to the presence of toxin-producing cyanobacteria, the effectiveness of the process needs to be confirmed by testing for toxin in the product water.

METHOD OF IDENTIFICATION AND DETECTION

The established method for measuring toxicity due to the presence of saxitoxins/PSPs is the mouse bioassay (Hollingworth and Wekell 1990) which provides a result in terms of equivalence to µg saxitoxin activity (STX-eq). This is the standard method used in association with the shellfish industry and recognised by Foods Standards Australia and New Zealand. Where appropriate standards are available, the analytical technique of high performance liquid chromatography with post-column derivatisation can be used to quantify a range of saxitoxins in both water and cell material (Rositano *et al.* 1998, Chorus and Bartram 1999 Chapter 13). This information can then be used to derive an estimate of total toxins in terms

of saxitoxin equivalents (STX-eq) using a conversion based on specific mouse toxicities given by Oshima (1995) (see Rositano *et al.* 1998).

A number of immunoassay procedures (ELISA), developed for application to contaminated shellfish, are available for detection of saxitoxins. These assays are highly sensitive to the individual toxins against which antibodies have been generated, however they all show poor cross-reactivity to other saxitoxins. In particular, if antibodies have been generated against STX, there is virtually no response to the C toxins (Cembella and Lamoureux 1993), which are the predominant toxins in some cyanobacteria such as neurotoxic *A. circinalis (D. circinalis)*, and thus these assays may be very poor in determining these compounds

Cyanobacteria are detected by light microscopy, identified using morphological characteristics, and counted per standard volume of water (Hotzel and Croome 1999). Practical keys for the identification are provided in Baker and Fabbro (2002).

HEALTH CONSIDERATIONS

There is no evidence of human health effects caused directly by consuming water containing saxitoxin-producing cyanobacteria or PSP-producing dinoflagellates. There are, however, numerous reports of human toxicity associated with consumption of shellfish containing relatively high concentrations of PSPs (Kao 1993). Paralytic shellfish poisoning is an acute disorder that can lead to paraesthesia of the mouth and throat progressing to the neck and extremities, dizziness, weakness, ataxia and muscular paralysis with associated symptoms including nausea, vomiting, thirst and tachycardia. Symptoms can occur within 5 minutes and in fatal cases, death occurs within 2-12 hours. In non-fatal cases, intoxication generally resolves within 1-6 days. The toxin is rapidly cleared by urinary excretion. There are no known chronic effects but long-term animal studies are lacking.

In addition, it has been shown that saxitoxins can accumulate in the Australian freshwater mussel *Alathyria condola* by filter feeding on *A. circinalis (D. circinalis)* (Negri and Jones, 1995), and the consumption of contaminated shellfish from water affected by *A. circinalis (D. circinalis)* blooms therefore represents a potential alternative route of human exposure.

DERIVATION OF HEALTH ALERT

There are insufficient toxicity data to establish a guideline value. An analysis of data from reported events of paralytic shellfish poisoning found that most cases of illness were associated with consumption of in excess of 200 µg STX-eq per person, with a low effect level of 124 µg STX-eq. A health alert value of 3 µg STX-eq/L of drinking water can be calculated for acute exposure associated with occurrence of intermittent blooms of cyanobacteria based on the approach described in Fitzgerald *et al.* (1999).

$$\text{3.1 µg/L rounded to 3 µg/L} = \frac{\text{124 µg STX-eq} \times 0.5}{\text{2 L/day} \times 10}$$

where:

- 124 µg STX-eq is the Low Observed Adverse Effect Level (LOAEL) from published human poisonings (Fitzgerald *et al.* 1999).
- 0.5 is the proportion of total daily intake attributed to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 10 is the safety factor derived from use of a LOAEL rather than a NOAEL.

Based on Australian monitoring data, this would require cell densities exceeding 20,000 cells/mL

NOTE: Important general information is contained in PART II, Chapter 5

(biovolume of 5 mm^3/L; based on a mean cell volume of 250 μm^3). Water associated with cell densities of this magnitude would normally be malodorous and unpalatable, with the threshold for off-tastes in water being 1,000-2,000 cells/mL.

NOTIFICATION PROCEDURES

It is recommended that a notification procedure be developed by water and health authorities. A tiered framework should be considered. Initial notification to health authorities could be provided when numbers of *A. circinalis (D. circinalis)* reach 30% of the density equivalent to 3 µg/L STX-eq/L (6,000 cells/mL; biovolume 1.5 mm^3/L), while an alert could be provided when cell numbers are equivalent to 3 µg/L STX-eq/L (20,000 cells/mL; biovolume 5 mm^3/L). For saxitoxin producing species other than *A. circinalis (D. circinalis),* notifications and alerts should be based on biovolumes.

In all cases, cell numbers should only be used as preliminary signals and as triggers for toxin testing to enable assessment of potential health risks.

REFERENCES

Baker P, Humpage AR (1994). Toxicity associated with commonly occurring cyanobacteria in surface waters of the Murray-Darling Basin, Australia. *Australian Journal of Marine and Freshwater Research*, 45:773-786.

Baker PD, Fabbro LD (2002). *A Guide to the Identification of Common Blue-Green Algae (Cyanoprokaryotes) in Australian Freshwaters*. CRCFE Identification Guide No. 25, Cooperative Research Centre for Freshwater Ecology, Albury.

Chorus I, Bartram J (eds) (1999). *Toxic Cyanobacteria in Water: A guide to their public health consequences, monitoring and management*. E&FN Spon, London.

Drikas M, Newcombe, G, Nicholson B (2002). Water treatment options for cyanobacteria and their toxins. In: *Blue-Green Algae: Their significance and management within water supplies*. CRC for Water Quality & Treatment Occasional Paper 4, pp 75-92.

Fitzgerald DJ, Cunliffe DA, Burch MD (1999). Development of health alerts for cyanobacteria and related toxins in drinking water in South Australia. *Environmental Toxicology*, 14(1):203-207.

Hollingworth T, Wekell MM (1990). Paralytic Shellfish Poison In: *Official Methods of Analysis of the Association of Official Analytical Chemists,* 15th edition. Association of the Official Analytical Chemists, Washington DC, pp 881-882.

Hotzel G, Croome R (1999). *A Phytoplankton Methods Manual for Australian Freshwaters*. LWRRDC Occasional Paper 22/99, Land and Water Resources Research and Development Corporation, Canberra.

Jones GJ, Negri AP (1997). Persistence and degradation of cyanobacterial paralytic shellfish poisons (PSPs) in freshwaters. *Water Research*, 31:525-533.

Kao CY (1993). Paralytic Shellfish Poisoning. In: I Falconer, Ed, *Algal Toxins in Seafood and Drinking Water*. Academic Press, London, pp 75-86,

May V, McBarron EJ (1973). Occurrence of the blue-green alga, *Anabaena circinalis* Rabenh. in New South Wales and toxicity to mice and honey bees. *Journal of the Australian Institute of Agricultural Science*, 39:264-266.

MDBA (Murray Darling Basin Authority) (2010). River Murray algal blooms. http://www.mdba.gov.au/media_centre/media_releases/river-murray-algal-blooms (accessed April 2010).

NOTE: Important general information is contained in PART II, Chapter 5

Negri AP, Jones GJ (1995). Bioaccumulation of paralytic shellfish poisoning (PSP) toxins from the cyanobacterium *Anabaena circinalis* by the freshwater mussel *Alathyria condola*. *Toxicon*, 33:667-678.

Negri AP, Jones GJ, Blackburn SI, Oshima Y, Onodera H (1997). Effect of culture and bloom development and of sample storage on paralytic shellfish poisons in the cyanobacterium *Anabaena circinalis*. *Journal of Phycology*, 33:26-35.

Negri AP, Jones GJ, Hindmarsh M (1995). Sheep mortality associated with paralytic shellfish poisoning toxins from the cyanobacterium *Anabaena circinalis*. *Toxicon*, 33:1321-1329.

New South Wales Blue-Green Algal Taskforce (NSWBGATF) (1992). *Blue-Green Algae. Final Report of the New South Wales Blue-Green Algal Taskforce*. New South Wales Department of Water Resources, Parramatta, Australia.

New South Wales Office of Water (2009).The Murray River algal bloom. New South Wales Department of Climate Change and Water, Sydney, Australia.

Oshima Y (1995). Postcolumn derivatization liquid chromatography method for paralytic shellfish poisons. *Journal of AOAC International*, 78:528-532.

Ravn H, Anthoni U, Christophersen C, Nielsen PH, Oshima Y (1995). Standardized extraction method for paralytic shellfish toxins in phytoplankton. *Journal of Applied Phycology*, 7:589-594.

Rositano J, Nicholson B, Heresztyn T, Velzeboer R (1998). *Characterisation and Determination of PSP Toxins in Neurotoxic Cyanobacteria and Methods for Their Removal from Water*. UWRAA Research Report No. 148. Urban Water Research Association of Australia, Melbourne.

Velzeboer RMA, Baker PD, Rositano J (1998). *Characterisation of Saxitoxins Produced by the Cyanobacterial Genus* Anabaena *in Australia*. UWRAA Research Report No. 135. Urban Water Research Association of Australia, Melbourne.

NOTE: Important general information is contained in PART II, Chapter 5

MICROORGANISMS viruses

Adenovirus

(endorsed 2011)

GUIDELINE

No guideline value has been set for Adenovirus and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Adenoviruses have a DNA genome in a non-enveloped icosahedral capsid. They are widespread in nature and different species cause infections in birds, mammals and amphibians. However, they are not considered a cause of zoonotic transmission. There are six subgenera of human Adenoviruses (A-F) containing about 50 serotypes in total. Subgroup F is the only one of the 6 subgenera that does not grow well in culture.

Adenoviruses cause a wide range of symptomatic infections and can be transmitted by a number of routes including, faecal-oral, hand-eye and inhalation of aerosols. Person-to-person contact plays a major role in transmission and epidemics of acute respiratory and ocular disease have been reported in closed communities such as boarding schools and military camps. Contaminated food and water may also be significant sources and a number of outbreaks involving conjunctivitis and pharyngitis have been reported in association with recreational water exposure.

Adenoviruses cause up to 5% of febrile illnesses in children and also cause a high prevalence of asymptomatic infections (Mena and Gerba 2008). Serotypes that can potentially cause respiratory and other non-enteric infections are commonly detected in faecal material.

AUSTRALIAN SIGNIFICANCE

There have been limited surveys of Adenovirus in Australian drinking water but they have been detected in sewage (NRMMC, EPHC, AHMC 2006). There have been no reported outbreaks associated with Australian drinking water supplies.

Internationally several large outbreaks of pharyngitis and conjunctivitis have been associated with swimming pools (Foy *et al.* 1968, Cabelli 1978, Di Angelo *et al.* 1979, Mena and Gerba 2008). While transmission via drinking water is plausible, it has not been confirmed (WHO 2004)

METHOD OF IDENTIFICATION AND DETECTION

Detection of viruses in water typically requires concentration from large volumes of water (10–1000 litres depending on the source). The concentrate is then inoculated into cell cultures. The presence of infectious Adenovirus can be detected by cytopathic effects with enumeration determined using dilution series.

The presence of the virus can also be determined by PCR-based analyses. A limitation of PCR-based methods is that they do not measure infectivity.

NOTE: Important general information is contained in PART II, Chapter 5

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be used to minimise the risk of contamination. Human faecal waste is the source of human infectious Adenoviruses in water supplies, and protection of water catchments from contamination by human wastes is a priority. Water from catchments receiving human waste is likely to be susceptible to contamination, and treatment, including effective filtration and disinfection, will be required to ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

Sanitary surveys of water catchments should be undertaken to identify potential sources of human wastes, assess risk factors for contamination, provide a basis for catchment management to reduce these risks, and determine the level of water treatment required.

Groundwater from confined aquifers or from depth is not generally subject to contamination by adenoviruses; however, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Where adenoviruses are suspected or known to be present in the raw water, treatment will be required. Adenoviruses are relatively resistant to UV light and the dose required for a 90% (1 log 10) kill is 110mJ/cm^2 (Mena and Gerba 2008). Other disinfectants such as chlorine are more effective. Media filtration (with coagulation) and membrane filtration can reduce concentrations by 90% or more depending on membrane pore size and effectiveness of operation. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Backflow prevention policies should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

HEALTH CONSIDERATIONS

Adenoviruses cause a wide spectrum of symptoms including gastroenteritis, acute respiratory diseases, pneumonia, urethritis, haemorrhagic cystitis, epidemic keratoconjunctivitis ("shipyard eye") and pharyngoconjunctival fever ("swimming pool conjunctivitis"). Different serotypes are associated with specific illnesses; for example, types 40 and 41 are the main cause of enteric illness. Adenoviruses are an important source of childhood gastroenteritis.

High attack rates in outbreaks imply that infecting doses are very low and this has been confirmed by quantitative risk assessment (Mena and Gerba 2008).

DERIVATION OF GUIDELINE

The infectious dose for many viruses is very low (1-10 particles) and risk assessments have indicated that safe drinking water should contain less than 1 Adenovirus per 1000 litres of water (Mena and Gerba 2008). No guideline value is proposed and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures, including prevention of source water contamination by human waste, effective disinfection, and protection of distribution systems from ingress of faecal material.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

Cabelli VJ (1978). Swimming associated disease outbreaks. *Journal Water Pollution Control Federation*, 50:1374–1377.

Di Angelo LJ, Hierholzer JC, Keenlyside RA, Anderson LJ, Martone WJ (1979). Pharyngoconjunctival fever caused by *Adenovirus* type 4: report of a swimming pool outbreak with recovery of virus from pool water. *Journal of Infectious Diseases*, 140:42–47.

Foy HM, Cooney MK, Hatlen JB (1968). *Adenovirus* type 3 epidemic associated with intermittent chlorination of a swimming pool. *Archives on Environmental Health*, 17:795–802.

Mena KD, Gerba CP (2008). Waterborne *Adenovirus*. *Reviews of Environmental Contamination and Toxicology*, 198:133-167.

NRMMC (Natural Resource Management Ministerial Council), EPHC (Environment Protection and Heritage Council), AHMC (Australian Health Ministers' Conference) (2006). *Australian Guidelines for Water Recycling: Managing Health and Environmental Risks (Phase 1)*. Canberra.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Section 11.2.1, Adenoviruses. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Enterovirus

(endorsed 2011)

GUIDELINE

No guideline value has been set for Enterovirus and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

The genus Enterovirus is in the family Picornaviridae. They are among the smallest of the viruses with a diameter of 20-30nm and consist of a single-stranded RNA genome in a non-enveloped capsid. The genus includes a broad range of serotypes that infect humans including polioviruses, coxsackieviruses, echoviruses and enteroviruses. Enteroviruses have a worldwide distribution. In temperate climates, most major epidemics occur during the later summer months, whereas in the tropics, disease can occur throughout the year.

Enteroviruses are one of the most common causes of human infections and can cause a broad range of symptomatic infections. Transmission is mainly by person-to-person contact and inhalation of aerosols. Transmission from contaminated drinking water is plausible but has not been proven (WHO, 2004).

AUSTRALIAN SIGNIFICANCE

Enterovirus infections are common in Australia but there is little information on the occurrence of these viruses in Australian drinking water supplies.

Internationally, enteroviruses have been detected in source waters and drinking water supplies (Grabow *et al.* 2001).

METHOD OF IDENTIFICATION AND DETECTION

Detection of enteroviruses in water typically requires concentration from large volumes of water (10–1000 litres depending on the source). The concentrate is then inoculated into cell cultures. The presence of infectious enteroviruses can also be detected by cytopathic effects with enumeration determined using dilution series. Alternatively, a plaque-forming assay can be used.

The presence of the virus can also be determined by PCR-based analyses. A limitation of PCR-based methods is that they do not measure infectivity.

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be used to minimise the risk of contamination. Human faecal waste is the source of infectious enteroviruses in water supplies, and protection of water catchments from contamination by human wastes is a priority. Water from catchments receiving human waste is likely to be susceptible to contamination with enteroviruses, and treatment, including effective filtration and disinfection, will be required to ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

NOTE: Important general information is contained in PART II, Chapter 5

Sanitary surveys of water catchments should be undertaken to identify potential sources of human waste, assess risk factors for contamination, provide a basis for catchment management to reduce these risks, and determine the level of water treatment required.

Groundwater from confined aquifers or from depth is not generally subject to contamination by enteroviruses; however, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Where enteroviruses are suspected or known to be present in the raw water, treatment will be required. Enteroviruses are sensitive to disinfection using agents such as chlorine and UV light. Media filtration (with coagulation) and membrane filtration can reduce concentrations by 90% or more depending on membrane pore size and effectiveness of operation. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Backflow prevention policies should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

HEALTH CONSIDERATIONS

The genus Enterovirus includes a broad range of serotypes that can cause human infections. These serotypes collectively cause a spectrum of diseases including mild febrile illness, myocarditis, meningoencephalitis, poliomyelitis, hand-foot-and-mouth disease and neonatal multi-organ failure. However, most infections are asymptomatic.

DERIVATION OF GUIDELINE

The infectious dose for many viruses is very low (1-10 particles) and risk assessments have indicated that safe drinking water should contain less than 1 virus particle per 1000 litres of water (Regli *et al*. 1991, WHO 2006). No guideline value is proposed and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures, including prevention of source water contamination by human waste, effective disinfection, and protection of distribution systems from ingress of faecal material.

REFERENCES

Grabow WOK, Taylor MB and de Villiers JC (2001). New methods for the detection of viruses: call for review of drinking water quality guidelines. *Water Science and Technology* 43:1-8.

Regli S, Rose JB, Haas CN, Gerba CP (1991). Modelling the risk from Giardia and viruses in drinking water. *Journal of the American Water Works Association*, 83:76-84.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Sections 7.2 Health-based target setting and 11.2.4, Enteroviruses. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Hepatitis viruses

(endorsed 2011)

GUIDELINE

No guideline value has been set for Hepatitis viruses and their inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

The term hepatitis virus describes a group of viruses that target the liver and cause inflammation. The viruses that cause hepatitis are a functional rather than a genetic group, which have been have been designated A, B, C etc. Enteric hepatitis viruses including Hepatitis A and E are transmitted by the faecal-oral route and can be transmitted from contaminated food or water. The other types are parenterally transmitted blood-borne viruses that are not transmitted by water.

Hepatitis A is a single-stranded RNA non-enveloped virus from the family Picornaviridae. There is only one known genetic type. It is highly infectious and causes the disease commonly known as "infectious hepatitis". It enters the body by ingestion, infects epithelial cells and from there passes to the liver via the bloodstream. Like many of the picornaviruses, it causes mild and asymptomatic infections in children but more severe illness in adults. Immunity in adult populations in developing countries may exceed 95%, while it can be less than 50% in developed countries. Hepatitis A is more common in developing countries but has a wide geographic distribution (WHO 2002, 2004)

Hepatitis E is also a single-stranded RNA non-enveloped virus. Classification remains uncertain, but it is not from the family Picornaviridae. There is evidence of genetic variability. It causes hepatitis that is similar in many ways to that caused by Hepatitis A. However, it can have up to a 25% mortality rate in pregnant women. It is widespread but is endemic in regions such as Mexico, Nepal, India, central Asia and parts of Africa, where first infections typically occur in young adults. In these areas Hepatitis E can be the most common source of viral hepatitis. In regions such as North and South America, Japan, Great Britain and Australasia, clinical cases are uncommon and outbreaks rare.

AUSTRALIAN SIGNIFICANCE

There are about 300-500 cases of Hepatitis A and 10-30 cases of Hepatitis E recorded in Australia each year. There is little information on the occurrence of Hepatitis A or E in Australian drinking water supplies. There have been outbreaks of Hepatitis A in Australia. In 1997, 422 illnesses were caused through consumption of oysters grown in contaminated water at Wallis Lake (Kardamanidis *et al.* 2009).

Internationally, waterborne outbreaks have been caused by both Hepatitis A and E. In endemic areas, faecally contaminated water can play an important role in transmission of Hepatitis E and large outbreaks involving up to 100,000 people have been reported (WHO 2002, 2004).

NOTE: Important general information is contained in PART II, Chapter 5

METHOD OF IDENTIFICATION AND DETECTION

Detection of viruses in water typically requires concentration from large volumes of water (10–1000 litres depending on the source). Assays for Hepatitis A and E are based on PCR techniques. A limitation of PCR-based methods is that they do not measure infectivity.

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be used to minimise the risk of contamination. Human faecal waste is the source of infectious Hepatitis A and E in water supplies and protection of water catchments from contamination by human wastes is a priority. Water from catchments receiving human waste is likely to be susceptible to contamination with Hepatitis A. Treatment, including effective filtration and disinfection, will be required to ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

Sanitary surveys of water catchments should be undertaken to identify potential sources of human waste, assess risk factors for contamination, provide a basis for catchment management to reduce these risks, and determine the level of water treatment required.

Groundwater from confined aquifers or from depth is not generally subject to contamination by Hepatitis viruses; however, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Where Hepatitis viruses are suspected or known to be present in the raw water, treatment will be required. Hepatitis viruses are sensitive to disinfection using agents such as chlorine and UV light. Media filtration (with coagulation) and membrane filtration can reduce concentrations by 90% or more depending on membrane pore size and effectiveness of operation. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Backflow prevention policies should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

HEALTH CONSIDERATIONS

Incubation periods are relatively long, at 28-30 days for Hepatitis A and 15-60 days for Hepatitis E. Both appear to be highly infective.

Hepatitis A causes “infectious hepatitis”. Infants and young children rarely show symptoms following infection or will only have mild symptoms. Adults experience stronger symptoms including fever, weakness, fatigue, nausea, joint aches, vomiting and jaundice. Duration varies but liver function typically begins to normalise after 30-40 days. Death is rare and is normally associated with pre-existing conditions or people over 50 years of age.

Hepatitis E causes similar symptoms to Hepatitis Am but pregnant women are at a greater risk of severe illness and mortality in this group can be up to 25% in endemic areas.

DERIVATION OF GUIDELINE

The infectious dose for many viruses is very low (1-10 particles) and risk assessments have indicated that safe drinking water should contain less than 1 virus particle per 1000 litres of water (Regli *et al*. 1991, WHO, 2004). No guideline value is proposed and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures, including prevention of source water contamination by human waste, effective disinfection, and protection of distribution systems from ingress of faecal material.

NOTE: Important general information is contained in PART II, Chapter 5

REFERENCES

Kardamanidis K, Corbett SJ, Zammit AP (2009). Hepatitis A: Wallis Lake revisited. *NSW Public Health Bulletin*, 20:29-30.

Regli S, Rose JB, Haas CN, Gerba CP (1991). Modelling the risk from *Giardia* and viruses in drinking water. *Journal of the American Water Works Association*, 83:76-84.

WHO (World Health Organization) (2002). Enteric hepatitis viruses. In: *Guidelines for Drinking-water Quality* 2nd Edition, Addendum: Microbiological agents in drinking water. World Health Organization, Geneva, Switzerland.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Sections 7.2 Health-based target setting, 11.2.5 Hepatitis A and 11.2.6 Hepatitis E. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Norovirus

(endorsed 2011)

GUIDELINE

No guideline value has been set for Norovirus and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Noroviruses are single-stranded RNA non-enveloped viruses from the family of Caliciviridae. They were previously described as Norwalk-like viruses, after the original strain that caused an outbreak of illness in a school in Norwalk, Ohio in 1968. The viruses were discovered by electron microscopy and were referred to as "small round structured viruses" due to their appearance. Morphologically similar viruses known as Hawaii, Wollan, Ditchling, Parramatta, Snow Mountain and Montgomery County agents were subsequently identified.

Noroviruses are a major cause of acute viral gastroenteritis in all age groups and are typically more prevalent in winter. The major route of transmission is person-to-person by the faecal-oral route although transmission by fomites and via contact with contaminated surfaces has also been suggested. Environmental transmission from drinking water, recreational water and food has been reported.

Investigations of Norovirus occurrence have been hampered by the lack of a culture-based assay. They can be detected using electron microscopy, enzyme immunoassays and PCR-based methods.

AUSTRALIAN SIGNIFICANCE

There is little information on the occurrence of Norovirus and there have been no reported outbreaks associated with Australian drinking water supplies.

Internationally, numerous outbreaks have been attributed to contaminated drinking water and recreational water (Lodder and de Roda Husman 2005). Noroviruses have been detected in high concentrations in surface water and sewage (Lodder and de Roda Husman, 2005).

METHOD OF IDENTIFICATION AND DETECTION

Detection of viruses in water typically requires concentration from large volumes of water (10–1000 litres depending on the source). Assays for Norovirus in water are typically performed using PCR-based methods (Lodder and de Roda Husman 2005). A limitation of PCR-based methods is that they do not measure infectivity.

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be used to minimise the risk of contamination. Human faecal waste is the source of infectious Norovirus spp. in water supplies, and protection of water catchments from contamination by human wastes is a priority. Water from catchments receiving human waste is likely to be susceptible to contamination, and treatment, including effective filtration and disinfection, will be required to ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

NOTE: Important general information is contained in PART II, Chapter 5

Sanitary surveys of water catchments should be undertaken to identify potential sources of human waste, assess risk factors for contamination, provide a basis for catchment management to reduce these risks, and determine the level of water treatment required.

Groundwater from confined aquifers or from depth should be free from contamination by Norovirus. However, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Where Norovirus are suspected or known to be present in the raw water, treatment will be required. Norovirus spp are sensitive to disinfection using agents such as chlorine (Shin and Sobsey 2008) and UV light. Media filtration (with coagulation) and membrane filtration can reduce concentrations by 90% or more depending on membrane pore size and effectiveness of operation. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Backflow prevention policies should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

HEALTH CONSIDERATIONS

Norovirus is the most common cause of acute viral gastroenteritis in developed countries (Lopman *et al*. 2003). The incubation period is usually 24-48 hours, but cases can occur within 12 hours of exposure. Symptoms include nausea, vomiting (more common in children), abdominal cramps and diarrhoea. Low grade fever can occur. As infections can lead to vomiting and no diarrhoea, the condition is also known as "winter vomiting disease." Symptoms are usually mild and last for 24-60 hrs. High attack rates in outbreaks indicate that the infecting dose is very low. This has been confirmed by risk assessment (Teunis *et al*. 2008)

DERIVATION OF GUIDELINE

The infectious dose for many viruses is very low (1-10 particles) and risk assessments have indicated that safe drinking water should contain less than 1 virus per 1000 litres of water (Regli *et al*. 1991, WHO, 2004). No guideline value is proposed and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures including prevention of source water contamination by human waste, effective disinfection, and protection of distribution systems from ingress of faecal material.

REFERENCES

Lodder WJ, de Roda Husman AM (2005). Presence of Noroviruses and other enteric viruses in sewage and surface waters in The Netherlands. *Applied and Environmental Microbiology*, 71:1453–1461.

Lopman BA, Reacher MH, van Duijnhoven Y, Hanon FX, Brown D, Koopmans M (2003). Viral gastroenteritis outbreaks in Europe, 1995-2000. *Emerging Infectious Diseases,* 9:90-96.

Regli S, Rose JB, Haas CN, Gerba CP (1991). Modelling the risk from Giardia and viruses in drinking water. *Journal of the American Water Works Association,* 83:76-84.

Shin G-A, Sobsey M (2008). Inactivation of *Norovirus* by chlorine disinfection in water. *Water Research*, 42:4562-4568.

Teunis PFM, Moe CL, Liu P, Miller SE, Lindesmith L, Baric RS, Pendu JL, Calderon R (2008). Norwalk virus: How infectious is it ? *Journal of Medical Virology*, 80:1468-1476.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Sections 7.2 Health-based target setting. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

Rotavirus

(endorsed 2011)

GUIDELINE

No guideline value has been set for Rotavirus and its inclusion in routine monitoring programs is not recommended.

A multiple barrier approach from catchment to tap is recommended to minimise the risk of contamination. Protecting catchments from human and animal wastes is a priority. Operation of barriers should be monitored to ensure effectiveness.

GENERAL DESCRIPTION

Rotaviruses are in the family Reoviridae and consist of a double-stranded RNA genome in a non-enveloped capsid. The genus Rotavirus is divided into seven groups, A to G. Group A contain the most important human pathogens.

Rotaviruses are the most common cause of severe diarrhoea among young children. Approximately 50-60% of acute gastroenteritis leading to hospitalisation of children is caused by Rotavirus and it causes over 600,000 deaths each year. Rates of infection are lower in adults and disease tends to be milder. The primary mode of transmission is faecal-oral, with inhalation of aerosols also possible. Although large numbers of particles are excreted by infected people, water plays a smaller role than expected.

Investigations of Rotavirus occurrence have been hampered by the lack of a culture-based assay. Rotavirus can be detected using electron microscopy and PCR-based methods.

AUSTRALIAN SIGNIFICANCE

Rotavirus infections are very common in Australia and all children are likely to have been infected at least once. There is little information on the occurrence of Rotavirus in Australian drinking water supplies.

Internationally, Rotavirus has been detected in sewage and surface water (Percival *et al.* 2004, Lodder and de Roda Husman 2005) and occasionally has been associated with waterborne outbreaks (WHO 2004).

METHOD OF IDENTIFICATION AND DETECTION

Detection of viruses in water typically requires concentration from large volumes of water (10-1000 litres depending on the source). Assays for Rotavirus are based on PCR techniques. A limitation of PCR-based methods is that they do not measure infectivity.

PREVENTING CONTAMINATION OF DRINKING WATER

A multiple barrier approach operating from catchment to tap should be used to minimise the risk of contamination. Human faecal waste is the source of infectious Rotavirus in water supplies, and protection of water catchments from contamination by human wastes is a priority. Water from catchments receiving human waste is likely to be susceptible to contamination with Rotavirus, and treatment, including effective filtration and disinfection, will be required to ensure a safe supply. The lower the quality of source water, the greater the reliance on water treatment processes.

NOTE: Important general information is contained in PART II, Chapter 5

Sanitary surveys of water catchments should be undertaken to identify potential sources of human waste, assess risk factors for contamination, provide a basis for catchment management to reduce these risks, and determine the level of water treatment required.

Groundwater from confined aquifers or from depth is not generally subject to contamination by Rotavirus; however, bores need to be well maintained and protected from intrusion of surface and subsurface contamination. Integrity should be monitored using traditional indicators of faecal contamination.

Where rotaviruses are suspected or known to be present in the raw water, treatment will be required. Rotaviruses are sensitive to disinfection using agents such as chlorine and UV light. Media filtration (with coagulation) and membrane filtration can reduce concentrations by 90% or more depending on membrane pore size and effectiveness of operation. Filtration plants should be operated by trained and skilled personnel.

The integrity of distribution systems should be maintained. Backflow prevention policies should be applied and faults and burst mains should be repaired in a way that will prevent contamination.

HEALTH CONSIDERATIONS

The onset of *Rotavirus* symptoms is usually sudden, with vomiting, watery diarrhoea, and fever. The diarrhoea usually last 2-5 days and, in severe cases, can lead to dehydration. In developed countries such as Australia death is uncommon.

DERIVATION OF GUIDELINE

The infectious dose for many viruses is very low (1-10 particles) and risk assessments have indicated that safe drinking water should contain less than 1 virus particle per 1000 litres of water (Gerba *et al.* 1996). No guideline value is proposed and inclusion in routine verification monitoring programs is not recommended. The focus should be on monitoring of control measures, including prevention of source water contamination by human waste, effective disinfection, and protection of distribution systems from ingress of faecal material.

REFERENCES

Gerba CP (1996). Waterborne rotavirus: a risk assessment. *Water Research* 30:2929-2940.

Lodder WJ, de Roda Husman AM (2005). Presence of Noroviruses and other enteric viruses in sewage and surface waters in The Netherlands. *Applied and Environmental Microbiology*, 71:1453–1461.

Percival S, Chalmers R, Embrey M, Hunter P, Sellwood J, Wyn Jones P (2004). *Microbiology of Waterborne Diseases*. Elsevier Academic Press.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*, Section 11.2.7, Rotaviruses and orthoreoviruses. World Health Organization, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 5

PHYSICAL AND CHEMICAL CHARACTERISTICS

Acephate

(endorsed 2011)

GUIDELINE

Based on human health concerns, acephate in drinking water should not exceed 0.008 mg/L.

RELATED CHEMICALS

Acephate (CAS 30560-19-1) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including chlorpyrifos, diazinon, dichlorvos, ethion and temephos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, acephate would not be a health concern unless the concentration exceeded 0.008 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Acephate (O,S-dimethyl acetylphosphoramidothionate) is used for the control insects on fruit, vegetable and nut crops. Acephate breaks down and is metabolised to methamidophos, another insecticide.

There are registered products containing acephate in Australia. These products are intended for professional use and are available as soluble concentrates intended to be diluted and applied by ground or aerial spray application. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to acephate and its metabolites is residues in food. Residue levels in crops grown according to good agricultural practice are generally low.

Agricultural use of acephate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are few data on concentrations of acephate in drinking water in Australia. Acephate has been detected in mains water in Poland with concentrations generally in the range of 0.0001 to 0.0015 mg/L (0.1 to 1.5 µg/L), and up to a maximum of 0.0106 mg/L (10.6 µg/L) (Badach *et al.* 2007). Acephate has also been reported in river water in Spain, with a mean concentration of 0.00047 mg/L (0.47 µg/L) and a maximum of 0.00217 mg/L (2.17 µg/L) over 20 sampling sites (Espigares *et al.* 1997).

TREATMENT OF DRINKING WATER

No reported instances of reliable removal of acephate from drinking water at typical activated carbon doses and contact times have been identified. Some case studies on activated carbon have been reported with very high carbon doses; for example, on the treatment of wastewater from a pesticide manufacturing plant (Banerjee 2002) or in benchscale tests (Suzuki 2002). Although acephate has a relatively short soil half-life of between 3 and 6 days, the hydolysis half life is much longer, at 169 days, rendering reservoir detention an unreliable treatment method for the removal of acephate from water.

MEASUREMENT

Residues of acephate and methamidophos are traditionally determined by gas chromatography–electrodialysis (Espigares *et al.* 1997, Badach *et al.* 2007). The detection limit by this method is 0.00005 mg/L (0.05 µg/L) (Badach *et al.* 2007). Analysis of highly polar, water-soluble organophosphates, including acephate and methamidophos, using hydrophilic interaction liquid chromatography with tandem mass spectrometry has produced much lower limits of detection (Hayama *et al.* 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for acephate is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.22 mg/kg bw/day from a 2-year dietary rat study. The NOEL was based on inhibition of cholinesterase. The ADI incorporates a safety factor of 100 and it was established in 1998.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Acephate is rapidly absorbed from the gastrointestinal tract and widely distributed in the body. It is rapidly eliminated, mainly in the urine, as unchanged acephate. It has a low potential for bioaccumulation. The primary metabolite is methamidophos.

Acute effects: Acephate has low to moderate oral acute toxicity, and low dermal toxicity. It is not a skin sensitiser. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: Decreased erythrocyte cholinesterase activity was seen at the lowest dose tested of 0.5 mg/kg bw/day and above in a 21-day repeat-dose dermal study in rabbits. No other effects were seen.

Decreased plasma, erythrocyte, and brain cholinesterase activity was seen at a dose of 2.5 mg/kg bw/day (monkey) and 3.75 mg/kg bw/day (rat) in 1-month repeat dose dietary studies. Both studies used a single dose level and no other effects were seen. A NOEL was not established in either study.

Long-term effects: Long-term toxicity studies were performed in mice (2 years), rats (28 months), and dogs (2 years). Decreased brain, plasma and erythrocyte cholinesterase activity was seen at doses of 2 mg/kg bw/day (rats). The sole effect in dogs was decreased erythrocyte cholinesterase activity at the highest dose tested of 2.5 mg/kg bw/day. Histopathological changes in the liver (non-neoplastic lesions) at doses of 15 mg/kg bw/day and above were the only effect observed in mice. The lowest overall NOEL was 0.22 mg/kg bw/day in rats, and this is the basis for the current ADI.

NOTE: Important general information is contained in PART II, Chapter 6

Carcinogenicity: Based on a 2-year study in mice, there is no evidence of carcinogenicity for acephate.

Genotoxicity: Acephate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal developmental.

Poisons Schedule: Acephate is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF GUIDELINE

The health-based guideline value of 0.008 mg/L for acephate was determined as follows:

$$0.008 \text{ mg/L} = \frac{0.22 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.22 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Badach H, Nazimek T, Kaminska IA (2007). Pesticide content in drinking water samples collected from orchard areas in central Poland. *Annals of Agricultural and Environmental Medicine*, 14(1):109-114.

Banerjee G, Kumar B (2002). Pesticide (acephate) removal by GAC: A case study. *Indian Journal of Environmental Health*, 44(2):92 – 101.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Espigares M, Coca C, Fernandez-Crehuet M, Moreno O, Bueno A, Galvez R (1997). Pesticide concentrations in the waters from a section of the Guadalquivir River basin, Spain. *Environmental Toxicology and Water Quality*, 12(3): 249-256.

Hayama T, Yoshida H, Todoroki K, Nohta H, Yamaguchi M (2008). Determination of polar organophosphorus pesticides in water samples by hydrophilic interaction liquid chromatography with tandem mass spectrometry. *Rapid Communications in Mass Spectrometry*, 22(14):2203-2210.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NOTE: Important general information is contained in PART II, Chapter 6

Suzuki S, Numajiri S, Misawa K, Toyooka K (2002). Appropriate management of granular activated carbon with adsorption amount of organic compounds. *Yosui to Haisui*, 44(11):994-1000.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Acrylamide

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of acrylamide in drinking water should not exceed 0.0002 mg/L.

GENERAL DESCRIPTION

Acrylamide occurs as a minor impurity in polyacrylamide. It may be present in drinking water through the use of polyacrylamides as flocculant aids in water treatment, and through the use of grouting agents containing polyacrylamide. Overseas studies have reported concentrations of up to a few micrograms per litre in drinking water.

When nonionic or anionic polyacrylamides are used in water treatment at a typical dose level of 1 mg/L, the maximum theoretical concentration of acrylamide has been estimated at 0.0005 mg/L, with practical concentrations 2–3 times lower. Residual levels of acrylamide from the use of cationic polyacrylamides may be higher.

Concern over the health effects of acrylamide has led some countries to introduce tight restrictions on its use for water treatment.

Polyacrylamide is used in food processing and exposure to acrylamide may also occur from this source.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Acrylamide has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Acrylamide can be removed from drinking water by adsorption onto granular activated carbon. It is not removed effectively by conventional water treatment or with powdered activated carbon.

MEASUREMENT

Acrylamide can be analysed using high performance liquid chromatography with ultraviolet detection (Brown and Rhead 1979). The sample is brominated to form 2,3-dibromopropionamide, which is extracted with ethyl acetate and analysed. The limit of determination is 0.0002 mg/L.

HEALTH CONSIDERATIONS

Acrylamide is readily absorbed following ingestion or inhalation, or through the skin, and it forms a number of metabolites. It can accumulate in nervous system tissues and blood. The results of animal studies indicate that it is largely excreted as metabolites in urine and bile. It can cross the placenta.

An extensive review and summary of the human and animal toxicity data for acrylamide is available (IPCS 1985).

Humans exposed for a short time to well water contaminated with up to 400 mg/L of acrylamide showed

effects including confusion, disorientation, memory disturbances and hallucinations. They recovered fully within 4 months. Long-term occupational exposure has resulted in skin irritation, fatigue, foot weakness and sensory changes.

In animals, acrylamide is well established as a neurotoxicant. Short- and long-term effects are similar, with exposure causing paralysis in the hind limbs of cats, dogs and rats at doses from 5 mg/kg body weight per day. The animals recovered completely when short-term exposure stopped. Acrylamide can also impair reproductive organs in rats, cats and dogs at the same dose.

Animal studies indicate that acrylamide is a carcinogen. Male rats receiving low oral doses (0.5 mg/kg body weight per day) for 2 years had increased incidence of scrotal, thyroid and adrenal tumours. Female rats exposed for 18 months had increased tumours of the mammary glands, central nervous system, thyroid and uterus. Mice exposed to higher doses for 8 weeks showed an increased incidence of lung adenomas.

Several studies have reported that acrylamide is not mutagenic in bacteria, but induces gene mutations and chromosomal aberrations in mammalian cells both *in vitro* and *in vivo*.

The International Agency for Research on Cancer (IARC) has concluded that acrylamide is probably carcinogenic to humans (Group 2A, inadequate evidence in humans, sufficient evidence in experimental animals, and supporting mechanistic evidence) (IARC 1994).

DERIVATION OF GUIDELINE

The guideline value for acrylamide of 0.0002 mg/L is based on a consideration of health effects in relation to the limit of determination for analysis using commonly available techniques.

Health-based derivations can be determined as follows:

i) $$0.0007 \text{ mg/L} = \frac{0.2 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 0.2 mg/kg body weight per day is the no-effect level from a 93-day drinking water study using rats (Burek ***et al***. 1980). Longer-term studies only identify lowest effect levels, which are significantly higher than the no-effect level used in the calculation.
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for a less than lifetime study). An additional factor of 10 for carcinogenicity was not applied as tumours occur at doses above those that cause neurotoxic effects. The use of this safety factor was recommended by the NHMRC Standing Committee on Toxicity.

ii) On the basis of a drinking water study using rats (Johnson *et al.* 1986), the excess cancer risk of lifetime consumption of water with an acrylamide concentration of 0.00005 mg/L (50 ng/L) was conservatively estimated by the World Health Organization (WHO), using a linear multistage model, at one additional cancer per million people.

NOTE: Important general information is contained in PART II, Chapter 6

The guideline value was set at the limit of determination because this is within the values derived from health considerations, and provides an adequate degree of protection. This is consistent with the general approach adopted for compounds that are known genotoxic carcinogens (see Section 6.4). The higher WHO guideline value of 0.0005 mg/L is based on an estimated lifetime risk of one additional cancer per 100,000 people.

REFERENCES

Brown L, Rhead MM (1979). Liquid chromatographic determination of acrylamide monomer in natural and polluted aqueous environments. *Analyst*, 104:391–399.

Burek JD, Albee RR, Beyer JE, Bell TJ, Carreon RM, Morden DC, Wade CE, Hermann EA, Gorzinski SJ (1980). Subchronic toxicity of acrylamide administered to rats in the drinking water followed by up to 144 days of recovery. *Journal of Environmental Pathology and Toxicology*, 4:157–182.

IARC (International Agency for Research on Cancer) (1994). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Some Industrial Chemicals. World Health Organization, IARC, 60, Lyon.

IPCS (International Programme on Chemical Safety) (1985). Acrylamide. Environmental Health Criteria, 49. World Health Organization, IPCS.

Johnson KA, Gorzinski SJ, Bodner KM, Campbell RA, Wolf CH, Friedman MA, Mast RW (1986). Chronic toxicity and oncogenicity study on acrylamide incorporated in the drinking water of Fischer 344 rats. *Toxicology and Applied Pharmacology*, 85:154–168.

NOTE: Important general information is contained in PART II, Chapter 6

Aldicarb

(endorsed 2011)

GUIDELINE

Based on human health concerns, aldicarb in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Aldicarb (CAS 116-06-3) belongs to the carbamate class of chemicals. There are many other pesticides in this class, including asulam, carbaryl, methomyl and pirimicarb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, aldicarb would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Aldicarb is an insecticide used in agriculture for the control of insects and nematodes in the soil. Aldicarb is taken up by plants, where it is metabolised into toxicologically active metabolites.

There is at least one registered product that contains aldicarb in Australia. Aldicarb products are intended for professional use and are available as a concentrated granular formulation to be incorporated by tractor equipment directly into the soil of citrus, cotton, and cane crops. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to aldicarb and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of aldicarb may potentially lead to contamination of source waters through processes such as leaching into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are few data on concentrations of aldicarb in Australian drinking waters. In Canada, aldicarb was reported in 111 of 1017 samples in surveys of private and municipal drinking-water supplies (detection limits 0.00001–0.003 mg/L [0.01–3.0 µg/L]); the maximum concentration was 0.028 mg/L (28 µg/L) (WHO 2003)

TREATMENT OF DRINKING WATER

Aldicarb removal in municipal water treatment has been investigated (Kruithof 1994). For the removal of pesticides, operational techniques such as air-stripping, GAC-filtration and ozonation are available and of these, GAC-filtration is considered moderately effective, although is very susceptible to competitive adsorption by natural organic matter.

Reverse osmosis and nanofiltration have been tested for effectiveness at removing Aldicarb degredates from environmental water samples and has been shown to be effective in some applications (Baier *et al.* 1987, Kiso *et al*, 2002).

The effectiveness of ultraviolet irradiation and peroxide, ozonation and chlorination for the removal of aldicarb from drinking water has been identified in laboratory studies (Mason *et al.* 1990, Huston and Pignatello 1999).

MEASUREMENT

Aldicarb and its sulfoxide and sulfone oxidation products can be determined simultaneously by capillary gas chromatography with a nitrogen-phosphorus detector (Health Canada 1995, WHO 2003). The detection limit is 1 µg/L for all three compounds.

High-performance liquid chromatography (HPLC) is often the method of choice and is the basis of United States Environmental Protection Agency (USEPA) Method 531 for carbamate pesticides. Aldicarb, its sulfoxide and its sulfone are separated by reversed-phase HPLC, and the analytes are then hydrolysed to methylamine followed by post-column derivatisation with ortho-phthalaldehyde and fluorescence detection. The detection limits are 0.0013, 0.0008 and 0.0005 mg/L (1.3, 0.8 and 0.5 µg/L) for aldicarb, the sulfoxide and the sulfone, respectively (Health Canada 1995, WHO 2003).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for aldicarb is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.01 mg/kg bw/day from an acute dietary study in human volunteers. The NOEL is based on cholinesterase inhibition in plasma and erythrocytes at 0.025 mg/kg bw. The ADI incorporates a safety factor of 10 and it was established in 1999.

The previous *Australian Drinking Water Guidelines* health value was 0.002 mg/L (NHMRC and NRMMC, 2004).

HEALTH CONSIDERATIONS

Metabolism: Aldicarb is readily absorbed from the gastrointestinal tract and widely distributed in the body. It is rapidly excreted, mainly via the urine. The primary metabolites are the sulfoxide and the sulfone.

Acute effects: Aldicarb has a high acute oral and dermal toxicity. Aldicarb is a skin sensitiser in guinea pigs. Significant plasma and erythrocyte cholinesterase inhibition was seen at doses of 0.025 mg/kg bw and above in a single dose study in human volunteers using drinking water as the dosing vehicle. Clinical symptoms of toxicity typical of cholinesterase inhibition, including tremors, prostration, coma, piloerection, ataxia, and salivation, were seen at the highest dose tested (0.06 mg/kg bw/day) in this study. The NOEL was 0.01 mg/kg bw/day and this is the basis for the ADI.

Short-term effects: Fourteen-day dietary studies in dogs reported reduced bodyweight and clinical signs of cholinesterase inhibition. Cholinesterase inhibition was observed at 0.02 mg/kg bw/day. In 3-month dietary studies in rats and dogs, effects included decreased body weight gain and food consumption in rats, and cholinesterase inhibition was reported at doses of 0.07 mg/kg bw/day and above.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: In medium-term (13-week) dietary studies in rats and dogs, effects were typical of cholinesterase inhibitors and included decreased cholinesterase activity in plasma (dogs and rats), erythrocytes and brain (rats only), at doses of 0.05 mg/kg bw/day and above (rats). Mortality was seen at doses of 0.1 mg/kg bw/day and above (rats). No other effects were seen (in both rats and dogs). The lowest overall NOEL was 0.02 mg/kg bw/day (rats).

In long-term dietary studies in rats and dogs, effects were typical of cholinesterase inhibitors and included plasma and erythrocyte cholinesterase inhibition at the lowest dose tested, 0.024 mg/kg bw/day (dogs), and above. Neuromuscular disturbances, hair loss, decreased bodyweight gain and associated decreases in food consumption, were seen at the highest dose tested in rats, 1.87 mg/kg bw/day. The NOEL in rats was 0.05 mg/kg bw/day. A NOEL was not found in the long-term dietary study in dogs.

Carcinogenicity: Based on long-term studies in rats, there is no evidence of carcinogenicity for aldicarb.

Genotoxicity: Aldicarb and its metabolites are not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats, and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Neurotoxicity: Reduced grip strength, inactivity, and delayed sensorimotor responses of offspring were seen in oral dosing studies in pregnant female rats given 0.3 mg/kg bw/day from gestation day 6 to lactation day 10 (23-26 days). The NOEL for effects on the developing nervous system was 0.1 mg/kg bw/day.

Poisons Schedule: Aldicarb is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.004 mg/L for aldicarb was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.01 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.01 mg/kg bw/day is the NOEL based on an acute human study.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated amount maximum of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from human studies to account for intraspecies variation.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Baier JH, Lykins BW, Fronk CA, Kramer SJ (1987). Using reverse osmosis to remove agricultural chemicals from groundwater. *Journal of the American Water Works Association*, 79(8):55-60.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Health Canada (1995). Guidelines for Canadian Drinking Water Quality – Technical Documents: Aldicarb. http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/aldicarb-aldicarbe/aldicarb-aldicarbe-eng.pdf

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction. *Water Research*, 33:(5):1238-1246.

Kiso Y, Mizuno A, Othman RA, Jung Y, Kumano A, Ariji A (2002). Rejection properties of pesticides with a hollow fiber NF [nanofiltration] membrane (HNF-1). *Desalination*, 143(2):147-157.

Kruithof JC, Hopman R, Meijers RT, Hofman J (1994). Presence and removal of pesticides in Dutch drinking water practice. *Water Supply*, 12(1-2): SS11-10-SS11-14.

Mason Y, Choshen E, Rav-Acha C (1990). Carbamate insecticides: removal from water by chlorination and ozonation. *Water Research*, 24(1):11-21.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (2003) Background documents for the development of WHO Drinking Guidelines for Drinking-water Quality. Aldicarb in drinking water. WHO/SDE/WSH/03.04/72.

NOTE: Important general information is contained in PART II, Chapter 6

Aldrin and Dieldrin

(endorsed 2011)

GUIDELINE

Based on human health concerns, aldrin and dieldrin when measured together in drinking water should not exceed 0.0003 mg/L.

RELATED CHEMICALS

Aldrin and dieldrin (CAS 309-00-2/CAS 60-57-1) belong to the organochlorine class of chemicals and are classified as persistent organic pollutants (POP). Other POPs that were previously used as pesticides include DDT and heptachlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present as contaminants in drinking water, aldrin and dieldrin when measured together would not be a health concern unless the concentration exceeded 0.0003 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Aldrin and dieldrin have been used previously as insecticides for the control of soil-dwelling pests and for the protection of wood structures against termites and wood borers.

There are no registered products that contain aldrin or dieldrin in Australia, but de-registered compounds may still be detected in water.

Exposure sources: Aldrin is largely converted to dieldrin, which persists in the environment. The general public may be exposed to low levels of dieldrin through residues in food and/or contaminated source waters from previous use of aldrin and dieldrin as insecticides. The residue definition of aldrin and dieldrin is defined as “sum of HHDN (aldrin) and HEOD (dieldrin)”.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data of aldrin or dieldrin in Australian drinking water could be found. The concentrations of aldrin and dieldrin in aquatic environments and drinking water in industrialised countries are normally less than 0.01 µg/L (WHO 2003).

TREATMENT OF DRINKING WATER

Aldrin is completely removed by either chlorination or activated carbon, while dieldrin is more recalcitrant, with removal efficacies of 30% for chlorination and 85% for activated carbon (Ormad *et al.* 2008).

MEASUREMENT

Aldrin and dieldrin are determined by extraction with pentane followed by gas chromatography with electron capture detection. The detection limits in tap water and river water are about 0.001 µg/L for aldrin and 0.002 µg/L for dieldrin (WHO 2003).

HISTORY OF THE HEALTH VALUES

The current tolerable daily intake (TDI) for both aldrin and dieldrin is 0.0001 mg per kg bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.025 mg/kg bw/day from 2-year dietary studies in rats and dogs. The NOEL is based on liver damage. The TDI incorporates a safety factor of 250 and was established in 2003

When aldrin and dieldrin were in use in Australia, the ADI was 0.0001 mg/kg bw, based on a NOEL of 0.025 mg/kg bw/day from the long-term dietary studies. As aldrin and dieldrin are no longer used in agricultural practice in Australia, the ADI was not maintained.

The previous *Australian Drinking Water Guidelines* health value for aldrin and dieldrin was 0.0003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Since aldrin is converted to dieldrin in the environment, the toxicity of dieldrin is the major consideration in relation to the health impact of aldrin and/or dieldrin in drinking water.

Metabolism: Aldrin and dieldrin are rapidly absorbed via the gastrointestinal tract in animals and humans. Aldrin is rapidly metabolised to dieldrin, and dieldrin is further metabolised, mainly in the liver. Aldrin and dieldrin accumulate in adipose tissue, from which they can be mobilised into blood. Some excretion of both compounds occurs, mainly as metabolites via urine and faeces. The major metabolite of dieldrin identified is the 9-hydroxy derivative.

Acute effects: Both aldrin and dieldrin have high acute oral and dermal toxicity. Skin sensitisation data are not available for aldrin or dieldrin.

Short-term effects: Short-term dietary studies in rats with both aldrin and dieldrin indicated an increase in relative liver weight and reversible hypertrophy of hepatocytes at dose levels above 5 mg/kg bw/day. In dogs, liver weight changes were observed at 0.05 mg/kg bw/day and muscular spasms and convulsions at high dose levels. In a two-year human volunteer study, no clinical signs of toxicity were reported up to doses of 0.2 mg/kg bw/day.

Long-term effects: Long-term dietary studies in mice, rats and dogs showed the liver to be the target organ of toxicity. In mice, there was liver enlargement and hyperplasia, and benign and malignant liver tumours at doses of 0.187 mg/kg bw/day and above. Clinical signs of toxicity, which included hair loss, diarrhoea, hyper-excitability, abdominal distention and tremors, were reported at doses of 0.5 mg/kg bw/day and above. Two-year dietary studies in rats and dogs reported by the Joint Meeting of Pesticide Residues (JMPR 1966, 1970, 1977) showed increased liver weights and microscopic liver lesions in rats at 0.1 mg/kg bw/day and above, and increased liver weight and liver damage at 0.075 mg/kg bw/day in dogs. The NOEL from these studies was 0.025 mg/kg bw/day, and this is the basis for the current Australian TDI.

Carcinogenicity: There was evidence of liver tumors in mice, but not rats, trout or hamsters. In epidemiological studies in workers, there was no evidence of an increased incidence of cancer related to exposure to aldrin and dieldrin.

Genotoxicity: Aldrin and dieldrin are not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: Reproductive studies in mice, rats and dogs up to 6 generations reported an increase in foetotoxicity and pre-weaning pup mortality at 0.125 mg/kg bw/day and above. Developmental toxicity studies in mice, rats and rabbits did not produce any evidence of effects on foetal development.

Immunotoxicity: There was no evidence of immunotoxicity based on studies in mice.

Poisons Schedule: Aldrin and dieldrin are included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.0003 mg/L for aldrin and dieldrin was determined as follows:

$$0.0003 \text{ mg/L} = \frac{0.025 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 250}$$

where:

- 0.025 mg/kg bw/day is the NOEL based on long-term (2-year) studies in rats and dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 250 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation and an additional safety factor of 2.5 based on concern about carcinogenicity observed in mice.

The World Health Organization established a health-based guideline value of 0.00003 mg/L for aldrin and dieldrin in 2003 (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

JMPR (Joint FAO/WHO Meeting on Pesticide Residues) (1966). Toxicological evaluation of aldrin (FAO/PL:CP/15).

JMPR (Joint Meeting on Pesticide Residues) (1970). Toxicological evaluation of dieldrin (AGP:1970/M/12/1).

JMPR (Joint Meeting on Pesticide Residues) (1977). Toxicological evaluation of aldrin/dieldrin (Pesticide residues in food: 1977 evaluations)

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71: 97-106.

NOTE: Important general information is contained in PART II, Chapter 6

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2003). Aldrin and Dieldrin in Drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality. The World Health Organization.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Aluminium

(endorsed 2001)

GUIDELINE

Based on aesthetic problems caused by post-flocculation, the concentration of acid-soluble aluminium in drinking water should not exceed 0.2 mg/L. Water authorities are strongly encouraged to keep acid-soluble aluminium concentrations as low as possible, preferably below 0.1 mg/L.

No health-based guideline is set for aluminium at this time but this issue will be kept under review.

GENERAL DESCRIPTION

Aluminium may be present in water through natural leaching from soil and rock, or from the use of aluminium salts as coagulants in water treatment.

Aluminium is used in many industrial and domestic products including antacids, antiperspirants and food additives, and in vaccines. It is commonly used by the food industry for food containers and packaging, and many cooking utensils are made from aluminium.

Surveys in the United States and the United Kingdom have reported aluminium concentrations in natural water sources of 0.014–1.2 mg/L. Concentrations in some Australian water sources can be considerably higher due to the presence of clay minerals (aluminosilicates); for example, up to 18 mg/L in the Murray River. Residual aluminium concentrations in treated water depend on the concentration in the water source, the alum dose used, the pH, and the filtration efficiency.

Where alum is used as a coagulant in water treatment, post-flocculation effects can occur if the soluble aluminium concentration in the treated water exceeds 0.2 mg/L. Depending on pH, a whitish gelatinous precipitate of aluminium hydroxide can be formed in the distribution system which may result in customer complaints about 'milky coloured' water. Aluminosilicates in source water are very insoluble and do not cause post-flocculation problems.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, the concentration of aluminium varies from 0.01 mg/L to 0.9 mg/L, with typical concentrations of approximately 0.1 mg/L for fully treated supplies.

TREATMENT OF DRINKING WATER

Aluminium concentrations in drinking water can be reduced using the conventional water treatment practices of flocculation and filtration. A well-operated water filtration plant (even using aluminium as a flocculant) can achieve aluminium concentrations in the finished water of less than 0.1 mg/L.

MEASUREMENT

The term 'soluble' should be taken to mean truly soluble, not 'filterable through a 0.45 µm pore size filter'. Finely suspended aluminosilicate clay particles can pass through a 0.45 µm filter but are not truly soluble and will not cause post-flocculation problems.

Acid-soluble aluminium is determined after acidifying the sample to pH 1.5–2, followed by filtration through a 0.45 µm membrane filter. If analysis of the filtrate by the normal method (e.g. graphite furnace atomic absorption spectroscopy, APHA Method 3500-Al Part B 1992) gives a result above the guideline value, the filtrate should be re-analysed using the catechol violet colorimetric method (APHA Method 3500-Al Part E 1992), which provides a better estimate of the reactive aluminium component. The limit of determination for the latter method is approximately 0.01 mg/L.

Based on experience with their water supplies, authorities may choose to monitor total aluminium concentration and perform specific assays for acid-soluble aluminium only if total aluminium concentration exceeds 0.1 mg/L.

HEALTH CONSIDERATIONS

It has been estimated that for Australian adults, the intake of aluminium from food and beverages is approximately 5–7 mg/day. Drinking water contributes less than 2% of the total daily intake, and only 0.3–0.4% of the aluminium in water is absorbed by the body. Recent studies have shown that the bioavailability (i.e. uptake into the bloodstream) of aluminium in drinking water is similar to that of food (Stauber *et al*. 1999).

The metabolism of aluminium in humans is poorly understood. Studies indicate that less than 1% of dietary aluminium is absorbed by the gastrointestinal tract, with the remainder excreted in faeces. The small amount absorbed passes into the blood stream. Some aluminium accumulates in bone, liver and brain tissue but most is removed from the blood stream by the kidneys and excreted. In healthy adults, the total accumulated body load of aluminium has been estimated at about 35 mg. Whether this remains constant with age has not been determined.

There is considerable evidence that aluminium is neurotoxic. Kidney dialysis patients, in whom the gut barrier is bypassed, can accumulate aluminium in their blood resulting in an encephalopathy known as dialysis dementia. Investigations have established a correlation between the concentration of aluminium in water used to prepare dialysis fluid and the incidence of dialysis dementia. If this condition is not too far advanced it responds to chelation therapy. It appears that dialysis patients are much more susceptible to aluminium in dialysis fluid than from other sources such as food and antacids. Aluminium has also been linked to other conditions associated with the use of dialysis units including osteomalacia (a softening of the bones) and anaemia. Reverse osmosis or deionisation units are now used to treat dialysis water before use, and aluminium concentrations are kept below 0.01 mg/L.

Aluminium has been associated with two severe neurodegenerative diseases: Parkinsonism dementia (PD) and amyotrophic lateral sclerosis (ALS). Both conditions have a high incidence amongst the Chamorro people of Guam, an area where aluminium is naturally present in food and drinking water. ALS is common in the Pacific, Western New Guinea and the Kii peninsula of Japan. Both PD and ALS are characterised by loss of motor function and the presence of neurofibrillary tangles in the brain. One hypothesis suggests that chronic nutritional deficiencies of calcium and magnesium lead to increased absorption of aluminium, resulting in its deposition in neurons of the brain (Garruto and Yase 1986, Garruto *et al*. 1990). There was an appreciable decrease in the incidence of these conditions when the areas became westernised, with associated changes in dietary habits, importing of food and improvements to the water supply.

Elevated concentrations of aluminium have been found in the autopsied brains of people who had suffered Alzheimer's disease, in regions of the brain containing large numbers of the neurofibrillary tangles which are characteristic of the disease, and aluminium has been proposed as one of a number of causal agents (Perl and Brody 1980). There have been a number of epidemiological studies to determine if aluminium in drinking water plays a role in Alzheimer's disease. Although some studies indicated that a tentative link may exist, more recent evidence (Martyn *et al*. 1997) suggests that aluminium in drinking water is not associated with increased risk of Alzheimer's disease.

NOTE: Important general information is contained in PART II, Chapter 6

A number of animal studies of aluminium toxicity have been undertaken although there has been very little research done using aged animals. Most studies have used rats fed or injected with large amounts of aluminium and have reported only minor changes to bodyweight, with some behavioural changes and locomotor effects. Elevated concentrations of aluminium have been reported in the brain, liver and kidneys. The studies are not adequate to set a reliable no observable effect level (NOEL).

Aluminium is not generally thought to be mutagenic or genotoxic, although aluminium has been shown to bind to DNA of a number of animal species and has displayed mutagenic activity in some, but not all, tests using bacteria. Ingestion of aluminium is not known to cause cancer in humans or animals.

The NHMRC Standing Committee on Toxicity has reviewed the toxicological data for aluminium and concluded that there are insufficient data to set a NOEL.

DERIVATION OF GUIDELINE

Post-flocculation problems (described above) associated with the use of alum as a coagulant may occur if acid-soluble aluminium exceeds 0.2 mg/L. As the alum floc is soluble in dilute acid (pH 1.5–2), post-flocculation problems will generally be avoided if the acid-soluble concentration of aluminium is below 0.2 mg/L. Water authorities are strongly encouraged to keep acid-soluble aluminium concentrations as low as possible, preferably below 0.1 mg/L. Well operated water filtration plants, even those using aluminium salts as flocculants, should have little difficulty in achieving this.

A guideline value lower than 0.2 mg/L may need to be adopted by some water authorities, depending on the amount of naturally occurring organic material in the water.

Although data are insufficient to set a guideline value based on health considerations, there is public concern over the possible health effects of aluminium. This issue should be reviewed when further studies are undertaken.

REFERENCES

APHA Method 3500-Al Part B (1992). Aluminium: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Al Part E (1992). Aluminium: Automated Pyrocatechol violet method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Garruto RM, Yase Y (1986). Neurodegenerative disorders of the Western Pacific: the search for mechanisms of pathogenesis. *Trends in Neurosciences*, 9:368–374.

Garruto RM, Yanagihara R, Gajdusek DC (1990). Models of environmentally induced neurological disease: epidemiology and etiology of amyotrophic lateral sclerosis and parkinsonism-dementia in the Western Pacific. *Environmental Geochemistry and Health*, 12:137–151.

Martyn CN, Osmond C, Edwardson JA, Barker DJP, Harris EC, Lacey RF (1989). Geographical relation between Alzheimer's disease and aluminium in drinking water. *Lancet* (January):59–62.

Perl DP, Brody AR (1980). Alzheimer's disease: X-ray spectrometric evidence of aluminium accumulation in neurofibrillary tangle-bearing neurons. *Science*, 208:297–299.

Stauber JL, Florence TM, Davies CM, Adams MS, Buchanan SJ (1999). Bioavailability of aluminium in alum-treated drinking water. *Journal American Water Works Association*, 91:84–93.

NOTE: Important general information is contained in PART II, Chapter 6

Ametryn

(endorsed 2011)

GUIDELINE

Based on human health concerns, ametryn in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Ametryn (CAS 834-12-8) belongs to the triazine class of chemicals. Other pesticides in this class include atrazine, propazine, symazine and cyanazine (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, ametryn would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Ametryn is a pre- and post-emergent herbicide for the control of summer grasses and broad-leaf weeds in sugarcane, pineapples and in industrial areas, such as roadsides and railway lines.

There are registered products containing ametryn in Australia. These products are intended for professional use and are formulated as a liquid concentrate or a water dispersible granule, either alone or with other active ingredients. These products are applied using ground boom, aerial and hand-held methods of spraying. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to ametryn is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of ametryn may potentially lead to contamination of sources waters through processes such as run-off, spray-drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Ametryn has been reported in Australian source waters at concentrations up to 0.3 µg/L in sugar cane growing areas (Mitchell *et al*, 2004). Similar results have been reported in drinking source waters of a sugar cane growing area of Brazil (Lanchote *et al*, 2000).

TREATMENT OF DRINKING WATER

Ametryn has been shown to be completely removed from water by chlorination when the chlorine dose is adjusted to match chlorine demand (Ormad *et al.* 2008).

Ozonation and activated carbon adsorption for ametryn removal has also been reported with moderate to low success (Ormad *et al.* 2008). Conventional coagulation/flocculation has been shown to be unreliable for removal, although complete removal can be obtained if activated carbon adsorption is practiced in conjunction with conventional clarification. Jar testing with different oxidants, adsorbents, coagulants, dose rates and contact times is recommended to optimise removal if ametryn is detected.

MEASUREMENT

Ametryn can be measured in drinking waters using solid phase extraction followed by high performance liquid chromatography with ultraviolet detection (Carabias-Martinez *et al.* 2006). The practical limit of detection for this method is 1 μg/L. Alternatively, ametryn can be monitored by direct aqueous injection liquid chromatography with electrospray ionisation–tandem mass spectrometry, with a reported detection limit of 0.05 μg/L (Huang *et al.* 2008).

HISTORY OF THE HEALTH VALUES

The acceptable daily intake (ADI) for ametryn is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2 mg/kg bw/day from a reproduction study in rats. The ADI incorporates a safety factor of 100 and was established in 2006.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Ametryn is absorbed readily from the gastrointestinal tract and is rapidly excreted (89% in urine and faeces over 7 days). Thirty-five metabolites were identified in the urine and faeces in the rat.

Acute effects: Ametryn has low acute oral and dermal toxicity. It is a skin sensitiser in guinea pigs. Symptoms associated with acute toxicity were dyspnoea, ruffled fur, diarrhoea, sedation, and curved body posture.

Short-term effects: Short-term dietary studies in rats and a 21-day dermal study in rabbits showed transient decreases in body weight and food consumption at dose levels of 1000 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in rats and dogs. A 2-year dietary study in rats reported anaemia and evidence of liver damage at dose levels of 2.2 mg/kg bw/day. A one-year study in dogs reported anaemia and degenerative changes in a number of organs at dose levels above 8 mg/kg bw/day.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for ametryn.

Genotoxicity: Ametryn is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproduction and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development. There was a transient decrease in bodyweight gain at 2 mg/kg bw/day, which was the overall NOEL. This is the basis for the ADI.

Poisons Schedule: Ametryn is included in Schedule 5 of the *Standard for the Uniform Scheduling of*

NOTE: Important general information is contained in PART II, Chapter 6

Medicines and Poisons No.1, 2010 (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for ametryn was determined as follows:

$$0.07 \text{ mg/L} = \frac{2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2 mg/kg bw/day is the NOEL based on a reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Carabias-Martinez R, Rodriguez-Gonzalo E, Miranda-Cruz E, Dominguez-Alvarez J, Hernandez-Mendez J (2006). Comparison of a non-aqueous capillary electrophoresis method with high performance liquid chromatography for the determination of herbicides and metabolites in water samples. *Journal of Chromatography A*, 1122(1-2):194-201.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Huang, S. B., Mayer, T. J. and Yokley, R. A. (2008) Direct aqueous injection LC-ESI/MS/MS analysis of water for 11 chloro- and thiomethyltriazines and metolachlor and its ethanesulfonic and oxanilic acid degradates. *Journal of Agricultural and Food Chemistry*, 56(8): 2595-2602.

Lanchote VL, Bonato PS, Cerdeira AL, Santos NAG, de Carvalho D, Gomes MA (2000). HPLC screening and GC-MS confirmation of triazine herbicides residues in drinking water from sugar cane area in Brazil. *Water Air and Soil Pollution*, 118(3-4):329-337.

Mitchell, C., Brodie, J. and White, I. (2004) Sediments, nutrients and pesticide residues in event flow conditions in streams of the Mackay Whitsunday Region, Australia. In: *Conference for Reef Research Townsville,* Australia, pp. 23-36.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71: 97–106.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Amitraz

(endorsed 2011)

GUIDELINE

Based on human health concerns, amitraz in drinking water should not exceed 0.009 mg/L.

RELATED CHEMICALS

Amitraz (CAS 33089-61-1) is in the amidine class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, amitraz would not be a health concern unless the concentration exceeded 0.009 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Amitraz is an acaricide (miticide) and insecticide used for the control of heliothis and aphids on cotton; for the control ticks on cattle, deer, sheep, goats, circus animals and dogs; and for the control of mange in pigs.

There are registered products containing amitraz in Australia. These are intended for both professional and home veterinary use. The products are applied by ground spray or aircraft when used on cotton. They are used as a spray or in a pour-on formulation when used on livestock. Amitraz is also incorporated into a collar or a shampoo for use on dogs. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure are use of the home veterinary products, and residues in the food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of amitraz may potentially lead to contamination of source waters through processes such as run-off, spray-drift or entry into groundwater. The veterinary use of amitraz provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of amitraz in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of amitraz in drinking water have been identified.

MEASUREMENT

No suitable analytical methods for amitraz in drinking water have been identified. If there is an identified need to monitor amitraz in drinking water, it is expected that gas-chromatography–mass spectrometry and high performance liquid chromatography–mass spectrometry would be suitable techniques. Both of these approaches have previously been used for monitoring amitraz in food products such as fruit and honey.

HISTORY OF THE HEALTH VALUES

The acceptable daily intake (ADI) for amitraz is 0.002 mg per kg of bodyweight (mg/kg bw) based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day from a 2-year study in dogs. The ADI incorporates a safety factor of 100 and was established in 1986.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Amitraz is absorbed rapidly from the gastrointestinal tract of humans, and is metabolised and excreted mainly in the urine within 72 hours. The main metabolites are 4-formamido-3-methyl benzoic acid and 4-acetamido-3-methyl benzoic acid. In dogs, peak blood levels were seen at 8 hours and 80% of the dose was excreted within 24 hours.

Acute effects: Amitraz has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: *S*hort-term dietary studies in mice indicated liver toxicity at 3 mg/kg bw/day and above.

Long-term effects: A 2-year dietary study in rats showed an increase in nervous and aggressive behaviour at 10 mg/kg bw/day. A 2-year study in dogs showed central nervous system depression on days 1 and 2 of treatment. There was also an increase in the blood glucose levels at doses above 0.25 mg/kg bw/day. The NOEL of 0.25 mg/kg bw/day is the basis for the ADI.

Carcinogenicity: An increase in hepatocellular adenomas and carcinomas was observed in female B6C3F1 mice at 60 mg/kg bw/day. This is considered to be a species-specific effect and not relevant to humans.

Genotoxicity: Amitraz is not considered genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats showed decreased bodyweight gain and reduced fertility in the dams together with decreased pup survival at 21 days in all generations treated at 50 mg/kg bw/day. A 28-week study in mice reported hormonal changes and effects on the oestrus cycle at 14.3 mg/kg bw/day. In developmental studies in rats and rabbits, there was evidence of foetal toxicity at dose levels of 12 and 25 mg/kg bw/day, respectively, but no evidence of teratogenicity.

Poisons Schedule: Amitraz is included in Schedule 6 of the of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.009 mg/L for amitraz was determined as follows:

$$0.009 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.25 mg/kg bw/day is the NOEL based on a long-term (2-year) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variations.

Amitraz is included in the World Health Organization guidelines for drinking water quality list of chemicals from agricultural activities excluded from guideline value derivation because it is "degrades rapidly in the environment and is not expected to occur at measurable concentrations in drinking-water supplies" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Amitrole

(endorsed 2011)

GUIDELINE

Based on human health concerns, amitrole in drinking water should not exceed 0.0009 mg/L.

RELATED CHEMICALS

Amitrole (CAS 61-82-5) belongs to the triazole class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, amitrole would not be a health concern unless the concentration exceeded 0.0009 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Amitrole is a herbicide used to kill weeds in public places and home gardens.

There are registered products containing amitrole in Australia. These are for professional and home garden use. Amitrole is used alone or in combination with other herbicides as concentrated solutions or as wettable powders, and these are generally applied as a spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to amitrole is direct exposure following home garden use. Amitrole is not currently registered for use on food crops and the maximum residue limits are set at the level of detection.

Use of amitole in public places and in the home garden may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATERS

Amitrole is a persistent herbicide that can easily pollute ground and surface waters used in drinking water production. No Australian data was found but concentrations in ground and surface water of up to 1.1 µg/L have been reported (Bobeldijk *et al.* 2001).

TREATMENT OF DRINKING WATER

Amitrole has been shown to be effectively removed by ozonation (Bozkaya-Schrotter *et al.* 2008). Moderate removal can also be achieved using powdered activated carbon adsorption (Lopez-Ramon *et al.* 2007).

MEASUREMENT

Numerous analytical methods have been described for the detection of amitrole. Gas chromatography can achieve a limit of detection of 0.1 µg/L in drinking water (Pachinger *et al.* 1992). High-performance liquid chromatographic (HPLC) methods are currently the analytical techniques of choice for amitrole and other polar compounds. Different HPLC methods for the determination of amitrole after derivatisation have been described. HPLC with electrochemical detection can achieve limits of detection of 0.1 µg/L. Solid-phase extraction followed by HPLC with atmospheric pressure chemical ionisation–tandem mass spectrometry can achieve a limit of detection of 0.025 µg/L in drinking water (Bobeldijk *et al.* 2001).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for amitrole is 0.0003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.025 mg/kg bw/day from a 1-year rat study. The ADI incorporates a safety factor of 100 and was established in 1984.

An acute reference dose has not been established for amitrole.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC, 2004).

HEALTH CONSIDERATIONS

Metabolism: Amitrole is readily absorbed through the gastrointestinal tract. It undergoes minimal metabolism and is excreted largely unchanged in the urine.

Acute effects: Amitrole has a low acute oral and dermal toxicity. Its potential for skin sensitisation is unknown.

Short-term effects: Short-term oral toxicity studies in rats reported histopathological changes in the thyroid and changes to the uptake of iodine by the thyroid at 20 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies were conducted in mice and rats. In mice, there were effects on thyroid weight and uptake of iodine at 15 mg/kg bw/day. In rats, there was a decrease in thyroid hormone levels, an increased incidence of thyroid hyperplasia and an increase in thyroid tumours at 2.5 mg/kg bw/day. The NOEL in the 1-year rat study was 0.025 mg/kg bw/day, and this is the basis of the ADI.

Carcinogenicity: In long-term studies in rats, amitole caused an increase in thyroid tumours, which is likely to be a consequence of an increased incidence of thyroid hyperplasia.

Genotoxcity: Amitole gave equivocal results in some *in vitro* studies, but negative results in *in vivo* studies. Based on the weight of evidence, amitole was not considered genotoxic.

Reproductive and developmental effects: In a reproduction study in rats and development studies in mice and rats, there was no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Amitrole is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.0009 mg/L for amitole was determined as follows:

$$0.0009 \text{ mg/L} = \frac{0.025 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.025 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bozkaya-Schrotter B, Daines C, Lescourret A, Bignon A, Breant P, Schrotter J (2008). Treatment of trace organics in membrane concentrates I: pesticide elimination. *Water Science and Technology: Water Supply*, 8(2):223-230.

Bobeldijk I, Broess K, Speksnijder P, van Leerdam T (2001). Determination of the herbicide amitrole in water with pre-column derivatisation, liquid chromatography and tandem mass spectrometry. *Journal of Chromatography A,* 938(1-2):15-22.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Lopez-Ramon MV, Fontecha-Camara MA, Alvarez-Merino MA, Moreno-Castilla C (2007). Removal of diuron and amitrole from water under static and dynamic conditions using activated carbons in form of fibers, cloth, and grains. *Water Research*, 41(13):2865-70.

Pachinger A, Eisner E, Begutter H, Klus H (1992). A simple method for the determination of amitrole in drinking and ground water. *Fresenius' Journal of Analytical Chemistry*, 342:413-415.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Ammonia

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (corrosion of copper pipes and fittings), the concentration of ammonia (measured as ammonia) in drinking water should not exceed 0.5 mg/L.

No health-based guideline value is set for ammonia.

GENERAL DESCRIPTION

Ammonia dissolves rapidly in water to form an equilibrium mixture of free ammonia and the ammonium cation. It may be present in unchlorinated drinking water due to contamination of source water or through microbial metabolism. Ammonia is used in conjunction with chlorine to form chloramines to disinfect water supplies. Some residual will be present in the water, particularly if the chlorinator is not operating properly.

Ammonia is used commercially in animal feeds and fertilisers, and in the manufacture of fibres, plastics and explosives. Ammonia products are widely used as cleaning agents and food additives.

Most uncontaminated source waters have ammonia concentrations below 0.2 mg/L. High concentrations (greater than 10 mg/L) have been reported where water is contaminated with animal waste. Ammonia is unlikely to be detected in chlorinated supplies as it reacts quickly with free chlorine.

Ammonia in water can result in the corrosion of copper pipes and fittings, causing copper stains on sanitary ware. It is also a food source for some microorganisms, and can support nuisance growths of bacteria and algae, often with a resultant increase in the nitrite concentration.

The odour threshold of ammonia in water is 1.5 mg/L.

Ammonia can be an important indicator of pollution as it can be formed as an intermediate product in the breakdown of nitrogen-containing organic compounds, or of urea from human or animal excrement.

Food can contain substantial amounts of ammonia/ammonium and is the principal source of intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies concentrations of ammonia range up to 0.4 mg/L, but are generally less than 0.02 mg/L.

TREATMENT OF DRINKING WATER

Ammonia concentrations in drinking water supplies can be reduced by chemical or biological oxidation of ammonia to nitrate.

MEASUREMENT

The concentration of ammonia in water can be determined by a number of methods including colorimetric, titrimetric and potentiometric techniques. For determination of low concentrations, the phenate colorimetric method is commonly used (APHA 4500-NH_3 Parts D or H, 1992). The limit of determination for this method is 0.02 mg/L. Alternatively, the ammonia selective electrode method can be used (APHA 4500-NH_3 Part F, 1992) with a limit of determination of 0.03 mg/L.

Both of these methods determine the total free ammonia and ammonium ion measured as ammonia (NH_3).

HEALTH CONSIDERATIONS

Ammonia is an important metabolite in humans and animals. It is formed in the liver by the deamination of amino acids, and in the gastrointestinal tract by the breakdown of food by enzymes and bacterial flora.

Only an extremely small proportion of the ammonia absorbed in the intestinal tract originates directly from food or water. The major part is formed in the gut as a by-product of the breakdown of food. Almost all ammonia is absorbed. It is then transported to the liver and used mostly in the urea cycle.

An extensive review and summary of the human and animal toxicity data for ammonia is available (IPCS 1986).

Ammonia has a toxic effect on humans only if the intake becomes higher than the detoxification capacity of the body. At doses above 32 mg ammonium per kilogram body weight per day (over 1000 mg/L) ammonium chloride influences the metabolism by shifting acid-base equilibrium, affecting glucose tolerance and reducing tissue sensitivity to insulin.

In studies with animals, high doses of ammonia (over 100 mg/kg body weight per day) have generally not produced any significant toxic effects. Ammonium hydroxide did not result in an increase in the incidence of cancer when given to mice in their drinking water over a lifetime; however, there is some evidence that ammonia may act with cancer-causing compounds to increase the incidence of tumours.

Ammonia and ammonium chloride have shown mutagenicity in some tests with bacteria and animal cells.

DERIVATION OF GUIDELINE

Ammonia concentrations above 0.5 mg/L may attack copper pipes and fittings, or result in nuisance growths of microorganisms. Concentrations of ammonia that may cause health effects are unlikely to occur in drinking water supplies; accordingly, no health-based guideline is set.

REFERENCES

APHA Method 4500-NH_3 Part D (1992). Nitrogen (ammonia): Phenate method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 4500-NH_3 Part F (1992). Nitrogen (ammonia): Ammonia-selective electrode method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IPCS (International Programme on Chemical Safety) (1986). Ammonia. *Environmental Health Criteria*, 54. World Health Organization, IPCS.

NOTE: Important general information is contained in PART II, Chapter 6

Antimony

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of antimony in drinking water should not exceed the limit of determination of 0.003 mg/L.

GENERAL DESCRIPTION

Antimony, as the trivalent (Sb(III)) or pentavalent (Sb(V)) salts, has occasionally been detected in natural source waters. Occurrences are more common in areas near lead or copper smelting operations. Antimony–tin solder is beginning to replace lead solder and hence exposure to antimony in drinking water may increase in the future.

Antimony alloys and compounds are used in semiconductors, batteries, anti-friction compounds, ammunition, cable sheathing, and flame-proofing compounds. Antimony salts are used in glass, and in the manufacture of ceramics and pottery.

Studies overseas have generally found low concentrations in drinking water, typically less than 0.005 mg/L, but higher concentrations have been reported occasionally.

There are few data available on antimony concentrations in food. The United States Agency for Toxic Substances and Disease Registry has suggested that average daily consumption of antimony in food is about 0.018 mg.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for antimony.

TREATMENT OF DRINKING WATER

There are no published methods for removal of antimony from drinking water.

MEASUREMENT

The concentration of antimony in drinking water can be determined by hydride generation followed by analysis using atomic absorption spectroscopy. The limit of determination is approximately 0.001 mg/L. Alternatively, graphite furnace atomic absorption spectroscopy can be used with a limit of determination of 0.003 mg/L (APHA Method 3500-Sb Part B 1992).

HEALTH CONSIDERATIONS

Studies using rats have shown that about 15% of antimony is absorbed by the gastrointestinal tract. It is distributed mainly to the liver, spleen and heart, and to the thyroid and adrenal glands, and it is excreted in faeces and urine.

There have been a number of studies on the effects of long-term human exposure to antimony. One early study of adult male workers in an antimony smelter reported no adverse effects after persistent exposure over periods from 2 to 13 years. A more recent study where workers were exposed for 9 to 31 years to dust containing antimony trioxide and antimony pentoxide reported respiratory and eye problems as well as staining of the front tooth surface. A dermatitis condition was observed in more than half of the exposed workers. Other studies have reported heart irregularities, lung cancer, and

spontaneous abortions among female workers.

The toxicity of antimony to animals varies considerably depending on the compound used. No adverse effects have been associated with long-term exposure of rats to antimony trioxide. However, potassium antimony tartrate reduced the animals' lifespan, and antimony was found to accumulate in the heart, liver, kidney and spleen. It also affected blood glucose and cholesterol concentrations.

Animal studies have shown that antimony can cross the placenta. It may cause sterility, fewer offspring, and foetal damage.

Studies using male and/or female rats have reported that inhalation of concentrates of antimony trioxide and antimony ore increased the incidence of lung tumours in females. In ingestion studies on rats and mice, antimony did not appear to cause tumours.

Trivalent and pentavalent antimony salts have demonstrated mutagenic activity in tests with bacteria. They also induced chromosome aberrations in cultured mammalian cells.

The International Agency for Research on Cancer has concluded that antimony trioxide is possibly carcinogenic to humans by the inhalation route (Group 2B, inadequate evidence in humans, sufficient evidence in animals); and antimony trisulfide is not classifiable as to its carcinogenicity to humans (Group 3, inadequate evidence in humans and limited evidence in animals) (IARC 1989).

DERIVATION OF THE GUIDELINE

The guideline value of 0.003 mg/L for antimony in drinking water was derived as follows:

$$0.003 \text{ mg/L} = \frac{0.43 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 500}$$

where:

- 0.43 mg/kg body weight per day is the lowest effect level based on decreased lifespan and altered blood levels of glucose and cholesterol in a lifetime study using rats (Schroeder ***et al***. 1970).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 500 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 5 because a lowest effect level was used instead of a no-effect level).

The WHO guideline value of 0.005 mg/L was derived from this calculation but rounded up.

REFERENCES

APHA Method 3500-Sb Part B (1992). Antimony: Atomic Absorption Spectrophotometric Method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1989). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: some organic solvents, resin monomers and related compounds, pigments and occupational exposures in paint manufacture and painting. World Health Organization, IARC, 47, Lyons.

Schroeder HA, Mitchener M, Nason AP (1970). Zirconium, niobium, antimony, vanadium and lead in rats: Lifeterm studies. *Journal of Nutrition*, 100:59–68.

NOTE: Important general information is contained in PART II, Chapter 6

Arsenic

(endorsed 2011)

GUIDELINE

Based on human health considerations, the concentration of arsenic in drinking water should not exceed 0.01 mg/L.

GENERAL DESCRIPTION

Arsenic is a naturally occurring element which can be introduced into water through the dissolution of minerals and ores (where it exists mainly in the sulfide form), or from industrial effluent, atmospheric deposition (through the burning of fossil fuels and waste incineration), drainage from old gold mines, or the use of some types of sheep dip. Natural sources can make a significant contribution to the arsenic concentration in drinking water. Arsenate (i.e. pentavalent (As(V)) is generally the most common form in well oxygenated surface waters and drinking water, but under reducing conditions, such as those found in deep lake sediments or groundwaters, the trivalent form (As(III), arsenite) predominates.

Arsenic compounds have commercial and industrial uses as alloying agents in the manufacture of transistors, lasers and semiconductors, and in the processing of glass, pigments, textiles, paper, metal adhesives, ceramics, wood preservatives, ammunition and explosives. They are also used in the hide-tanning process, and to a limited extent as feed additives, pesticides and pharmaceuticals. Although inorganic forms of arsenic are the most common, organic arsenic compounds are also used.

In surface- and groundwater not affected by arsenic mineral deposits or pollution, the concentration of arsenic is generally less than 0.005 mg/L

Food may be an important source of arsenic intake. The average Australian adult dietary intake of arsenic is approximately 0.04 mg per day. Arsenic is concentrated by many species of fish and shellfish, and is present in poultry and livestock. Concentrations in vegetables are usually an order of magnitude less than those found in fish and meat. It is difficult to make direct comparisons between the arsenic intake from food and water because the form of arsenic and biological availability differ markedly. For example, a major portion of the arsenic in fish is present as highly complexed forms that are biologically unavailable, or as simple organic compounds (arsenobetaine and arsenocholine) that are essentially nontoxic.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In Australian drinking water supplies, typical concentrations of arsenic range from <0.001 mg/L to 0.03 mg/L.

TREATMENT OF DRINKING WATER

Arsenic can be removed from drinking water using conventional coagulation processes. It is desirable to convert trivalent arsenic to the pentavalent form before treatment by oxidation using chlorine or potassium permanganate. Lime softening can also be effective for removal from hard waters, but the efficiency is dependent on pH and valence state. The World Health Organization (WHO) (2003) states: “removal of arsenic to concentrations below 10 µg/litre is difficult in many circumstances,” and Health Canada (2006) indicates that devices designed to remove arsenic from drinking water are certified to perform to 0.01 mg/L (10 µg/L) or less.

MEASUREMENT

The arsenic concentration in drinking water can be determined by hydride generation followed by atomic absorption spectroscopy. The limit of determination is approximately 0.001 mg/L. Alternatively, graphite furnace atomic absorption spectroscopy can be used, with a limit of determination of approximately 0.005 mg/L (APHA Method 3500-As Part B 1992).

HEALTH CONSIDERATIONS

The health considerations apply mainly to inorganic arsenic compounds, as they are more likely than the organic compounds to be present in drinking water supplies.

Soluble arsenic salts are readily absorbed by the gastrointestinal tract. After absorption, inorganic arsenic binds to haemoglobin, and is deposited in the liver, kidney, lungs, spleen, and skin. Inorganic arsenic does not appear to cross the blood-brain barrier but can cross the placenta. Very little ingested arsenic is excreted in faeces, but approximately 45-85% appears in the urine within 1 to 3 days.

Extensive reviews and summaries of the human and animal toxicity data for arsenic are available (IPCS 2001, WHO 2003, IARC 2004, Health Canada 2006, ATSDR 2007). Consumption of elevated levels of arsenic through drinking-water is causally related to the development of cancer at several sites, particularly skin, bladder kidney and lung. Cancer is considered to be the most sensitive toxicity end-point for setting a drinking water guideline for arsenic, however the mechanisms or modes of action by which arsenic causes cancer are yet to be definitively elucidated (WHO 2003).

Inorganic arsenic undergoes sequential reduction and methylation reactions leading to the formation of monomethylarsinic acid (MMA) and dimethylarsinic acid (DMA). This metabolism is regarded as bioactivating arsenic to more toxic forms. *In vitro* and *in vivo* chromosomal and DNA damage by arsenics are dose-dependent, with arsenite more potent than arsenate. Both MMA and DMA are directly genotoxic and are many times more potent than arsenite at inducing DNA damage (ATSDR 2007). Sub-populations that may be at greater risk from arsenic toxicity are those with gene variants of arsenic metabolism that lead to a higher percentage of MMA in the urine (Chen 2009, Smith and Steinmaus 2009).

The International Agency for Research on Cancer has concluded there is sufficient evidence in humans that arsenic in drinking-water causes cancers of the urinary bladder, lung and skin and has classified arsenic in drinking-water as carcinogenic to humans (Group 1) (IARC 2004).

DERIVATION OF GUIDELINE

The European Union (1998), WHO (2004), Health Canada (2008), USEPA (2008), and New Zealand (MoH NZ 2008) have drinking water guidelines for arsenic of 0.01 mg/L. Most of these were based on assessments conducted between 2003 and 2006; the previous Australian DWG was based on a 1988 evaluation. These overseas guidelines were based on considerations of the lowest concentration that is reasonably and economically achievable with water treatment technologies, measurability of arsenic at low concentrations, and lack of observed effects in humans at such low concentrations.

Epidemiological studies show that elevated cancer risks and other adverse health effects are not demonstrable at arsenic concentrations around 0.01 mg/L (e.g. Mazumder *et al.* 1998, Baastrap *et al.* 2008, Celik *et al.* 2008, Chen *et al.* 2009, Smith and Steinmaus 2009). On the other hand, there are many recent studies demonstrating a range of adverse health effects at higher concentrations (>0.05 or 0.1 mg/L) (e.g. Mazumder 2008, Majumdar *et al.* 2009, Mazumder *et al.* 2009, Smith and Steinmaus 2009). These health effects include skin, lung and bladder cancer; skin pigmentation and keratosis; diseases of the lung, liver, peripheral- and cardio-vascular systems; peripheral neuropathy; and diabetes. The majority of affected populations are from Indian, Bangladesh or Asian rural areas. Health Canada (2006) noted that recent epidemiological studies conducted in the United States had not found a clear association between cancer

NOTE: Important general information is contained in PART II, Chapter 6

risks and arsenic in drinking water at levels greater than 0.01 mg/L and below 0.05 mg/L. The study by Baastrap *et al*. (2008) is also informative as it was conducted in populations of similar socio-economic and nutritional status to those in Australia. This was a prospective Danish cohort of 57,053 people that was followed from 1970 to 2003; arsenic drinking water concentrations were up to 0.0253 mg/L (mean 0.0012 mg/L, 99th percentile 0.0057 mg/L), and no association with lung, bladder, liver, kidney, prostate, colorectum, or skin melanoma cancers was found.

After considering the above, a drinking water guideline of 0.01 mg/L was adopted for arsenic.

REFERENCES

APHA Method 3500-As Part B (1992). Arsenic: Atomic Absorption Spectrophotometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

ATSDR (Agency for Toxic Substances and Disease Registry) (2007). Toxicological Profile for Arsenic. ATSDR. PB2008-100002, August 2007.

Baastrup R, Sørensen M, Balstrøm T, Frederiksen K, Larsen CL, Tjønneland A, Overvad K, Raaschou-Nielsen O (2008). Arsenic in drinking-water and risk of cancer in Denmark. *Environmental Health Perspectives,* 116 (2):231-237.

Celik I, Gallicchio L, Boyd K, Lam TK, Matanoski G, Tao X, Shiels M, Hammond E, Chen L, Robinson KA, Caulfield LE, Herman JG, Guallar E, Alberg AJ (2008). Arsenic in drinking water and lung cancer: A systematic review. *Environmental Research,* 108(1):48-55.

Chen Y, Parvez F, Gamble M, Islam T, Ahmed A, Argos M, Graziano JH, Ahsan H (2009). Arsenic exposure at low-to-moderate levels and skin lesions, arsenic metabolism, neurological functions, and biomarkers for respiratory and cardiovascular diseases: Review of recent findings from the Health Effects of Arsenic Longitudinal Study (HEALS) in Bangladesh. *Toxicology and Applied Pharmacology*. In Press, Corrected Proof.

European Union (1998). Council Directive 98/83/EC on the quality of water intended for human consumption. *Official Journal of the European Communities*. L330.

Health Canada (2006). Guidelines for Canadian Drinking Water Quality: Guideline Technical Document for Arsenic. Health Canada, Ottawa, Ontario.

Health Canada (2008). Guidelines for Canadian Drinking Water Quality Summary Table, p. 14. Federal-Provincial-Territorial Committee on Drinking Water, Federal-Provincial-Territorial Committee on Health and the Environment.

IARC (International Agency for Research on Cancer) (2004). Arsenic in Drinking Water. IARC.

IPCS (International Programme on Chemical Safety) (2001). Environmental Health Criteria 224: Arsenic and arsenic compounds. World Health Organization, IPCS, Geneva, Switzerland.

Majumdar KK, Mazumder DNG, Ghose N, Ghose A, Lahiri S (2009). Systemic manifestations in chronic arsenic toxicity in absence of skin lesions in West Bengal. *Indian Journal of Medical Research,* 129(1):75-82.

Mazumder DNG, Haque R, Ghosh N, De BK, Santra A, Chakraborty D, Smith AH (1998). Arsenic levels in drinking water and the prevalence of skin lesions in West Bengal, India. *International Journal of Epidemiology*, 27(5):871-877.

Mazumder DNG (2008). Chronic arsenic toxicity and human health. *Indian Journal of Medical Research*, 128:436-447.

NOTE: Important general information is contained in PART II, Chapter 6

Mazumder DNG, Majumdar KK, Santra SC, Kol H, Vicheth C (2009). Occurrence of arsenicosis in a rural village of Cambodia. *Journal of Environmental Science and Health, Part A*, 44(5):480 - 487.

MoH NZ (Ministry of Health New Zealand) (2008). *New Zealand Ministry of Health, Drinking-water standards for New Zealand* 2005 (Revised 2008), p. 175. Ministry of Health, Wellington.

Smith AH, Steinmaus CM (2009). Health effects of arsenic and chromium in drinking water: recent human findings. *Annual Review of Public Health*, 30:107-122.

USEPA (Unites States Environmental Protection Agency) (2008). National Primary Drinking Water Standards. Available Online: http://www.epa.gov/safewater/consumer/pdf/mcl.pdf

WHO (World Health Organization) (2003). Arsenic in drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality, WHO.

WHO (World Health Organization) (2004). Guidelines for Drinking-water Quality. First addendum to 3rd edition. Volume 1. Recommendations. WHO.

NOTE: Important general information is contained in PART II, Chapter 6

Asbestos

(endorsed 1996)

GUIDELINE

Data are insufficient to determine a guideline value for asbestos in drinking water.

GENERAL DESCRIPTION

Asbestos is a general term for certain fibrous silicate minerals. It can be present in drinking water from the dissolution of asbestos-containing minerals, industrial effluent, atmospheric deposition, and deterioration of asbestos cement pipes commonly used in water distribution systems.

The chemical and crystalline structure of asbestos results in products with a high tensile strength, durability, flexibility, and heat and chemical resistance. Asbestos has been used in construction materials such as asbestos cement pipes and sheets, electrical and thermal insulation, brake linings and clutch pads.

The extent of asbestos contamination of food has not been well studied because of the lack of a simple and reliable analytical method. Limited data indicate that the amount in food may be 10 times higher than that found in drinking water.

Studies in the United States and Canada have reported typical asbestos fibre numbers in drinking water of less than 1 MFL (million fibres per litre). Severe deterioration of asbestos cement pipes has been known to produce fibre numbers of up to 2000 MFL.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for asbestos; however, fibre numbers are probably similar to those reported overseas.

TREATMENT OF DRINKING WATER

Asbestos fibre numbers can be reduced by the standard water treatment processes of coagulation and filtration.

MEASUREMENT

Asbestos can be analysed using transmission electron microscopy with identification of the fibres by selected-area electron diffraction. This procedure is both costly and time consuming and is not suitable for routine analysis. The limit of determination is about 0.3 MFL.

HEALTH CONSIDERATIONS

The health hazards associated with inhalation of asbestos have been recognised for a long time. They include asbestosis, cancer of the bronchial tubes, malignant mesothelioma, and possibly cancers of the gastrointestinal tract and larynx (IPCS 1986).

In contrast, there has been little evidence that ingested asbestos causes cancer. A number of quite extensive epidemiological studies have been carried out on the effects of asbestos in the water supply. On the basis of these data there is no demonstrated excess risk of cancer even with high numbers of asbestos fibres in drinking water.

It is not clear whether asbestos fibres ingested in drinking water can pass through the walls of the

gastrointestinal tract in sufficient numbers to cause adverse effects. Experiments with laboratory animals indicate that penetration, if it occurs at all, is extremely limited.

Animal studies on the carcinogenic effects of ingested asbestos have been inconclusive.

Asbestos did not exhibit mutagenic activity in tests with bacteria but has induced chromosomal aberrations, malignant transformation of mammalian cells *in vitro*, and various biochemical alterations associated with tumour promoters.

The International Agency for Research on Cancer has concluded that asbestos is carcinogenic to humans by the inhalation route (Group 1, sufficient evidence of carcinogenicity in humans) (IARC 1987).

DERIVATION OF GUIDELINE

There are insufficient data to set a guideline value for asbestos in drinking water. It is unlikely, however, that the numbers of asbestos fibres present in most drinking water supplies would be a health concern. The weight of evidence indicates that ingested asbestos is not hazardous to health.

The guideline should be reviewed as soon as more toxicological data are available.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1986). Asbestos and other natural mineral fibres. Environmental Health Criteria, 53. World Health Organization, IPCS.

NOTE: Important general information is contained in PART II, Chapter 6

Asulam

(endorsed 2011)

GUIDELINE

Based on human health concerns, asulam in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Asulam (CAS 3337-71-1) belongs to the carbamate class of chemicals. Other pesticides in this class include aldicarb, methomyl, carbaryl and methiocarb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, asulam would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Users: Asulam (methyl sulfanilylcarbamate) is a selective, post-emergent systemic herbicide. It is used to control weeds in crops and pastures.

There are registered products containing asulam in Australia. These products are intended for professional use and all are aqueous concentrate liquids. Application methods include boom spray, aircraft and knapsack. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to asulam is residues in food. Residue levels in crops produced according to good agricultural practice are generally low.

Agricultural use of asulam may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Although asulam is considered to be amongst the most significant herbicides in use (Radcliffe 2002), it is not widely tested in drinking water. Where it has been tested, it has not generally been detected above guideline values.

TREATMENT OF DRINKING WATER

Asulam is a highly water soluble compound and therefore the effectiveness of its removal by activated carbon processes is limited (Matsui *et al.* 2003).

Partial removal of asulam may be achieved by ozonation (Abe and Tanaka 1996). However, advanced

oxidation techniques have been shown to remove asulam much more effectively than ozone treatment.

MEASUREMENT

Asulam may be detected in drinking waters by liquid chromatography–tandem mass spectrometry, with a practical limit of detection of 0.001 mg/L (1 µg/L) (Honing 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for asulam is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 40 mg/kg bw/day from a 2-year rat study. The NOEL is based on effects on the thyroid and adrenal medulla. The ADI incorporates a safety factor of 2000 and was established in 1985.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: There is no information available on the absorption or metabolism of asulam.

Acute effects: Asulam has a low oral and dermal toxicity. Its skin sensitisation potential has not been determined, although it is structurally similarity to sulfonamide, which is a sensitiser. There was no evidence of neurotoxic symptoms normally associated with carbamates.

Short-term effects: A 6-month dietary study in dogs reported haematological changes at dose levels above 60 mg/kg bw/day and thyroid hypertrophy at 1500 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in mice, rats and dogs. In all species, effects were noted on haematological parameters and on the thyroid gland. Thryoid hypertrophy was reported in mice at 180 mg/kg bw/day and reductions in white blood cells in dogs at 60 mg/kg bw/day. In rats, there were effects on the adrenals in both sexes and on the thyroid of males. The NOEL in the rat was 40 mg/kg/day, which was used as the basis for the ADI.

Carcinogenicity: Long-term studies in rats indicated an increased incidence of phaeochromocytomas in the adrenal medulla at the highest dose level only. This was not considered relevant at the dose levels to which humans are exposed.

Genotoxicity: Asulam is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Reproduction studies in rats and developmental toxicity studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Asulam is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.07 mg/L for asulam was determined as follows:

$$0.07 \text{ mg/L} = \frac{40 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 2000}$$

where:

- 40 mg/kg bodyweight/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 2000 is the safety factor applied to the NOEL from the long-term study in rats. It incorporates a safety factor of 10 for interspecies extrapolation, 10 for intraspecies variation, and an additional 20 for the failure of some studies to demonstrate NOELs.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Abe K, Tanaka K (1996). Degradation of phenol, asulam and lignin in aqueous solution by ozonation. *Toxicological and Environmental Chemistry*, 54(1):187-193.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Honing M (2000). Carbamate and carbamoyloxime insecticides: single-class, multiresidue analysis of. In: *Encyclopedia of Analytical Chemistry*, John Wiley & Sons.

Matsui Y, Fukuda Y, Inoue T, Matsushita T (2003). Effect of natural organic matter on powdered activated carbon adsorption of trace contaminants: characteristics and mechanism of competitive adsorption. *Water Research*, 37:4413-4424.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Radcliffe JC (2002). *Pesticide use in Australia: a review undertaken by the Australian Academy of Technological Sciences and Engineering*. Australian Academy of Technological Sciences and Engineering, Victoria, Australia.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Atrazine

(endorsed 2011)

GUIDELINE

Based on human health concerns, atrazine in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Atrazine (CAS 1912-24-9) belongs to the triazine class of chemicals. There are a large number of herbicides in this class, including simazine, cyanazine, propazine, and ametryn (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, atrazine would not be a health concern unless the concentration exceeded 0.02 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline value is based on long term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Atrazine is a herbicide for the control of weeds and grasses in agricultural crops.

There are registered products that contain atrazine in Australia. These products are intended for professional use. Atrazine is available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to atrazine and its metabolites is residues in food and drinking water. Residue levels in food produced according to good agricultural practice are generally low. Maximum residue limits (MRLs) are at the level of detection.

Agricultural use of atrazine may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATERS

Atrazine has been occasionally reported in Australian drinking waters, including in New South Wales, Queensland and Tasmania. It was detected at up to 0.0009 mg/L (0.9 µg/L) in surface waters in New South Wales (Tran *et al.* 2007) and 0.0013 mg/L (1.3 µg/L) in Queensland (Mitchell *et al.* 2005). In many countries, after application in agricultural areas, atrazine has been found in groundwater at levels of 0.00001–0.006 mg/L (0.01–6 µg/L) (WHO 2003). It has also been detected in drinking-water in several countries at levels of 0.00001–0.005 mg/L (0.01–5 µg/L) (WHO 2003) and as high as 0.0294 mg/L (29.4 µg/L) in Canada (Health Canada 1993). Regulation of atrazine use has become more stringent since the mid 1990s.

In cases where atrazine is present in drinking waters, there is a high likelihood that other closely related s-triazine metabolites with similar mammalian toxicity may also be present at similar concentrations.

TREATMENT OF DRINKING WATER

Atrazine can be a relatively difficult pesticide to treat in drinking water. Oxidation by chlorine or ozone is only partially effective at typical doses, and adsorption to activated carbon can be incomplete (Ormad *et al.* 2008). Conventional clarification/chlorination has been shown to be unreliable for the removal of atrazine from water (CARAT 2000). However, a combination of ozone, activated carbon and coagulation-flocculation can be effective (Ormad *et al.* 2008).

Atrazine has been shown to be near-completely removed when water undergoes advanced oxidation with iron-catalysed ultraviolet (UV) irradiation and peroxide, i.e. Fenton reaction (Huston *et al.* 1999), with only moderate removal reported for UV-peroxide in the absence of added iron (Kruithof *et al.* 2002), although much lower removal rates have been obtained at full-scale plants (CARAT 2000). Photodegredation of the pesticide has been investigated (Azenha *et al.* 2003). Relatively high removal rates through powdered activated carbon adsorption have been reported (Bozkaya-Schrotter *et al.* 2008).

MEASUREMENT

Atrazine can be measured by routine gas chromatrography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for atrazine is 0.005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.5 mg/kg bw/day from a 2-year dietary rat study. The NOEL is based on an increased incidence of mammary tumours in female rats at the next highest dose (2.8 mg/kg bw/day). The ADI incorporates a safety factor of 100, and was established in 1990. Subsequently, in 1994, the Advisory Committee on Pesticides and Health concluded that the rat mammary tumours were not relevant to human health. However, it was considered that the NOEL of 0.5 mg/kg bw/day continued to be an appropriately conservative endpoint on which to base the ADI, as the tumours were considered to reflect a hormonal interaction considered relevant to humans (see 'Long-term effects').

The previous ADI for atrazine was set in 1985 at 0.0003 mg/kg bw/day, based on a NOEL of 0.6 mg/kg bw/day in a 2-year rat study and using a 2000-fold safety factor. This ADI was amended to its present level after submission of additional toxicity studies.

The previous *Australian Drinking Water Guidelines* health value was 0.04 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Atrazine is readily absorbed via the gastrointestinal tract in humans and rats. It is extensively metabolised, and is rapidly excreted in the urine and faeces, almost completely within 72 hours. Levels in tissues were low. The main metabolites were dealkylated forms that were of similar toxicity to atrazine.

Acute effects: Atrazine has low acute oral and dermal toxicity. It is not a skin sensitiser in humans, based on large-scale occupational studies, but is a sensitiser in guinea pigs.

Short-term effects: Four-week dietary studies in rats and dogs reported decreased food-use efficiency (rats only), and bodyweight gain at 21 mg/kg bw/day and above.

NOTE: Important general information is contained in PART II, Chapter 6

A 3-week oral study in female rats reported irregular ovarian cycles (but not persistent oestrus or diestrus) at 75 mg/kg bw/day.

Ninety-day dietary studies in rats and dogs reported reduced bodyweight gain and food consumption at 3.3 mg/kg bw/day in rats and at 5 mg/kg bw/day in dogs. Reduced testicular weights and anaemia were seen at 15 mg/kg bw/day in rats. In a 6-month dietary study in rats, there was suppression of the luteinising hormone surge (a process initiated in the pituitary that normally initiates ovulation), and disruption of the oestrous cycle at 3.6 mg/kg bw/day

Long-term effects: A 2-year dietary study in mice reported only decreased bodyweight gain at 36 mg/kg bw/day. One-year and 2-year dietary studies in rats reported decreased bodyweight gain, increased pituitary weight, pituitary adenomas, and mammary tumours (one strain only) at 4.2 mg/kg bw/day after 1-year. Behavioural effects, skeletal muscle degeneration, and mammary growths symptomatic of hormone perturbation were seen at 20 mg/kg bw/day and above in these studies. While the rat mammary tumours themselves are not considered relevant to humans, the increases in gonadotropin-releasing hormone and luteinising hormone from the pituitary, which are considered a precursor event to the development of rat mammary tumours, are considered relevant to humans. This is an area of ongoing research. In a long-term dog study, lethargy, increased heart rate, myocardial degeneration, and decreased heart weight were seen at 33 mg/kg bw/day. The lowest overall NOEL from these studies is 0.5 mg/kg bw/day, based on mammary tumours in rats. This NOEL is the basis for the current ADI.

Carcinogenicity: The weight of evidence from long-term studies in mice and rats indicates that atrazine is not carcinogenic in humans, since the rat mammary tumours are not considered relevant to humans.

Genotoxicity: Atrazine is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two- and 3-generation reproductive studies in rats did not produce any evidence of effects on reproductive parameters. Developmental studies in rats produced some equivocal evidence of effects on foetal and post-natal development, but at doses much higher than those used in long-term studies.

Poisons Schedule: Atrazine is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for atrazine was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.5 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

NOTE: Important general information is contained in PART II, Chapter 6

The World Health Organization has established a health-based guideline value of 0.002 mg/L for atrazine in 1993 (WHO 2004). The WHO incorporated an additional 10-fold safety factor to reflect potential neoplasia; however, Australian authorities considered that the induction mechanism for the mammary tumours was not directly relevant to humans.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Azenha M, Burrows HD, Canle M, Coimbra R, Fernandez MI, Garcia MV, Peiteado MA, Santaballa JA (2003). Kinetic and mechanistic aspects of the direct photodegraclation of atrazine, atraton, ametryn and 2-hydroxyatrazine by 254 nm light in aqueous solution. *Journal of Physical Organic Chemistry,* 16(8):498-503.

Bozkaya-Schrotter B, Daines C, Lescourret A, Bignon A, Breant P, Schrotter J (2008). Treatment of trace organics in membrane concentrates I: pesticide elimination. *Water Science and Technology: Water Supply*, 8(2):223-230.

CARAT (Committee to Advise on Reassessment and Transition) (2000). Summary of Pesticide Removal/Transformation Efficiencies from Various Drinking Water Treatment Processes. United States Environmental Protection Agency & United States. Department of Agriculture Committee to Advise on Reassessment and Transition.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Health Canada (1993). Guidelines for Canadian Drinking Water Quality: Technical documents – Atrazine. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/atrazine/]

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction. *Water Research,* 33(5):1238-1246.

Kruithof J, Kamp P, Belosevic M (2002). UV/H_2O_2 treatment: the ultimate solution for pesticide control and disinfection. *Water Science Technology: Water Supply*, 2(1):113-122.

Mitchell C, Brodie J, White I (2005). Sediments, nutrients and pesticide residues in event flow conditions in streams of the Mackay Whitsunday Region, Australia. *Marine Pollution Bulletin*, 51(1-4):23-36.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71(1):97-106.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Tran AT, Hyne RV, Doble P (2007). Determination of commonly used polar herbicides in agricultural drainage waters in Australia by HPLC. *Chemosphere,* 67(5):944-53.

WHO (World Health Organization) (2003). Atrazine in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. WHO.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Azinphos-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, azinphos-methyl in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

Azinphos-methyl (CAS 86-50-0) belongs to the organophosphate class of chemicals and is structurally related to azinphos-ethyl. There are many pesticides in this chemical class, including acephate, chlorfenvinphos, diazinon, ethion, phorate, and terbufos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, azinphos-methyl would not be a health concern unless the concentration exceeded 0.03 mg/L. Excursions above this level even for a limited period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Azinphos-methyl is an insecticide, acaricide (miticide), and molluscicide for the control of pests such as moths, grasshoppers, beetles, aphids, and slugs in agricultural crops.

There are registered products that contain azinphos-methyl in Australia. These products are intended for professional use on pome fruit, citrus fruit, macadamia nuts, grape, lychee, and blueberry crops. They are available as concentrated solutions to be applied in both concentrated and diluted forms by ground and aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to azinphos-methyl and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of azinphos-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on occurrence of azinphos-methyl in Australian waters. In the USA, the estimated environmental concentration in surface waters was 0.016 mg/L (USEPA 2006). Azinphos-methyl was not found in a survey of Canadian drinking water (Health Canada 1989).

TREATMENT OF DRINKING WATER

No specific data on the treatment of azinphos-methyl in drinking water have been identified.

MEASUREMENT

Azinphos-methyl can be measured by routine gas chromatrography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for azinphos-methyl is 0.025 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day from a short-term (28-day) oral study in humans. The NOEL is based on the absence of cholinesterase inhibition at this dose, which was the only dose tested. The ADI incorporates a safety factor of 10, and was established in 2002.

The previous ADI was 0.001 mg/kg bw, based on a NOEL of 0.125 mg/kg bw/day for cholinesterase inhibition in a 2-year dog study, with a 100-fold safety factor. The ADI was amended following a review in 2002 of all available data on azinphos-methyl.

The acute reference dose (ARfD) of 0.075 mg/kg bw for azinphos-methyl was also established in 2002, based on the absence of cholinesterase inhibition and clinical signs of toxicity at the highest dose tested of 0.75 mg/kg bw/day in a single oral dosing study in humans. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Azinphos-methyl is readily absorbed via the gastrointestinal tract in rats. It is extensively metabolised, and is rapidly excreted in the urine and faeces, almost completely within 48 hours. The major metabolite was benzamide.

Acute effects: Azinphos-methyl has high acute oral toxicity in rats and mice, and high dermal toxicity in rats. It is a skin sensitiser in guinea-pig. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes.

Short-term effects: A 28-day dietary studies in rats reported dose-dependent red blood cell and plasma cholinesterase inhibition at 0.8 mg/kg bw/day and above, and brain cholinesterase inhibition at 3.2 mg/kg bw/day. A 28-day oral dosing study with humans reported no toxic effects at 0.25 mg/kg bw/day. This NOEL is the basis for the current ADI.

In 12-16 week dietary studies in rats and dogs, cholinesterase in brain, erythrocytes, and plasma was inhibited at 1 mg/kg bw/day in rats. No changes were noted histologically. Deaths were reported at doses of 2.5 mg/kg bw/day and above. In dogs, red blood cell cholinesterase was inhibited at 0.25 mg/kg bw/day.

Long-term effects: In 2-year dietary studies in mice, rats, and dogs, erythrocyte and plasma cholinesterase were inhibited at doses of 0.5 mg/kg bw/day in dogs and brain cholinesterase was inhibited at 3 mg/kg bw/day and above. Clinical signs of toxicity were noted at 6.25 mg/kg bw/day and above.

Carcinogenicity: Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for azinphos-methyl.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Azinphos-methyl was positive in some *in vitro* short-term studies, but not in *in vivo* studies. Overall, it was not considered to be genotoxic.

Reproductive and developmental effects: Three-generation reproduction studies in rats and mice, and developmental studies in rats, mice and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: Special neurotoxicity studies in rats by dietary administration found no evidence of delayed neurotoxicity.

Poisons Schedule: Azinphos-methyl is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for azinphos-methyl was determined as follows:

$$0.03 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 30}$$

where:

- 0.25 mg/kg bw/day is the NOEL based on a short-term (28-day) oral dosing study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 30 is the safety factor applied to the NOEL derived from human studies. This safety factor incorporates a factor of 10 for intraspecies variation, with an additional safety factor of 3 to account for the use of short-term data.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Health Canada (1989). Guidelines for Canadian Drinking Water Quality – Technical documents: Azinphos-methyl. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/azinphos/]

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2006). Reregistration Eligibility Decision for Azinphos-Methyl. USEPA, Office of Pesticide Programs.

NOTE: Important general information is contained in PART II, Chapter 6

Barium

(endorsed 2011)

GUIDELINE

Based on health considerations, the concentration of barium in drinking water should not exceed 2 mg/L.

GENERAL DESCRIPTION

Barium makes up approximately 0.04 per cent of the Earth's crust, and is the 16th most abundant non-gaseous element. Barium in drinking water is primarily from natural sources. Some barium salts such as the chloride and nitrate are soluble in water; others, including the carbonate, fluoride, phosphate and sulfate, are insoluble. Barium is not considered to be an essential nutrient for humans.

Barium compounds have a wide variety of industrial applications. They are used in the plastics, rubber, electronics, steel, optical, and textile industries. They are also used in ceramic glazes and enamels, in glass and paper making, as a lubricant additive, in pharmaceuticals and cosmetics, and as a rodenticide.

The concentration of barium in drinking water overseas is usually low, typically less than 0.02 mg/L.

Most foods contain small quantities of barium. The major dietary sources are milk, potatoes and flour. Some cereal products and nuts can contain large amounts. It has been estimated that average dietary intake is approximately 1 mg per day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In Australian drinking water supplies, typical concentrations of barium range from <0.002 mg/L to 1.1 mg/L.

TREATMENT OF DRINKING WATER

Conventional water treatment using alum or ferric coagulation is not effective in removing barium from drinking water. Lime softening can remove more than 90%.

MEASUREMENT

The barium concentration in drinking water can be determined using inductively coupled plasma emission spectroscopy (APHA Method 3500-Ba Part C 1992), or atomic absorption spectroscopy (APHA Method 3500-Ba Part B 1992). For both methods the limit of determination is approximately 0.01 mg/L.

HEALTH CONSIDERATIONS

Reviews of the human and animal toxicity data for barium are available (IPCS 2001, OEHHA 2003, WHO 2004a, USEPA 2005, ATSDR 2007).

The degree of absorption from the gastrointestinal tract depends on the solubility of the barium compound, and on other factors including age, study duration, species, and fasting status of the animals. The presence of food in the gastrointestinal tract appears to decrease barium absorption, and absorption appears to be higher in young animals than in older ones. The range of reported oral absorption in animal studies was 0.7–85.0%; a default value of 20% gastrointestinal absorption of dissolved barium in drinking water is assumed by the Safe Drinking Water Committee of the National Research Council of the USA (IPCS 2001, OEHHA 2003). After absorption, barium is deposited in bone and teeth.

Barium toxicity is caused by the free cation, and highly soluble barium compounds are more toxic than insoluble compounds. In rodents, kidney toxicity appears to be the most sensitive effect, whereas in humans, cardiovascular (hypertension) effects have been of prime concern.

The identification of hypertension as a health end-point of concern for humans is supported by findings of hypertensive effects in humans who ingested acutely high doses of barium compounds, in workers who inhaled dusts of barium ores and barium carbonate, in experimental animals given barium intravenously, and in rats exposed to barium in drinking water while on calcium-restricted diets (IPCS 2001).

A number of epidemiological studies have been carried out on the effects of barium in drinking water on cardiovascular disease. Wones *et al*. (1990) exposed eleven normotensive volunteers to barium in drinking water for 10 weeks at concentrations up to 10 mg/L. No cardiovascular effects were observed at the maximum estimated dose of 0.21 mg per kg bodyweight per day (mg/kg bw/day). Between 1976 and 1977, Brenniman and Levy (1985) studied two populations (n = 1175 and 1203) in the United States where water softeners were not used and mean barium concentrations were 0.1 mg/L or 7.3 mg/L (range 2–10 mg/L for the latter). No differences were observed in blood pressure or incidence of kidney disease between the two communities. Assuming 70 kg body weight and 2 L/day drinking water consumption, the mean doses of barium were 0.003 mg/kg bw/day and 0.21 mg/kg bw/day. Thus, 0.21 mg/kg bw/day is a no-observed-adverse-effect level (NOAEL) in this study, however because no adverse effects were found, the NOAEL is likely to be higher than this value.

Chronic toxicity studies of barium chloride in drinking water of rats and mice caused kidney effects at the higher doses used. Relevant NOAELs in these studies were 45 mg/kg bw/day for female rats and 75 mg/kg bw/day in male mice (NTP 1994)

There is no evidence from chronic rodent studies that barium causes cancer. The weight of evidence indicates barium is not mutagenic in tests with bacteria and does not damage DNA.

DERIVATION OF GUIDELINE

A drinking water guideline of 2 mg/L has been set after considering the following.

Using animal data:

Based on the chronic male mouse-study data of the National Toxicology Program (NTP 1994), the United States Environmental Protection Agency (USEPA 2005) and Agency for Toxic Substances and Disease Registry (ATSDR 2007), the lower confidence limit for the benchmark dose at 5% incidence ($BMDL_{05}$) of renal effects was determined to be 63 and 61 mg/kg bw/day. A drinking-water guideline value of 6 mg/L (rounded) can be derived following the standard procedure:

$$5.6 \text{ mg/L} = \frac{60 \text{ mg/kg bw/day} \times 70\text{kg} \times 0.8}{300 \times 2\text{L/day}}$$

Where:

- 60 mg/kg bw/day is the $BMDL_{05}$ for kidney effects determined from a long-term drinking water study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.8 is a proportionality factor based on the assumption that 80% of daily intake attributable to drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 300 is the safety factor applied to the $BMDL_{05}$ derived from animal studies (10 for interspecies variations, 10 for intraspecies variations and 3 for database inadequacies).

NOTE: Important general information is contained in PART II, Chapter 6

The lowest NOAEL of 45 mg/kg bw/day from the chronic rat and mouse studies can also be used in the above equation to give a guideline value of 4 mg/L (rounded).

Using human data:

The NOAEL of 0.2 mg/kg bw/day from the Brenniman and Levy (1985) epidemiological study gives a drinking water guideline of 2 mg/L (rounded) as follows:

$$1.87 \text{ mg/L} = \frac{0.2 \text{ mg/kg bw/day} \times 70\text{kg} \times 0.8}{3 \times 2\text{L/day}}$$

Where:

- 0.2 mg/kg/day is the NOAEL in humans identified by Brenniman and Levy (1985).
- 3 is the safety factor applied to account for potential variability in response between humans. Justified on the grounds the study population was randomly selected from all people above the age of 18 years and therefore inherently included sensitive sub-populations. Furthermore, as the highest mean barium water concentrations in the study did not cause an adverse effect, the NOEL is likely to be higher than the one used to derive the drinking water guideline.

The World Health Organization (WHO 2004) established a guideline of 0.7 mg/L by dividing the mean barium water concentration of 7.3 mg/L in the Brenniman and Levy (1985) study by an uncertainty factor of 10 to account for human intraspecies variation. WHO (2006) acknowledges that this may be highly conservative.

The United States Environmental Protection Agency (USEPA 2006) indicates that 2 mg/L is the lowest level to which present technology and resources can reasonably be required to remove barium should it occur in drinking water.

REFERENCES

APHA Method 3500-Ba Part B, (1992). Barium: Atomic Absorption Spectrophotometric method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Ba Part C, (1992). Barium: Inductively Coupled Plasma method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

ATSDR (Agency for Toxic Substances and Disease Registry) (2007). Toxicological Profile for Barium and Barium Compounds. ATSDR, US Department Of Health And Human Services. PB2008-100003.

Brenniman GR, Levy PS (1985). Epidemiological study of barium in Illinois drinking water supplies. *Advances in Modern Environmental Toxicology*, Princeton Publishing Co, New Jersey, 9:231–249.

IPCS (International Programme on Chemical Safety) (2001). Barium and barium compounds. Concise International Chemical Assessment Document 33. IPCS, World Health Organization.

NTP (National Toxicology Program) (1994). Technical report on the toxicology and carcinogenesis studies of barium chloride dihydrate (CAS No. 10326-27-9) in F344/N rats and B6C3F1 mice. NTP, Washington, DC.

OEHHA (Office of Environmental Health Hazard Assessment) (2003). Public Health Goal for Barium in Drinking Water. OEHHA, California Environmental Protection Agency.

USEPA (United States Environmental Protection Agency) (2005). Toxicological Review of Barium and Compounds. USEPA, EPA/635/R-05/001.

NOTE: Important general information is contained in PART II, Chapter 6

USEPA (United States Environmental Protection Agency) (2006). Consumer Factsheet on: Barium. USEPA. http://www.epa.gov/safewater/contaminants/dw_contamfs/barium.html.

WHO (World Health Organization) (2004a). Barium in Drinking-Water, Background document for development of WHO *Guidelines for Drinking-water Quality*, World Health Organization. WHO/SDE/WSH/03.04/76

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, First Addendum, WHO, Geneva, Switzerland.

Wones RG, Stadler BL, Frohman LA (1990). Lack of effect of drinking water barium on cardiovascular risk factors. *Environmental Health Perspectives*, 85:355-359.

Benomyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, benomyl in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Benomyl (CAS 17804-35-2) belongs to the benzimidazole class of carbamate compounds. Other pesticides in this class include carbendazim, dicamba and diflubenzuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, benomyl would not be a health concern unless the concentration exceeded 0.09 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Benomyl is a systemic, broad-spectrum fungicide for use on fruit trees, nuts, vegetables, cereals, ornamentals and turf.

There are no registered products containing benomyl in Australia, as the active was suspended and subsequently withdrawn due to health concerns in December 2006. However, de-registered compounds may still be detected in water.

Exposure sources: The main source of public exposure to benomyl and its metabolites if used in the future would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of benomyl in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of benomyl detected in Australian drinking water supplies have been identified.

TREATMENT OF DRINKING WATER

No definitive literature regarding the removal of benomyl from drinking water has been identified. Some field trials have been undertaken regarding the removal of benomyl from water using activated carbon and other adsorbents (Massey *et al.* 1992, Giry *et al.* 2001). Biodegredation (Matsumura *et al.* 1991) and ultraviolet irradiation (Grechko *et al.* 1982) in wastewater have been reported.

MEASUREMENT

No standard methods for the analysis of benomyl in drinking water have been identified. However, benomyl can be analysed by high–performance liquid chromatography with detection by ultraviolet absorbance (Marvin *et al*. 1991). The reported detection limit for this method is 0.009 $mg.L^{-1}$.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for benomyl is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.5 mg/kg bw/day from a 2-year dog study. The NOEL is based on an increase in testicular degeneration. The ADI incorporates a safety factor of 100 and was established in 2003.

The acute reference dose (ARfD) of 0.06 mg/kg bw/day for benomyl was established in 2003, based on a NOEL of 6.25mg/kg bw/day from three developmental studies, which showed an increase in micro-/ anophthalmia. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Benomyl is rapidly and extensively absorbed via the gastrointestinal tract and distributed widely thoughout the body. It is metabolised to carbendazim (70% within 1 hour). Excretion is via the urine and, to lesser extent, faeces, and is essentially complete within 72 hours.

Acute effects: Benomyl has a very low acute oral toxicity; there are no data on acute dermal toxicity. There are reported cases of skin sensitisation in humans.

Short-term effects: In 70/90 day dietary studies in rats and dogs, there was reduced sperm count in rats at the highest dose tested of 20 mg/kg bw/day and increased liver weights at 125 mg/kg bw/day. In dogs, there were increased liver enzymes at 125 mg/kg bw/day.

Long-term effects: Long-term (2-year) dietary studies in mice and rats did not demonstrate any toxicity. In a 2-year study in dogs, there was testicular atrophy at doses of 12.5 mg/kg bw/day and above, and increased levels of liver enzymes in serum were seen at the highest dose tested of 62.5 mg/kg bw/day. The lowest overall NOEL was 2.5 mg/kg bw/day in dogs, which was used as the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there was evidence of carcinogenicity only in the mouse, where liver tumours were observed. These were considered to be species-specific, possibly as a result of liver toxicity. Based on the available data, benomyl was not considered to be a carcinogenic risk to humans.

Reproduction and developmental effects: Reproduction studies in rats and dogs reported decreased sperm production at 45 mg/kg bw/day. Testicular degeneration and atrophy was also reported at doses of 45 mg/kg bw/day (rats) and 12.5 mg/kg bw/day (dogs). Developmental studies in mice and rats (but not rabbits) reported teratogenic effects in the foetuses in the absence of any maternal toxicity at doses of 10 mg/kg bw/day.

Poisons Schedule: Benomyl is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.09 mg/L for benomyl was determined as follows:

$$0.09 \text{ mg/L} = \frac{2.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.5 mg/kg bw/day is the NOEL based on a long-term (2-year) study in dogs.
- 70 kg is taken as the average weight of an adult
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult
- 100 is the safety factor applied to the NOEL derived from the acute human study. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variations.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Giry G, Ayele J, Gauthier C (2001). Removal of carbendazim from fruit conditioning waters by adsorption on different materials. *Environmental Technology*, 22(7):803-811.

Grechko AV, Marchenko PV, Shevchenko MA (1982). Removal of pesticides from greenhouse drainage wastewater. *Khimiya i Tekhnologiya Vody*, 4(1):56-58.

Marvin CH, Brindle ID, Hall CD, Chiba M (1991). Rapid on-line precolumn high-performance liquid chromatographic method for the determination of benomyl, carbendazim and aldicarb species in drinking water. *Journal of Chromatography*, 555(1-2):147-154.

Massey JH, Lavy TL, Skulman BW (1992). Field and laboratory evaluations of an activated charcoal filtration unit. In: *Proceedings of International Workshop on Research in Pesticide Treatment/Disposal/Waste Minimization*, Department of Agronomy,University of Arkansas, Fayetteville, AR, USA, pp. 85-94.

Matsumura F, Katayama A (1991). Photochemical and microbial degradation technologies to remove toxic chemicals. In: *Pesticide Waste Management: Technology and Regulation*. From a symposium sponsored by the Division of Agrochemicals at the Fourth Chemical Congress of North America, New York, New York 1992, pp 201-209.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Bentazone

(endorsed 2011)

GUIDELINE

Based on human health concerns, bentazone in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Bentazone (CAS 25057-89-0) belongs to the benzimidazole class of chemicals. Other pesticides in this class include benomyl, carbendazim and dicamba (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, bentazone would not be a health concern unless the concentration exceeded 0.4 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Bentazone is a herbicide for the control of broad-leaf weeds in home garden lawns, turf and agricultural crops.

There are registered products containing bentazone or its sodium salt in Australia. These products are intended for professional and home garden use and are available as concentrated solutions to be applied using ground, aerial or hand-held spray methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to bentazone are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of bentazone may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No values for concentrations of bentazone in Australian waters were found. Bentazone has been used for a considerable period of time and has been detected in surface waters overseas at levels of 0.1 to 6 µg/L (WHO/SDE/WSH 2004).

TREATMENT OF DRINKING WATER

Bentazone has been shown to be completely removed by ozonation (Bozkaya-Schrotter *et al.* 2008). Moderate removal can be achieved using powdered activated carbon adsorption. Advanced oxidation using ultraviolet irradiation and peroxide has been demonstrated to achieve a moderate level of bentazone removal (Kruithof *et al.* 2002).

MEASUREMENT

Measurement of bentazone can readily be achieved to detection limits of 0.05 µg/L using dichloromethane extraction and gas chromatography with electron capture detection (FAO/WHO, 1991). Enhancement of sensitivity for analysis of bentazone in water can be achieved by use of solid phase extraction and gas-chromatography–mass spectrometry, achieving a detection limit of 0.02 µg/L (Thortensen *et al.* 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for bentazone is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 10 mg/kg bw/day. This NOEL is based on decreased bodyweight and significant changes in organ weights observed in a 2-year rat study. The ADI incorporates a safety factor of 100 and was established in 1988.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Bentazone is readily and extensively absorbed via the gastrointestinal tract. It is not extensively metabolised, and is rapidly excreted in the urine, almost completely within 24 hours.

Acute effects: Bentazone has low acute oral toxicity and moderate acute dermal toxicity.

Short-term effects: Three-month dietary studies in rats and dogs reported clinical signs of neurotoxicity, as well as changes in biochemical and haematological parameters and organ weight changes at 120 mg/kg bw/day.

Long-term effects: Long-term dietary studies in mice and rats reported decreased food consumption and bodyweight gain as well as significant changes in relative organ weights at 45.7 mg/kg bw/day.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for bentazone.

Genotoxicity: Based on *in vitro* and *in vivo* short-term studies, bentazone is not considered to be genotoxic.

Reproductive and developmental effects: A 3-generation reproduction study in rats and a developmental study in rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Bentazone is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for bentazone was determined as follows:

$$0.4 \text{ mg/L} = \frac{10 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 10 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has established a health-based guideline value of 0.3 mg/L for bentazone in 1998 (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bozkaya-Schrotter B, Daines C, Lescourret A, Bignon A, Breant P, Schrotter J (2008). Treatment of trace organics in membrane concentrates I: pesticide elimination. *Water Science Technology: Water Supply*, 8(2):223-230.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

FAO/WHO (World Health Organization/Food and Agriculture Organization) (1991). *Pesticide residues in food – 1991 evaluations. Part I – Residues*. Rome, Food and Agriculture Organization of the United Nations, Joint FAO/WHO Meeting on Pesticide Residues (FAO Plant Production and Protection Paper 113/1).

Kruithof J, Kamp P, Belosevic M (2002). UV/H_2O_2 treatment: the ultimate solution for pesticide control and disinfection. *Water Science Technology: Water Supply*, 2(1):113-122.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Thorstensen CW, Lode O, Christiansen AL (2000). Development of a solid-phase extraction method for phenoxy acids and bentazone in water and comparison to a liquid–liquid extraction method. *Journal of Agricultural and Food Chemistry*, 48(12):5829-5833.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

WHO/SDE/WSH (2004). Bentazone in Drinking Water. Background Document for Development of WHO *Guidelines for Drinking Water Quality*. World Health Organization, Geneva, WHO/SDE/WSH/03.04/77.

NOTE: Important general information is contained in PART II, Chapter 6

Benzene [CASRN 71-43-2]

(endorsed 2013)

GUIDELINE

Based on health considerations the concentration of benzene in drinking water should not exceed 0.001 mg/L.

GENERAL DESCRIPTION

Benzene is a clear, colourless-to-yellow liquid and highly flammable aromatic hydrocarbon. It is present in petroleum products such as motor fuels and solvents, and motor vehicle emissions constitute the main source of benzene in the environment. Benzene occurs naturally in crude oil and coal and is an additive and a by-product of oil-refining processes. It constitutes approximately 1-2% of unleaded gasoline by volume (US DHHS, 2011). Tobacco smoke is another significant source of exposure (WHO, 2010). It also occurs in natural gas and emissions from volcanoes and forest fires.

Human exposure to benzene occurs primarily through inhalation (WHO, 2010). When released to surface waters, benzene rapidly volatilises to the air (WHO, 2010). Benzene is not persistent in surface water or soil and either volatilises to air or is degraded by bacteria under aerobic conditions (WHO, 2010). For water contamination, benzene is therefore of most concern in groundwater. Benzene can also occur in foods and drinks as a product of the reaction between benzoate and ascorbic acid, and has been found in soft drinks in the UK at concentrations as high as 0.028 mg/L (FSA, 2006).

Benzene is also used widely as an industrial solvent by the chemical and pharmaceutical industries in the production of styrene/ethylbenzene, cumene/phenol and cyclohexane. The use of benzene as a solvent has been greatly reduced in recent years.

Unlike other petroleum hydrocarbons such as ethylbenzene, toluene and xylene the odour threshold for benzene is relatively high at 10 mg/L (WHO, 2003).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Benzene has only rarely been identified in Australian drinking waters. Natural concentrations in most water sources are usually very low. Benzene can occur naturally in groundwater as a result of proximity to, or contact with, coal seams, petroleum and gas deposits, and shales. It may be mobilised by extraction activities (Lesage *et al.*, 1997; Leusch and Bartkow, 2011; Volk *et al*, 2011). However, contamination can occur, usually via exposure to petrochemicals in surface waters or groundwater. Known sources of groundwater and surface water contamination include leakage from sub-surface fuel storage tanks (do Rego & Netto, 2007) and proximity to natural hydrocarbon deposits (IPCS, 1993). Emissions of fuel components from boating use is a known source of contamination of multiple-use lakes and reservoirs (Schmidt *et al.*, 2004). Benzene was reported in 9% of samples from an extensive groundwater survey undertaken in Denmark with the highest concentration being 0.034 mg/L (Juhler & Felding, 2003). Concentrations of up to 0.0027 mg/L were recorded in a NSW town water supply contaminated with petrol (Allen *et al.*, 2005). Groundwater from a contaminated well in the USA contained up to 0.3 mg/L of benzene (IPCS, 1993). Benzene has been reported at up to 0.004 mg/L in municipal drinking water in Taiwan (Kuo *et al.*, 1997), up to 0.01 mg/L in Germany (IPCS, 1993), and is occasionally detected in drinking waters in the USA (Williams *et al.*, 2004).

TREATMENT OF DRINKING WATER

Volatile organic chemicals such as benzene are most commonly treated in drinking water by aeration stripping and/or adsorption to granular activated carbon (GAC). A conventional biologically active sand filter has been shown to be highly effective for the removal of benzene from contaminated water, under suitable conditions (Arvin *et al.*, 2004). Effective bioremediation of highly contaminated groundwaters has also been demonstrated (Sedran et al., 2004; Zein *et al.*, 2006).

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for the analysis of benzene (APHA, AWWA & WEF, 2012). An inert gas is bubbled through the sample and benzene is trapped on an adsorbent. The adsorbent is then heated and benzene analysed using gas chromatography with mass spectrometric (GC-MS) detection (Method 6200 B) or photoionisation (PI) detection (Method 6200 C) (APHA, AWWA & WEF, 2012). The method detection limit is 36 ng/L for GC-MS and 17 ng/L for GC-PI (APHA, AWWA & WEF, 2012).

HEALTH CONSIDERATIONS

Benzene is rapidly and efficiently absorbed (30-50%) following inhalation. Following ingestion, animal data indicate that nearly all is absorbed from the gastrointestinal tract. Less than 1% is absorbed through the skin. Following absorption it is widely distributed throughout the body. It is metabolised predominantly into phenol by the liver, and also by bone marrow (WHO, 2003).

Human health data are mainly from studies where benzene had been inhaled. Acute exposure to high concentrations affects the central nervous system causing dizziness, nausea, vomiting, headache and drowsiness. Inhalation of very high concentrations can cause death. Chronic and subchronic exposure to lower concentrations leads to a range of adverse effects on the blood system including pancytopenia, aplastic anaemia, thrombocytopenia, granulocytopenia and lymphocytopenia with white blood cells being the most sensitive (WHO, 2003; Health Canada, 2009). There is considerable evidence that occupational exposure to low benzene concentrations in air for periods as short as 1-5 years may result in leukaemia (ATSDR, 2007).

In animal studies, benzene caused leukaemia and other cancers when administered orally and by inhalation to rats and mice. It can also induce chromosome damage and gene mutation in mammalian cells. It was not found to be mutagenic in tests with bacteria.

The International Agency for Research on Cancer has concluded that benzene is carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans) (IARC, 1987).

DERIVATION OF GUIDELINE

The European Union (1998), WHO (2011), Health Canada (2006), USEPA (2008) and New Zealand (MoH NZ 2008) have set drinking water guidelines for benzene of 0.001-0.01 mg/L based on carcinogenic potential (leukaemia) of benzene in humans from inhalation associated with occupational exposures and/or a 2 year oral study in rats and mice (NTP 1986).

USEPA (2003) derived a cancer slope factor (CSF) of 0.015 to 0.055 per mg/kg/day based on linear extrapolation of leukaemia data from occupational exposure. This translated to a lifetime risk of one excess cancer case per 1 million people associated with a concentration of 0.001-0.01 mg/L of benzene (USEPA 2003).

NOTE: Important general information is contained in PART II, Chapter 6

Both USEPA (2003) and Health Canada (2006) identified that inhalation of volatilized benzene and dermal adsorption need to be added to ingestion of drinking water. Based on exposure from showering and bathing, Health Canada (2006) derived equivalent doses of 1.2L water per day to account for inhalation and 0.8L water per day to account from dermal adsorption. Using these equivalent doses and the USEPA (2003) cancer slope factor a guideline value can be calculated using the formula:

$$0.00032\text{-}0.0012 \text{ mg/L} = \frac{70 \text{ kg body weight} \times 10^{-6}}{4\text{L/day} \times 0.015\text{-}0.055 \text{ mg/kg/day (CSF)}}$$

where:

- 0.015 -0.055 mg/kg/day is the CSF range calculated by USEPA (2003) from occupational exposure to benzene
- 70 kg is the average weight of an adult
- 10-6 is the additional lifetime risk of one cancer from drinking water exposure
- 4L/day is the average dose including 2L/day for ingestion plus 1.2L equivalent dose/day for inhalation and 0.8L equivalent dose/day for dermal adsorption.

WHO (2003) also used the occupational leukaemia data to determine that a concentration of 0.001 mg/L in drinking water would entail a maximum lifetime risk of one additional case of cancer per 1 million people. Analysis of data from a 2 year gavage study in rats and mice (NTP 1986) using the robust linear extrapolation model produced similar results with an excess lifetime cancer risk of 1 per million people associated with 0.001-0.008 mg/L benzene based on leukaemia and lymphomas in female mice and oral cavity squamous cell carcinomas in male rats respectively (WHO, 2003). On the basis of these two calculations WHO (2003) identified that concentrations of 0.01 mg/L and 0.001mg/L were associated with excess cancer rates of 1 per 100,000 and 1 per 1,000,000 people respectively. WHO (2011) adopted a guideline value of 0.01 mg/L based on an estimated additional lifetime risk of one cancer per 100,000 people.

The concentration of 0.001mg/L associated with an excess cancer risk of 1 per 1,000,000 people calculated by WHO (2003) is within the range of 0.00032-0.0012mg/L. For consistency with WHO (2003, 2011), a health-based guideline of 0.001 mg/L has been adopted.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR) (2007) Toxicological profile for Benzene. Public Health Service, US, Department of Health and Human Services, Atlanta, Georgia.

Allen, K., Thorne, S. and Byleveld, P. (2005) You found benzene - where? Benzene in source drinking water in northern New South Wales. Presented at Australian Water Association Specialty Conference "Contaminants of Concern in Water", Canberra 22-23 June, Australia Water Association, Canberra.

American Public Health Association (APHA), American Water Works Association (AWWA) and Water Environment Federation (WEF) (2012). Standard Methods for the Examination of Water and Wastewater, 22nd Edition. Eds. Rice EW, Baird RB, Eaton AD and Clesceri LS.

Arvin, E., Engelsen, P. and Sebber, U. (2004) Biodegradation of gasoline compounds (BTEX) in a water works sand filter. *Water Science & Technology: Water Supply*, **4**(5-6), 29-33.

do Rego, E. C. P. and Netto, A. D. P. (2007) PAHs and BTEX in groundwater of gasoline stations from Rio de Janeiro City, Brazil. *Bull. Environ. Contam. Tox.*, **79**(6), 660-664.

European Union (1998) Council Directive 98/83/EC on the quality of water intended for human consumption. Official Journal of the European Communities. L330.

NOTE: Important general information is contained in PART II, Chapter 6

FSA (2006). Survey of benzene in soft drinks. Food Survey Information Sheet No 06/06 March 2006. Food Standards Agency, UK

Health Canada (2006) Guidelines for Canadian Drinking Water Quality. Ottawa, Ontario.

Health Canada (2009) Guidelines for Canadian Drinking Water Quality. Guideline Technical Document Benzene, Ottawa, Ontario.

IARC (International Agency for Research on Cancer) (1987) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. Supplement 7., Lyon.

IPCS (1993). Environmental Health Criteria 150: Benzene. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland.

Juhler, R. K. and Felding, G. (2003) Monitoring methyl tertiary butyl ether (MTBE) and other organic micropollutants in groundwater: Results from the Danish National Monitoring Program. *Water Air Soil Poll.*, **149**(1-4), 145-161.

Kuo, H. W., Chiang, T. F., Lo, L. I., Lai, J. S., Chan, C. C. and Wang, J. D. (1997) VOC concentration in Taiwan's household drinking water. *Sci. Total Environ.*, **208**(1-2), 41-47.

Lesage, S., Hao, X., and Kent, S. (1997) Distinguishing Natural Hydrocarbons from Anthropogenic Contamination in Ground Water. *Groundwater*, 35(1), 149-160

Leusch, F. and Bartkow, M. (2010) A short primer on benzene, toluene, ethylbenzene and xylenes (BTEX) in the environment and in hydraulic fracturing fluids http://www.ehp.qld.gov.au/management/coal-seam-gas/pdf/btex-report.pdf (accessed 19 March 2013)

MoH NZ (2008) New Zealand Ministry of Health, Drinking-water standards for New Zealand 2005 (Revised 2008), p 9. Ministry of Health, Wellington.

National Toxicology Program (1986) Toxicology and carcinogenesis studies of benzene in F344/N rats and B6C3F1 mice (gavage studies). Research Triangle Park, NC, US Department of Health and Human Services (Technical Reports Series No. 289).

Schmidt, T. C., Haderlein, S. B., Pfister, R. and Forster, R. (2004) Occurrence and fate modeling of MTBE and BTEX compounds in a Swiss Lake used as drinking water supply. *Water Res.*, **38**(6), 1520-1529.

Sedran, M. A., Pruden, A., Wilson, G. J., Suidan, M. T. and Venosa, A. D. (2004) Biodegradation of methyl tert-butyl ether and BTEX at varying hydraulic retention times. *Water Environ. Res.*, **76**(1), 47-55.

US Department of Health and Human Services (US DHHS) (2011) Report on Carcinogens. 12th ed., pp. 60.

US Environmental Protection Agency (2003) Integrated risk information system: Benzene

US Environmental Protection Agency (2008) National Primary Drinking Water Standards.

Volk, H., Pinetown, C., Johnston,W. (2011) A desktop study of the occurrence of total petroleum hydrocarbon (TPH) and partially water-soluble organic compounds in Permian coals and associated coal seam groundwater. CSIRO Petroleum and Geothermal Research Portfolio Report EP-13-09-11-11 2011 (Bentley, WA, Australia). Available at http://www.agl.com.au/Downloads/Literature%20Review% (accessed 19 March 2013)

Williams, P. R. D., Benton, L. and Sheehan, P. J. (2004) The risk of MTBE relative to other VOCs in public drinking water in California. *Risk Anal.*, **24**(3), 621-634.

World Health Organization (1993) Benzene. Environmental Health Criteria No.150.

NOTE: Important general information is contained in PART II, Chapter 6

World Health Organization (2003) Background document for development of WHO Guidelines for Drinking-water Quality. WHO/SDE/WSH/03.04/24.

World Health Organization (2010) Exposure to Benzene: A Major Public Health Concern.

World Health Organization (2011) Guidelines for drinking-water quality, fourth edition.

Zein, M. M., Suidan, M. T. and Venosa, A. D. (2006) Bioremediation of groundwater contaminated with gasoline hydrocarbons and oxygenates using a membrane-based reactor. *Environ. Sci. Technol.*, **40**(6), 1997-2003.

NOTE: Important general information is contained in PART II, Chapter 6

Beryllium

(endorsed 2011)

GUIDELINE

Based on health considerations the concentration of beryllium in drinking water should not exceed 0.06 mg/L.

GENERAL DESCRIPTION

Beryllium can enter source water through the weathering of rocks, atmospheric deposition, and discharges. The primary source of beryllium in the environment is the burning of fossil fuels. Other less significant sources are slag and ash dumps.

Beryllium is used in a number of specialised applications including ceramic formulations, electrical and electronic components, and X-ray tubes. It is also used to stiffen the mantles of gas acetylene lamps.

Beryllium concentrations in drinking water overseas are generally very low, usually less than 0.001 mg/L. For example, the concentration of beryllium in drinking water was based on a survey of 1577 drinking-water samples throughout the United States, where beryllium was detected in 5.4% of samples with mean and maximum concentrations of 0.00019 and 0.00122 mg/L (0.19 and 1.22 µg/L), respectively. In Australian river waters the levels are reported to be < 0.00001 to 0.00012 mg/L (10–30 ng/L average) (WHO 2001).

If it is assumed the maximum concentration in drinking water in Australia is 0.0012 mg/L then 2 L/day will give a daily intake of 0.0024 mg beryllium. The World Health Organization (WHO 2001) reports an intake of 0.00012 mg/day from food as the mid point from an Australian survey. Thus the total intake of beryllium may be about 0.0025 mg/day. The proportion from water may approximately be as much as 95%.

Atmospheric exposure to beryllium is generally much less than from food or water, but constitutes a greater hazard. Cigarette smokers can be exposed to higher concentrations than nonsmokers.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for beryllium.

TREATMENT OF DRINKING WATER

There are no published methods for the removal of beryllium from drinking water supplies.

MEASUREMENT

The concentration of beryllium in drinking water can be determined by graphite furnace atomic absorption spectroscopy or inductively coupled plasma emission spectroscopy (APHA Method 3500-Be Parts B or C 1992). The limit of determination is approximately 0.002 mg/L.

HEALTH CONSIDERATIONS

Beryllium compounds are not readily absorbed by the gastrointestinal tract since they tend to be insoluble at pH values normally found in the gut. A significant proportion of the beryllium that is absorbed is incorporated into bone and has a biological half-life of more than one year.

The toxicology of beryllium has been reviewed by United States Environmental Protection Agency (USEPA 1998), the WHO (2001) and US Agency for Toxic Substances and Disease Registry (ATSDR 2002). Several chronic oral animal studies in dogs, rats and mice were reviewed. Dogs chronically exposed to soluble beryllium sulfate in the diet developed gastrointestinal lesions and bone marrow hypoplasia. Rickets were observed in rats exposed to sparingly soluble beryllium carbonate in the diet for 2–24 weeks, possibly due to decreased gastrointestinal absorption of phosphorus subsequent to formation of insoluble beryllium phosphate in the intestine.

WHO (2001) described the derivation of a tolerable daily intake (TDI) of 0.002 mg per kg bodyweight (mg/kg bw) by application of an uncertainty factor of 300 to a benchmark dose (BMD) of 0.46 mg/kg bw/day. The BMD was calculated at the lower 95% confidence interval for a 10% incidence of small intestinal lesions, assumed to be equal to a no-observed-adverse-effect level (NOAEL), in dogs chronically exposed to dietary beryllium sulfate tetrahydrate. The uncertainty factor of 300 included a factor of 10 intraspecies variation, 10 for interspecies variation, and a 3-fold factor for database deficiencies (no studies available on developmental effects and no mechanistic/mode of action data to suggest this may not be an issue). Although there are several chronic oral animal studies, there is a lack of human toxicity data by the oral route, reproductive/developmental end-points have not been adequately assessed, and oral studies examining immunological end-points are lacking. Since the principal study in dogs is of chronic duration and a benchmark dose was used, there are no uncertainty factors for duration or NOAEL/LOAEL (lowest observed adverse effect level) extrapolation. This derivation of the TDI uses the same logic, BMD_{10} and uncertainty factors as those described by the United States Environmental Protection Agency (USEPA 1998). The Agency for Toxic Substances and Disease Registry, using the same dog data but a different curving-fitting program to the USEPA (1998), determined a BMD_{10} of 0.56 mg/kg bw/day, to which they applied an uncertainty factor of 300 (same rationale as WHO 2001) to obtain a minimum risk level of 0.002 mg/kg bw/day (ATSDR 2002).

There are no reliable data on the human health effects of oral exposure to beryllium. Inhalation is known to cause serious health effects, with long-term exposure resulting in pulmonary granulomatosis (a type of lung tumour). The inhalation data led the International Agency for Research on Cancer to conclude that beryllium and beryllium compounds are carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans and sufficient evidence in animals) (IARC 1993).

There is no clear evidence that the compounds are carcinogenic when administered orally. Beryllium was not mutagenic in tests with different strains of bacteria, but caused chromosomal aberrations and gene mutations in cultured mammalian cells.

Experiments with laboratory mice have shown that beryllium can cross the placenta and is foetotoxic (toxic to the foetus).

DERIVATION OF GUIDELINE

Using the oral TDI of 0.002 mg/kg bw from WHO (2001), a drinking water guideline can be derived as follows:

$$0.06 \text{ mg/L} = \frac{0.002 \text{ mg/kg bw} \times 70 \text{ kg} \times 0.8}{2 \text{ L/day}}$$

Where:

- 0.002 mg/kg bw is the tolerable daily intake and includes a 300 fold safety factor to allow for intraspecies and interspecies variation.
- 70 kg is taken as the average weight of an adult.
- 0.8 is a proportionality factor based on the assumption that 80% of total daily intake is attributable to the consumption of drinking water.

NOTE: Important general information is contained in PART II, Chapter 6

- 2 L/day is the estimated maximum amount of water consumed by an adult.

REFERENCES

APHA Method 3500-Be Part B, (1992). Beryllium: Atomic Absorption Spectrophotometric method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Be Part C, (1992). Beryllium: Inductively Coupled Plasma method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

ATSDR (Agency for Toxic Substances and Disease Registry) (2002). Toxicological Profile for Beryllium. ATSDR.

DoHA (2010) *The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons*, Department of Health and Ageing, Commonwealth of Australia, Canberra.

IARC (International Agency for Research on Cancer) (1993). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Beryllium, Cadmium, Mercury, and Exposures in the Glass Manufacturing Industry. World Health Organization, IAARC 58, Lyons.

USEPA (United States Environmental Protection Agency) (1998). Toxicological review of beryllium and compounds (CAS No. 7440-41-7). USEPA, Washington, DC.

WHO (World Health Organization) (2001). Concise International Chemical Assessment Document 32: Beryllium and beryllium compounds. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Bioresmethrin

(endorsed 2011)

GUIDELINE

Based on human health concerns, bioresmethrin in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Bioresmethrin (CAS 28434-01-7) belongs to the pyrethroid class of chemicals. This is one of the largest groups of insecticides, and includes cyfluthrin, esfenvalerate, fenvalerate, permethrin, and flucythrinate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, bioresmethrin would not be a health concern unless the concentration exceeded 0.1 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Bioresmethrin is a household insecticide used for the control of flies, mosquitoes, spiders, ants, cockroaches, fleas, silverfish, and moths.

There are registered products that contain bioresmethrin in Australia. These products are intended for domestic use and are applied by spray directly onto insects or laid as baits in areas of infestation. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to bioresmethrin and its metabolites is the use of household insecticide products. Levels of exposure from these uses are low.

The domestic use of this chemical provides only limited potential for contamination of drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on occurrence of bioresmethrin in Australian waters could be found.

TREATMENT OF DRINKING WATER

No specific data on the treatment of bioresmethrin in drinking water have been identified.

MEASUREMENT

Bioresmethrin can be measured by routine gas chromatrography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for bioresmethrin is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 3 mg/kg bw/day from a 2-year dietary rat study. The NOEL is based on evidence of mild liver toxicity in the form of increased relative liver weight, discolouration, hepatocellular hypertrophy, and fatty vacuolisation (steatosis) at doses of 15 mg/kg bw/day and above. The ADI incorporates a safety factor of 100 and was established in 1991.

The previous ADI for bioresmethrin was set in 1975 at 0.3 mg/kg bw, based on a NOEL of 30 mg/kg bw/day from a medium-term dietary study in rats, which showed evidence of mild liver toxicity. The ADI was amended after submission of a long-term dietary study in rats showing a lower NOEL for liver toxicity.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Bioresmethrin is rapidly absorbed via the gastrointestinal tract and extensively metabolised. It has wide, uniform distribution including the brain and testes in mammals. The primary metabolic pathway is hydrolysis and oxidation to 5-benzyl-3-furan carboxylic acid (BFCA). Bioresmethrin is slowly eliminated in the urine and to a lessor extent in faeces, almost completely within 6 days.

Acute effects: Bioresmethrin has low acute oral and acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: Two-week studies in rats and dogs did not demonstrate any toxicity at low dose levels. Effects in rats were confined to decreased liver and thymus weights, and decreased haemoglobin levels at doses of 1000 mg/kg bw/day. No toxicity was seen in dogs at this dose level. Three-month studies in rats and dogs reported increased liver weight, fatty deposits in the liver, increased liver enzyme activity, and decreased red blood cells at doses of 100 mg/kg bw/day in rats and at 250 mg/kg bw/day in dogs.

Long-term effects: A long-term dietary study in rats reported evidence of mild liver toxicity (increased weight, hepatocellular hypertrophy, fatty deposits, and increased serum enzyme activity) at doses of 15 mg/kg bw/day. The NOEL was 3 mg/kg bw/day and this is the basis for the current ADI.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for bioresmethrin.

Genotoxicity*:* Bioresmethrin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: Short-term dietary studies in rats reported evidence suggestive of nervous system toxicity but only at extremely high dose levels, which are well in excess of the likely level of human exposure. The metabolite 5-benzyl-3-furancarboxylic acid (BFCA) is responsible for most of the nervous system toxicity from bioresmethrin in mammals.

Poisons Schedule: Bioresmethrin is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for bioresmethrin was determined as follows:

$$0.1 \text{ mg/L} = \frac{3.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 3.0 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization (WHO) has not established a health-based guideline value for bioresmethrin. This relates to the WHO's policy to exclude the use of pyrethroids for direct addition to drinking water as mosquito larvicides. This is based on concern over the developing of resistance to synthetic pyrethroids, which interferes with the global anti-malaria strategy (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, First Addendum. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Boron

(endorsed 2011)

GUIDELINE

Based on human health considerations, the concentration of boron in drinking water should not exceed 4 mg/L.

GENERAL DESCRIPTION

Boron can be present in drinking water through the natural leaching of boron-containing minerals, or by contamination of water sources. The environmental chemistry of boron is not well understood. In water, the predominant form is probably boric acid, which does not dissociate readily.

Boron compounds are used in glass manufacture, cleaners, wood and leather preservatives, flame retardants, cosmetic products, antiseptics, and occasionally food preservatives; and as agricultural fertilisers, algicides, herbicides and insecticides.

In other countries, concentrations of boron in uncontaminated water sources are usually less than 1 mg/L. Concentrations up to 6.5 mg/L have been reported in ground water supplies, but these higher concentrations are associated with seawater intrusion.

Boron is present naturally in many food products, with high amounts found in foods of plant origin, especially fruits, leafy vegetables, nuts and legumes. It has been estimated that intake of boron from food is about 10 times that from water. The daily consumption of boron is 10-25 mg. This value, however, will vary from country to country depending on population dietary habits, geographical area and soil geochemistry. In the United States, average intake values for adults range from 0.87 to 1.34 mg/day and 90 percentile intakes are about 1.5 to 2 mg/day (IOM 2001, USEPA 2008a). In Australia, the estimated dietary intake for boron is 2.2 mg/day (Samman *et al*. 1998).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Boron is not often monitored in Australian drinking water supplies, but the limited information available indicates that boron concentrations are less than 0.1 mg/L.

TREATMENT OF DRINKING WATER

The concentration of boron in drinking water can be reduced by the use of granular activated carbon, or by lime softening.

MEASUREMENT

The boron concentration in drinking water can be determined using inductively coupled plasma emission spectroscopy (APHA Method 4500-B Part D 1992). The limit of determination is approximately 0.05 mg/L.

HEALTH CONSIDERATIONS

Boron, as soluble borate (borax) or boric acid, is rapidly and completely absorbed after ingestion. It is widely distributed throughout the body and up to 90% is excreted in urine as unchanged compound.

There have been a number of reported cases of poisoning following the ingestion of high doses of boron. Symptoms include gastrointestinal disturbances, skin eruptions, and central nervous system stimulation and depression. Long-term occupational exposure to boron can lead to similar symptoms.

Short-term studies with rats and dogs reported testicular atrophy at high doses (5000 mg/kg bodyweight) of boric acid and borate. This condition was also observed in longer-term studies with rats, mice and dogs over 2 years. Reproductive studies reported that rats became sterile at the highest doses. No increase in the incidence of tumours was observed in long-term studies using mice.

A tolerable daily intake (TDI) of 0.16 mg/kg bodyweight (mg/kg bw) has been derived by the the World Health Organization (WHO 2004a). This TDI is based on the no-observed-adverse-effects level (NOAEL) of 9.6 mg/kg bw/day for foetal bodyweight effects in a rat developmental study (Price *et al.* 1996), with an uncertainty factor of 60 (10 for interspecies and 6 for human intraspecies variation).

Tests for mutagenicity using bacteria and mammalian cells have been mostly negative. Neither boric acid nor borate induced chromosomal aberrations in mammalian cells.

Boron is suspected of being a trace nutrient in mammals and some authors argue for such a role in humans (Nielson 1996), however recent reviews concluded that a clear biological function for boron in humans has yet to be established (IOM 2001, USEPA 2008b).

Although boron is an essential trace element for plants, certain plants (e.g. citrus fruit, stone fruit, some nut trees) are sensitive to the toxic effects of boron if irrigation water has concentrations higher than about 0.5 mg/L (Lazarova and Bahri 2005). WHO (2004) indicates that this concentration is below the level that can be achieved by practical treatment methods. Application of waste water containing 0.8–1.3 mg/L to young orange trees for three years was well tolerated (Reboll *et al.* 2000).

DERIVATION OF GUIDELINE

The background intake from diet is given as 2.2 mg/day (Samman *et al.* 1998) and from consumer products, 0.1 mg/day (WHO 2003). Assuming a bodyweight of 70 kg for an average adult, this is equivalent to a background intake of 0.03 mg/kg bw/day. When subtracted from the TDI of 0.16 mg/kg bw, the remaining 0.13 mg/kg bw/day can be allocated to intake from water.

When the above considerations are applied in the standard equation for deriving a drinking water guideline in Australia, a value of 4 mg/L (rounded down from 4.55 mg/L) is attained.

$$4\ \text{mg/L} = \frac{0.13\ \text{mg/kg bw/day} \times 70\ \text{kg}}{(2\ \text{L/d})}$$

Where:

- 0.13 mg/kg bw/day is the tolerable intake allocated to water.
- 70 kg is taken as the average weight of an adult.
- 2 L/day is the estimated maximum amount of water consumed by an adult.

REFERENCES

APHA Method 4500-B Part D (1992). Boron: Inductively Coupled Plasma method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

IOM (Institute of Medicine) (2001). Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Institute of Medicine, National Academy Press, Washington, D.C.

NOTE: Important general information is contained in PART II, Chapter 6

Lazarova V, Papadopoulos I, Bahri A (2005). Chapter 5: Code of successful agronomic practices. In: Lazarova V, Bahri A, (eds), *Water Reuse for Irrigation: Agriculture, Landscapes and Turf Grass*, CRC Press, Boca Raton, FL, pp. 103-150.

Neilson FH (1996). Evidence for the Nutritional Essentiality of Boron. *Journal of Trace Elements in Experimental Medicine*, 9:215–229.

Price CJ, Strong PL, Marr MC, Myers CB, Murray FJ (1996). Developmental toxicity NOAEL and postnatal recovery in rats fed boric acid during gestation. *Fundamental and Applied Toxicology,* 32(2):179-193.

Reboll V, Cerezo M, Roig A, Flors V, Lapena L, Garcia-Agustin P (2000). Influence of wastewater vs groundwater on young Citrus trees. *Journal of the Science of Food and Agriculture,* 80:1441-1446.

Samman S, Naghii MR, Lyons Wall PM, Verus AP (1998). The nutritional and metabolic effects of boron in humans and animals. *Biologic Trace Element*, 66,1–9.

USEPA (United States Environmental Protection Agency) (2008a). Drinking Water Health Advisory for Boron. Health and Ecological Criteria Division, Office of Science and Technology, Office of Water, USEPA Document Number: 822-R-08-013, May 2008.

USEPA (United States Environmental Protection Agency) (2008b). Health Effects Support Document for Boron, USEPA, Office of Water, Health and Ecological Criteria Division, EPA-822-R-08-002, January 2008.

WHO (World Health Organization) (2003). Boron in drinking-water. Background document for preparation of WHO Guidelines for drinking-water quality. WHO, Geneva (WHO/SDE/WSH/03.04/54).

WHO (World Health Organization) (2004a). Boron in Drinking Water. Background document for development of WHO Guidelines for Drinking-water Quality, WHO, Geneva, Switzerland. WHO/SDE/WSH/03.04/54.

WHO (World Health Organization) (2004b). Guidelines for Drinking-water Quality. First addendum to 3rd edition. Volume 1. Recommendations. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Bromacil

(endorsed 2011)

GUIDELINE

Based on human health concerns, bromacil in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Bromacil (CAS 314-40-9) belongs to the urea group of chemicals. There are no other pesticides in this group of chemicals (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, bromacil would not be a health concern unless the concentration exceeded 0.4 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Bromacil is a herbicide for the control of weeds and grasses in fruit orchards and plantations, commercial and industrial areas, rights of way and around agricultural buildings.

There are currently products registered in Australia that contain bromacil or its sodium salts. Bromacil products are intended for professional use. The products are available as powdered or granulated formulations and applied in diluted form using ground or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to bromacil and its metabolites are residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of bromacil may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of bromacil in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Advanced oxidation using ultraviolet irradiation and peroxide has been demonstrated to achieve a moderate level of bromacil removal (Kruithof *et al.* 2002).

MEASUREMENT

Bromacil can be measured in drinking waters by solid phase extraction followed by liquid chromatography–mass spectrometry.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for bromacil is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 10 mg/kg bw/day from a long-term (2-year dietary) study. The NOEL is based on decreased bodyweight and increased relative thyroid weight in rats. The ADI incorporates a safety factor of 100 and was established in 1988.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Following gastrointestinal absorption, bromacil is excreted via the urine in rats and humans mainly as the metabolite 5-bromo-3-sec-butyl-6-hydroxymethyluracil. Urine samples from the formulation workers indicated that both bromacil and the major metabolite occur in a hydrolysed form. Five other metabolites have been identified.

Acute effects: Bromacil has low acute oral and dermal toxicity. Bromacil is a skin sensitiser in a guinea-pig test.

Short-term effects: In repeat-dose (90-day) dietary studies in rats, the target organs of toxicity were the liver, kidney and thyroid. At dose levels above 2.5 mg/kg bw/day, food consumption and relative organ weights were decreased. Toxic effects were seen in the liver and thyroid at the higher dose levels.

Long-term effects: In an 18-month dietary study, mice were fed at doses up to 714 mg/kg bw/day of bromacil. The reported effects were in the liver and testicle at high doses.

Two-year studies were conducted in rats and dogs. In rats, the reported effects were decreased bodyweight and increased relative thyroid weight at doses above 10 mg/kg bw/day. There were no effects reported in dogs up to the dose level of 30 mg/kg/day. The NOEL of 10 mg/kg/day found in the rat study was used to establish an ADI of 0.1 mg/kg bw/day for bromacil.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for bromacil.

Genotoxicity: Bromacil is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Three-generation reproduction and developmental studies in rats and rabbits showed no adverse effects on reproduction in either species. Evidence of delayed development occurred in rats and rabbits but only at maternally toxic doses which were at levels well in excess of the likely level of human exposure.

Poisons Schedule: Bromacil is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for bromacil was determined as follows:

$$0.4 \text{ mg/L} = \frac{10 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 10 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Kruithof J, Kamp P, Belosevic M (2002). UV/H_2O_2 treatment: the ultimate solution for pesticide control and disinfection. *Water Science Technology: Water Supply*, 2(1): pp 113-122

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Bromate

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of bromate in drinking water should not exceed 0.02 mg/L.

GENERAL DESCRIPTION

Bromate is not a normal component of water but may be formed from bromide during ozonation. Concentrations up to 0.09 mg/L have been reported in ozonated drinking water. Bromate is a strong oxidant and will probably react with organic matter in water, forming bromide as a by-product.

Bromate is used in home hair permanent-wave neutralising solutions. Although it is used in some foods overseas, Australian Food Standards do not allow bromate to be used in food in Australia.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

It is unlikely that bromate would be present in Australian reticulated drinking water supplies unless ozonation is used for disinfection.

TREATMENT OF DRINKING WATER

There are no published methods for the removal of bromate from drinking water supplies.

MEASUREMENT

The concentration of bromate in drinking water can be determined using ion chromatography with conductivity detection. The limit of determination is about 0.005 mg/L.

HEALTH CONSIDERATIONS

Bromate is rapidly absorbed from the gastrointestinal tract of rats. Although bromate was not subsequently detected in tissue, bromide concentrations were significantly increased in plasma, red blood cells, pancreas, kidney, stomach and small intestine.

Most cases of human poisoning from bromate are due to accidental or intentional ingestion of home permanent-wave solutions, which can contain 2–10% bromate. Toxic effects include nausea, abdominal pain and diarrhoea, central nervous system depression and pulmonary oedema, most of which are reversible. Irreversible effects include kidney failure and deafness.

In rats exposed to bromate in drinking water for 15 months, adverse effects included inhibited body-weight gain, marked kidney damage, and renal adenocarcinoma. Kidney tumours have been reported in other long-term studies using male and female rats, but not with female mice; male rats also exhibited peritoneal mesotheliomas. There is evidence that tumours occur only after a minimum total cumulative dose has been exceeded.

Bromate exhibited mutagenic activity in tests using bacteria, and caused chromosomal aberrations in cultured mammalian cells. Some evidence of DNA damage has also been reported in rats given potassium bromate.

The International Agency for Research on Cancer has concluded that bromate is possibly carcinogenic to humans (Group 2B, no data in humans but sufficient evidence in animals) (IARC 1986).

DERIVATION OF GUIDELINE

The guideline value for bromate in drinking water was derived as follows:

$$0.02 \text{ mg/L} = \frac{30 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day} \times 10{,}000}$$

where:

- 30 mg/kg body weight per day is a lowest effect level from a 15-month drinking water study using rats (Nakano ***et al***. 1989).
- 70 kg is the average weight of an adult.
- 0.2 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 10,000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations, 10 because a lowest effect level was used instead of a no-effect level and 10 for carcinogenic and mutagenic effects).

The World Health Organization (WHO) guideline value of 0.025 mg/L was based on a calculation that estimated an additional lifetime risk of seven fatal cancers per 100,000 people. It was recognised that this approach may not be appropriate if, as reported, tumours only occur above a dose threshold. The two different approaches, however, result in essentially the same guideline value.

This guideline should be reviewed when new data are available.

REFERENCES

IARC (International Agency for Research on Cancer) (1986). IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: some naturally occurring and synthetic food components, furocoumarins, ultraviolet radiation and potassium bromate. World Health Organization, IARC, 40.

Nakano K, Okada S, Toyokuni S, Midorikawa O (1989). Renal changes induced by chronic oral administration of potassium bromate or ferric nitrilotriacetate in Wistar rats. (In Japanese). *Japanese Archives of Internal Medicine*, 36:41–47.

NOTE: Important general information is contained in PART II, Chapter 6

Bromoxynil

(endorsed 2011)

GUIDELINE

Based on human health concerns, bromoxynil in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Bromoxynil (CAS 1689-84-5) is in the hydroxybenzonitrile class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, bromoxynil would not be a health concern unless the concentration exceeded 0.01 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Bromoxynil is a post-emergent herbicide used to control broad-leaf weeds.

There are registered products containing bromoxynil in Australia. These products are intended for professional and home garden use and are applied by aircraft or boom spray by professional use, and by hand-held spray in the home garden. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to bromoxynil are use in the home garden and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of bromoxynil may potentially lead to the contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of bromoxynil in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Current literature suggests that bromoxynil can be effectively removed by oxidation with ultraviolet (UV) radiation, UV and ultraviolet peroxide, chlorine and UV, ozonation, and by chlorination, with various levels of success depending on the water quality conditions (Guittonneau *et al.* 2005). Ozonation is considered the most effective, with chlorination the least effective (Guittonneau *et al.* 2005). Jar testing to identify the effectiveness of various oxidants in specific waters is recommended if bromoxynil is detected. Oxidation of any kind will result in the formation of by-products and therefore a by-product management

plan is also recommended.

Photodegradation has shown to provide some removal of bromoxynil (Texier *et al.* 1998); however more research is required to determine the optimal conditions. Activated carbon has also been shown to remove bromoxynil effectively from water, but is dependant on the water quality and the application methods of the carbon (Yang *et al.* 2004).

MEASUREMENT

The practical limit of quantification for bromoxynil in water is 0.001 mg/L, using liquid chromatography–tandem mass spectrometry (Alder *et al.* 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for bromoxynil is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.3 mg/kg bw/day from a 12-month dietary study in dogs. The ADI incorporates a safety factor of 100 and was established in 1993.

The previous *Australian Drinking Water Guidelines* health value was 0.03mg/L (NHMRC and NRMMC 2004)

HEALTH CONSIDERATIONS

Metabolism: Bromoxynil is readily absorbed via the gastrointestinal tract and widely distributed in the body in mammals. Virtually all absorbed bromoxynil is eliminated unchanged. It is slowly excreted mainly via the faeces, with some excretion in urine. Bromoxynil has a low potential for bioaccumulation.

Acute effects: Bromoxynil has a moderate acute oral toxicity and low dermal toxicity. There is no evidence of skin sensitisation.

Short-term effects: In medium-term dietary studies in dogs, there was decreased bodyweight gain, increased blood urea nitrogen, and kidney and liver weight changes at a dose of 12 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in rats and dogs. A 2-year rat study reported decreased bodyweight gain at 8.2 mg/kg bw/day and evidence of adverse effects in the liver, kidney and stomach at 26 mg/kg bw/day. A 1-year dog study reported reduced bodyweight gain at 1.5 mg/kg bw/day and evidence of anaemia at 7.5 mg/kg bw/day. The NOEL of 0.3 mg/kg bw/day in dogs is the basis of the ADI.

Carcinogenicity: Benign liver tumours were slightly increased in mice at 1.5 mg/kg bw/day and above, but these tumours were considered to be specific to mice and not of human relevance. Based on this and a 2-year study in rats, bromoxynil is not considered to have carcinogenic potential in humans.

Genotoxicity: Bromoxynil is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Bromoxynil is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for bromoxynil was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.3 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.3 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Alder L, Greulich K, Kempe G, Vieth B (2006). Residue analysis of 500 high priority pesticides: better by GC-MS or LC-MS/MS? *Mass Spectrometry Reviews*, 25(6):838-65.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Guittonneau S, Momege S, Schafmeier A, Viac P, Meallier P (2005). Dehalogenation of the herbicides bromoxynil (3,5-dibromo-4- hydroxybenzonitrile) and ioxynil (3,5-diiodino-4-hydroxybenzonitrile) by *Desulfitobacterium chlororespirans*. *Applied and Environmental Microbiology*, 71, 7, 3741-3746.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Texier I, Giannotti C, Malato S, Richter C, Delaire J (1998). Solar photodegradation of pesticides water by sodium decatungstate. In: *Proceedings of the 1998 9th SolarPaces International Symposium on Solar Thermal Concentrating Technologies*. Editions de Physique Font-Romeu.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Yang Y, Chun Y, Shang G, Huang M (2004). pH-dependence of pesticide adsorption by wheat-residue-derived black carbon. *Langmuir*, 20(16):6736-6741.

NOTE: Important general information is contained in PART II, Chapter 6

NOTE: Important general information is contained in PART II, Chapter 6

Cadmium

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of cadmium in drinking water should not exceed 0.002 mg/L.

GENERAL DESCRIPTION

Contamination of drinking water by cadmium may occur as a result of impurities in the zinc of galvanised pipes or in solders used in fittings, water heaters, water coolers and taps. Cadmium can also be released to the environment in waste water, through contamination of fertilisers, and by metallurgical industries.

Cadmium metal is used as an anticorrosive coating on steel but its use is being phased out. Cadmium compounds are commonly used as pigments in plastics, in batteries and in some electrical components.

Cadmium concentrations in nonpolluted natural waters overseas are usually lower than 0.001 mg/L.

Food is the main source of cadmium intake. The estimated average Australian adult dietary intake of cadmium is approximately 0.03 mg per day. Smoking is a significant additional source of cadmium.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies concentrations of cadmium are usually less than 0.002 mg/L.

TREATMENT OF DRINKING WATER

Cadmium can be effectively removed from drinking water by lime softening (98% removal in the pH range 8.5 to 11.3) and coagulation with ferric chloride (90% removal above pH 8 but less effective at lower pH).

MEASUREMENT

The cadmium concentration in drinking water can be determined using graphite furnace atomic absorption spectroscopy (APHA Method 3500-Cd Part B 1992). The limit of determination is approximately 0.0002 mg/L.

HEALTH CONSIDERATIONS

Absorption of cadmium in the gastrointestinal tract depends on a number of factors including the solubility of the compounds ingested, but a healthy person typically absorbs 3–7% of ingested cadmium. This figure may be higher in people with iron, calcium and protein deficiency. Cadmium accumulates in the kidney and is only released very slowly, with a biological half-life in humans of 10 to 15 years.

An extensive review and summary of the human and animal toxicity data for cadmium is available (IPCS 1992).

In humans, long-term exposure can cause kidney dysfunction leading to the excretion of protein in the urine. This may occur, in a certain proportion of people, if the amount of cadmium exceeds 200 mg/kg renal cortex tissue; about 10% of the population is estimated to possess this sensitivity. Other effects can include osteomalacia (softening of the bones). Cases of Itai-Itai disease have been reported in Japan among elderly women exposed to highly contaminated food and water. Symptoms are similar to osteomalacia accompanied by kidney dysfunction characteristic of cadmium poisoning.

Epidemiological studies have looked for a connection between lung cancer and workplace cadmium inhalation, but the results have been inconclusive.

Long-term inhalation studies with rats have reported an increase in the incidence of tumours of the lung. No increase in the incidence of tumours was found when cadmium salts were administered orally.

There is no clear evidence that cadmium is mutagenic. Many tests have reported negative results but there have been some reports of gene mutation and chromosome abnormalities in mammalian cells. The positive results are reported as being weak and only present at high concentrations.

The International Agency for Research on Cancer has concluded that cadmium is probably carcinogenic to humans (Group 2A, limited evidence of carcinogenicity in humans and sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value for cadmium in drinking water was derived as follows:

$$0.002 \text{ mg/L} = \frac{0.0007 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day}}$$

where:

- an intake of less than 0.0007 mg/kg body weight per day will ensure that over a 70 year lifetime, cadmium in the body will be kept below the critical amount of 200 mg/kg renal cortex tissue (JEFCA 2000). This figure was based on calculations that take into account an absorption rate of 5%, a daily excretion rate of 0.005% of body burden, and an adequate safety factor.
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

No additional safety factors are necessary as they have been included in the intake value.

The guideline value takes into account the higher cadmium intake, per kilogram of body weight, by infants and children.

The World Health Organization guideline value of 0.003 mg/L is slightly different due to rounding in the calculation. The difference is not significant.

REFERENCES

APHA Method 3500-Cd Part B, (1992). Cadmium: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1992). Cadmium. Environmental Health Criteria, 134. World Health Organization, IPCS.

JECFA (Joint FAO/WHO Expert Committee on Food Additives) (2000) *Summary and conclusions of the fifty-fifth meeting, Geneva, 6–15 June 2000*. Geneva,World Health Organization, Joint FAO/WHO Expert Committee on Food Additives.

NOTE: Important general information is contained in PART II, Chapter 6

Captan

(endorsed 2011)

GUIDELINE

Based on human health concerns, captan in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Captan (CAS 133-06-2) belongs to the phthalimide class of chemicals. There are currently no other pesticides in this class in use (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, captan would not be a health concern unless the concentration exceeded 0.4 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Captan is a fungicide for the control of fungal diseases in turf, ornamentals, agricultural crops and seedlings.

There are registered products that contain captan in Australia. These products are intended for professional use and are available as water-dispersible granules or as a dust. Captan is applied to turf, ornamentals, and agricultural crops as a dilute or concentrated spray using ground or hand-held equipment. Captan is applied as a powder to seedlings. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to captan is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of captan may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on captan occurrence in Australian drinking water supplies were found. In the Nile river, captan has been found at 0.0003 mg/L (0.3 µg/L) (Abbassy *et al.* 1999).

TREATMENT OF DRINKING WATER

Captan can be degraded by ozone, ultraviolet (UV) irradiation and chlorine. Destruction of captan in water using short wavelength UV light has been reported (Peterson *et al.* 1990). Captan is also rapidly degraded in 50 and 500 mg/L chlorine solutions at pH 7 and 10.7 (Ong *et al.* 1996). Ozonation is effective in degrading captan and degradation rates increased at higher pH and temperature (Ong *et al.* 1996).

MEASUREMENT

Captan can be extracted by liquid/liquid extraction with dichloromethane. The extract is dried with sodium sulfate, concentrated, and analysed by gas chromatography–mass spectrometry in selected ion monitoring mode. The method can achieve a limit of detection (LOD) of 0.0005 mg/L (0.5 µg/L). Captan can also be extracted using solid-phase extraction (SPE) and measured using high performance liquid chromatography (HPLC) (Marvin *et al.* 1990, Wang *et al.* 2007) or gas chromatography/negative chemical ionization-mass spectrometry (Barreda *et al.* 2006). A fully automated system for on-line SPE followed by HPLC with tandem detection with a photodiode array detector and a fluorescence detector (after post-column derivatisation) can achieve a LOD of 0.001 mg/L (1 µg/L) (Patsias and Papadopoulou-Mourkidou 1999).

Trace-level determination of captan can be achieved by solid-phase micro-extraction and gas chromatography coupled with electron-capture detection (LOD=0.000015 mg/L [0.015 µg/L]) or with mass spectrometric detection (LOD=0.00004 mg/L [0.04 µg/L]) (Lambropoulou *et al.* 2000). Captan can be analysed by EPA Method 617 (Determination of organohalide pesticides and PCBs in industrial wastewater) using electron capture gas chromatography or EPA Method 8081A (Organochlorine pesticides, by gas chromatography: capillary column technique) using electron capture detector or an electrolytic conductivity detector.

An optical biosensor consisting of a glutathione-S-transferase-immobilized gel film can detect captan in contaminated water at 0.002 mg/L (2 µg/L) (Choi *et al.* 2003). A spectrophotometric method for the determination of captan based on its reaction with thiosemicarbazide can achieve a LOD of 0.0005 mg/L (0.5 µg/L) at an absorbance of 315 nm (Galeano *et al.* 2002).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for captan is 0.1 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 10 mg/kg bw/day from a short-term (developmental toxicity) study in rabbits. The NOEL is based on decreased bodyweight and food consumption in dams and associated foetotoxicity at 30 mg/kg bw/day and above. The ADI incorporates a safety factor of 100, and was established in 1997.

The acute reference dose (ARfD) of 0.1 mg/kg bw/day for captan was established in 1997, based on a NOEL of 10 mg/kg bw/day from a developmental study in rabbits. The ARfD incorporates a safety factor of 100.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Captan is well absorbed via the gastrointestinal tract in mice and rats. In rats, the highest levels were found in kidneys, liver, blood or gastrointestinal tract, particularly in the distal small intestine. In mice, higher levels were found in the duodenum and large intestine. In mice and rats, captan is rapidly and extensively excreted. In mice, captan is excreted in the urine as metabolites (44%), in the faeces mostly unchanged (22%), and in expired air as CO_2 (19%). In rats, captan is extensively metabolised and rapidly excreted mostly in the urine (80-90%) and the remainder in the faeces. The major urinary metabolites in both mice and rats were thiazolidine-2-thione-4-carboxylic acid (TTCA) and dithiobis-methanesulfonic acid derivatives.

NOTE: Important general information is contained in PART II, Chapter 6

In human volunteers captan was rapidly excreted in urine as TTCA (4-9%) and tetrahydrophthalimide (THPI) (1-3%).

Acute effects: Captan has low acute oral and dermal toxicity. It is a skin sensitiser in both laboratory animals and humans.

Short-term effects: Short-term dietary studies in mice reported dose-related hyperplasia in the duodenum and associated inflammatory cell response at doses of 120 mg/kg bw/day and above. Decreased bodyweight gain, and hyperplasia and hypertrophy in the glandular forestomach were reported at higher doses.

Long-term effects: Long-term dietary studies in mice reported a dose-related increase in the incidence of hyperplastic lesions and benign and malignant tumours in the duodenum at 122 mg/kg bw/day. At higher doses, there was also an increase in benign and malignant tumours in the jejunum/ileum. In long-term dietary studies in rats, decreased bodyweight gain was observed at 96 mg/kg bw/day. Long-term dietary studies in dogs reported no treatment-related effects up to doses of 300 mg/kg bw/day.

Carcinogenicity: Long-term studies in rats provide no evidence of carcinogenicity for captan. In long-term studies in mice, tumours in the duodenum were observed but were considered to occur via an inflammatory mechanism specific to mice, and were reported at dose levels well in excess of the likely level of human exposure.

Genotoxicity: Captan was positive in some *in vitro* short-term assays, but overall it was not considered to be genotoxic.

Reproductive and developmental effects: One and three-generation reproduction studies in rats did not produce evidence of reproductive toxicity. In developmental toxicity studies in rabbits, there was no effect on the foetus at doses that were not maternotoxic. These doses are well in excess of the likely level of human exposure in drinking water. The most sensitive effects reported were decreased bodyweight and food consumption in dams, and in the foetus there was an associated increased incidence of cysts on the liver and increased number of skeletal variations at doses of 30 mg/kg bw/day and above. The NOEL for these effects was 10 mg/kg bw/day, and this is the basis for the current ADI.

Poisons Schedule: Captan is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for captan was determined as follows:

$$0.4 \text{ mg/L} = \frac{10 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 10 mg/kg bw/day is the NOEL based on a short-term (developmental toxicity) study in rabbits.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Abbassy MS, Ibrahim HZ, el-Amayem MM (1999). Occurrence of pesticides and polychlorinated biphenyls in water of the Nile river at the estuaries of Rosetta and Damiatta branches, north of Delta, Egypt. *Journal of Environmental Science and Health B,* 34(2):255-67.

Barreda M, Lopez FJ, Villarroya M, Beltran J, Garcia-Baudin JM, Hernandez F (2006). Residue determination of captan and folpet in vegetable samples by gas chromatography/negative chemical ionization-mass spectrometry. *Journal of AOAC International,* 89(4):1080-7.

Choi JW, Kim YK, Song SY, Lee IH, Lee WH (2003). Optical biosensor consisting of glutathione-S-transferase for detection of captan. *Biosensors and Bioelectronics,* 18(12):1461-6.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Galeano T, Guiberteau A, Mora NM, Alvarez PR, Salinas F (2002). Spectrophotometric determination of the fungicide captan. *Journal of Environmental Science and Health B,* 37(6):533-40.

Lambropoulou DA., Konstantinou IK, Albanis TA.(2000). Determination of fungicides in natural waters using solid-phase microextraction and gas chromatography coupled with electron-capture and mass spectrometric detection. *Journal of Chromatography A,* 893(1):143-56.

Marvin CH, Brindle ID, Hall CD, Chiba M (1990). Development of an automated high-performance liquid chromatographic method for the on-line pre-concentration and determination of trace concentrations of pesticides in drinking water. *Journal of Chromatography,* 503(1):167-76.

Ong KC, Cash JN, Zabik MJ, Siddiq M, Jones AL (1996) Chlorine and ozone washes for pesticide removal from apples and processed apple sauce. *Food Chemistry,* 55(2):153-160(8).

Patsias J, Papadopoulou-Mourkidou E (1999). A fully automated system for analysis of pesticides in water: on-line extraction followed by liquid chromatography-tandem photodiode array/postcolumn derivatization/fluorescence detection. *Journal of AOAC International,* 82(4):968-81.

Peterson D, Watson D, Winterlin W (1990). Destruction of pesticides and their formulations in water using short wavelength UV light. *Bulletin of Environmental Contamination and Toxicology,* 44(5):744-50.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Wang S, Yu Y, Tan P, Miao Z, Wei Y (2007) [Simultaneous determination of captan and folpet pesticide residues in apples by solid-phase extraction and high performance liquid chromatography]. *Se Pu,* 25(2): 226-9.

NOTE: Important general information is contained in PART II, Chapter 6

Carbaryl

(endorsed 2011)

GUIDELINE

Based on human health concerns, carbaryl in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

Carbaryl (CAS 63-25-2) is in the carbamate class of chemicals. Other pesticides in this class include aldicarb, methomyl, oxamyl and pirimicarb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, carbaryl would not be a health concern unless the concentration exceeded 0.03 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Carbaryl is an insecticide effective against a broad range of insects, mites, lice, millipedes and other pests.

There are registered products containing carbaryl in Australia. These products are for professional and home garden use on lawns and turf; on horses, ponies and dogs; and other agricultural uses. They are applied as a wettable formulation spray domestically or by aerial spraying or boom spray in agriculture. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to carbaryl are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of carbaryl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Monitoring studies for carbaryl in raw and tap water are limited. No specific data from Australia were found. Carbaryl has been detected in water at µg/L concentrations but degradation is relatively rapid, with 1-naphthol identified as the major degradation product. Indirect and direct photolysis of carbaryl produces different naphthoquinones as well as some hydroxyl substituted naphthoquinones (Gunasekara ***et al***. 2008).

Carbaryl is one of the three most frequently detected insecticides in surface water, according to US Geological Survey reports from 1991-2001. It is estimated that higher levels of carbaryl occur in surface water than in groundwater, based on available monitoring data and modeling. A 3-year monitoring study undertaken in the USA revealed that carbaryl was present in drinking water sources in the majority of the monitored sites (13 of 16 sites). The concentrations measured at these sites were low (0.002 to 0.031 µg/L) in raw water and generally lower in treated drinking water (USEPA 2004).

TREATMENT OF DRINKING WATER

It is estimated that conventional drinking water treatment (i.e. coagulation, flocculation and settling) can reduce carbaryl concentration by 43% of the concentration prior to treatment. (USEPA 2004). Therefore, it is estimated that a carbaryl concentration below 0.05 mg/L should be achievable by conventional drinking-water treatment (WHO 2008). Ozone has been shown to be 99% effective in removing carbaryl from water. Similarly, granular activated carbon adsorption can effectively remove carbaryl during treatment. However, chlorine and hypochlorite may be ineffective at removing or degrading carbaryl. Softening of hard waters will reduce carbaryl concentrations (via alkaline hydrolysis), as softening raises the pH of the water (USEPA 2004).

High and stable removal efficiency of carbaryl can also be achieved using an anion exchange membrane anodic fenton treatment. This treatment oxidises carbaryl to 1-naphthol and 1,4-naphthoquinone (Wang *et al*. 2002, Kong *et al*. 2007).

MEASUREMENT

There are several methods for analysing carbaryl in drinking water. Its concentration in drinking-water may be determined by extraction, hydrolysis, derivatisation and separation by gas-liquid chromatography with electron capture detection or mass spectrometry. Detection limits vary according to the method, but typical limits of detection (LODs) of 0.2 µg/L are reported, and high-performance liquid chromatography (HPLC) with mass spectrometry is considered the conventional method of analysis.

HPLC using pre-concentration, elution, separation and ultraviolet determination can achieve a LOD in tap water between 0.03-0.2 µg/L (Driss *et al*. 1993). Another study using HPLC with fluorometric detection reported a LOD of 1.4 mg/mL for carbaryl (Massey *et al*. 1995). However, HPLC with fluorometric detection does not always provide the required specificity for determining carbaryl residues (Makihata *et al*. 2003). Solid-phase extraction followed by reversed-phase liquid chromatography can achieve LODs in the range 3-15 ng/L for the determination of nine N-methylcarbamate pesticides, including carbaryl, from drinking water (Morrica *et al*. 2005). Liquid chromatography electrospay ionisation tandem mass spectrometry, without a concentration procedure, has also been used for the measurement of carbaryl (LOD 2 µg/L) (Makihata *et al*. 2003).

Carbaryl can be analysed using the United States Environmental Protection Agency (USEPA) method 8270: Semivolatile Organic Compounds by Gas Chromatography/Mass Spectrometry (GC/MS): Capillary Column Technique, or the USEPA method 531: Direct Aqueous Injection high performance liquid chromatography (HPLC) with Post Column Derivatisation.

Other methods include immunoassay techniques, which achieve a LOD of 1.38 µg/L (Mauriz *et al*. 2006), or direct analysis by laser-induced fluorescence, which achieves a LOD in tap water of 20 ng/L (Burel-Deschamps *et al*. 2006).

NOTE: Important general information is contained in PART II, Chapter 6

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for carbaryl is 0.008 mg per kg of bodyweight (mg/kg bw) based on a lowest-observed-effect level (LOEL) of 16 mg/kg bw/day observed in a 2-year dietary study in mice. The ADI incorporates a safety factor of 2000 and was established in 2002. There is an additional factor of 5 is for the inadequate database and an additional factor of 4 for the seriousness of the carcinogenic response.

The acute reference dose (ARfD) of 0.01 mg/kg bw/day for carbaryl was established in 2002, based on a no-observed-effec leve (NOEL) of 1 mg/kg bw/day from a medium-term (13-week) and neurotoxicity study in rats, where there were behavioural indications of autonomic neurotoxicity and brain, plasma and erythrocyte cholinesterase depression. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Carbaryl is rapidly absorbed via the gastrointestinal tract in rodents and humans and is extensively metabolised. Excretion is predominantly via the urine, where ten metabolites have been identified. The potential for bioaccumulation is low.

Acute effects: Carbaryl has moderate oral toxicity and low dermal toxicity. Symptoms of acute poisoning include hyperexcitability, salivation, bronchoconstriction, headache, and vomiting. Carbaryl does not cause skin sensitisation.

Short-term: In a short-term dietary study in dogs, the major effect was depression of plasma cholinesterase at dose levels greater than 1.4 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in rats and dogs. In a 2-year dietary study in rats, there was decreased bodyweight gain and cholinesterase inhibition at 70 mg/kg bw/day. A 1-year dietary study in dogs showed cholinesterase inhibition at 3.8 mg/kg bw/day and above,

Carcinogenicity: Studies in rodents reported renal and urinary bladder tumours in rats and liver tumours in mice at high dose levels only. These tumours are considered to develop via a non-genotoxic mechanism at dose levels greatly exceeding the likely human exposure level. In mice, vascular tumours were also observed at the lowest dose of 16 mg/kg bw/day. This LOEL is the basis for the ADI.

Genotoxicity: Carbaryl caused chromosome breakage in some *in vitro* studies, but overall, it was not considered to be genotoxic.

Reproductive and developmental effects: A 2-generation reproduction toxicity study in rats reported maternotoxicity and reduced pup survival at 4.7 mg/kg bw/day. Developmental studies in rats and rabbits reported maternotoxicity and foetotoxicity at 30 mg/kg bw/day and 150 mg/kg bw/day respectively. There was no evidence of teratogenicity.

Neurotoxicity: A 13-week oral neurotoxicity study in rats reported blood and brain cholinesterase depression and behavioural effects at 1 mg/kg bw/day and above. No pathological changes were noted.

Poisons Schedule: Carbaryl is included in Schedule 4, 5 or 6 in the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) No.1 (2010), depending on the concentration and use of the product. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for carbaryl was determined as follows:

$$0.03 \text{ mg/L} = \frac{16.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 2000}$$

where:

- 16.0 mg/kg body weight per day is the LOEL for the formation of vascular tumours observed in a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the average maximum amount of water consumed by an adult.
- 2000 is the safety factor applied to the LOEL derived from animal studies. This safety factor incorporated a factor of 10 for interspecies extrapolation and 10 for intraspecies variation. There is an additional factor of 10 for the use of a LOEL and an additional factor of 2 for the uncertainty as to the mode/mechanism of vascular tumour formation and for the inability to dismiss the relevance of vascular tumours to humans.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Burel-Deschamps L, Giamarchi P, Stephan L, Lijour Y, Le Bihan A (2006). Laser-induced fluorescence detection of carbamates traces in water. *Journal of Fluorescence*, 16(2):177-83.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Driss MR, Hennion MC, Bouguerra ML (1993). Determination of carbaryl and some organophosphorus pesticides in drinking water using on-line liquid chromatographic preconcentration techniques. *Journal of Chromatography*, 639(2):352-8.

Gunasekara AS, Rubin AL, Goh KS, Spurlock FC, Tjeerdema RS (2008). Environmental fate and toxicology of carbaryl. *Reviews of Environmental Contamination and Toxicology*, 196: 95-121.

Kong L, Lemley AT (2007). Effect of nonionic surfactants on the oxidation of carbaryl by anodic Fenton treatment. *Water Research,* 41(12):2794-802.

Makihata N, Kawamoto T, Teranishi K (2003). Simultaneous analysis of carbamate pesticides in tap and raw water by LC/ESI/MS. *Analytical Sciences,* 19(4): 543-9.

Massey KA, Van Engelen DL, Warner IM (1995). Determination of carbaryl as its primary metabolite, 1-naphthol, by reversed-phase high-performance liquid chromatography with fluorometric detection. *Talanta*, 42(10):1457-63.

Mauriz E, Calle A, Abad A, Montoya A, Hildebrandt A, Barcelo D, Lechuga LM (2006). Determination of carbaryl in natural water samples by a surface plasmon resonance flow-through immunosensor. *Biosensors and Bioelectronics,* 21(11):2129-36.

Morrica P, Fidente P, Seccia S (2005). Liquid chromatographic determination of nine N-methylcarbamates in drinking water. *Biomedical Chromatograpy,* 19(1):107-10.

NOTE: Important general information is contained in PART II, Chapter 6

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2004). *Carbaryl Interim Reregistration Eligibility Decision (IRED) Facts*. Office of Prevention, Pesticides and Toxic Substances, Washington.

Wang Q, Lemley AT (2002). Oxidation of carbaryl in aqueous solution by membrane anodic fenton treatment. *Journal of Agricultural and Food Chemistry,* 50(8):2331-7.

WHO (World Health Organization) (2008). Carbaryl in Drinking-water. Background document for preparation of WHO Guidelines for Drinking-water quality. Geneva, World Health Organization (WHO/HSE/AMR/08.03/5).

NOTE: Important general information is contained in PART II, Chapter 6

NOTE: Important general information is contained in PART II, Chapter 6

Carbendazim/Thiophanate-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, carbendazim in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Carbendazim (CAS 10605-21-7/ CAS 23564-06-9) belongs to the benzimidazole class of chemicals. Thiophanate methyl belongs to the thiophanate class of chemicals, although it is often regarded as a benzimidazole. Thiophanate-methyl is converted to carbendazim in the environment (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse of thiophanate-methyl or carbendazim, it would not be a health concern unless the concentration exceeded 0.09 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Carbendazim is a broad-spectrum systemic fungicide used for the control of a wide range of fungal diseases such as mould, spot, mildew, scorch, rot and blight in a variety of crops, including cereals, fruit (pome, stone, citrus, cucurbits, strawberries, bananas, mangoes, etc), vines, ornamentals, pasture, and turf.

Thiophanate-methyl is a fungicide used for the control of soil-borne diseases of ornamental plants.

There are registered products containing carbendazim and registered products containing thiophanate-methyl in Australia. Carbendazim-containing products are intended for professional use and are generally available as suspension concentrates, to be mixed with water for application as a spray or dip. Some products are also used as timber preservatives. Thiophanate-methyl containing products are intended for professional use and are available as concentrated powders or granules, to be mixed into soil/potting mix before sowing or applied as a diluted drench directly to plant beds. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: Carbendazim is the major environmental degradant of thiophanate-methyl and is therefore the relevant environmental contaminant for both chemicals. The main source of public exposure to carbendazim is residues in food. Residue levels in crops grown according to good agricultural practice are generally low.

Agricultural use of carbendazim and/or thiophanate-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of carbendazim in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of carbendazim in drinking water have been identified.

MEASUREMENT

Carbendazim may be measured in drinking water using high performance liquid chromatography coupled with a variety of spectrometric detection techniques (JunkerBuchheit and Witzenbacher 1995, Crescenzi *et al.* 1997, Di Corcia *et al.* 2000, Hogendoorn *et al.* 2000, Van Hoof *et al.* 2002, Deng *et al.* 2007). Limits of detection below 1 µg/L can be achieved by most of these techniques.

HISTORY OF THE HEALTH VALUES

The current acceptable dietary intake (ADI) for carbendazim is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.5 mg/kg bw/day from a 2-year dog study. The NOEL is based on evidence of adverse effects on the liver. The ADI incorporates a safety factor of 100, and was established in 1979.

The current ADI for thiophanate-methyl is 0.02 mg/kg bw, based on a NOEL of 2 mg/kg bw/day from a long-term rat study. The NOEL is based on degeneration and atrophy in the testes. The ADI incorporates a safety factor of 100 and was established in 1991.

The previous *Australian Drinking Water Guidelines* health value for carbendazim was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Since thiophanate-methyl is converted to carbendazim in the environment, the toxicity of carbendazim is the major consideration in relation to the health impact of thiophanate-methyl in drinking water.

Metabolism: Carbendazim is readily absorbed via the gastrointestinal tract, extensively metabolized, and excreted in the urine (86%) and faeces. The main metabolite is 5-hydroxy carbendazim (5-HBC-S). Approximately 98% of carbendazim and its metabolites are excreted within three days.

Acute effects: Carbendazim has low acute oral and dermal toxicity in rats. It is not a skin sensitiser.

Short-term effects: In short-term studies in rats, there were decreased sperm counts and testicular degeneration at 200 mg/kg bw/day. Liver toxicity was also observed at dose levels above 7.5 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in mice, rats and dogs. In rats, there was evidence of diffuse testicular atrophy at 250 mg/kg/bw/day. In dogs, there were histopathological changes in the liver, increases in cholesterol, and changes in liver enzyme levels at 15 mg/kg bw/day. In both rats and dogs, there were testicular effects at 250 and 125 mg/kg bw/day, respectively. The NOEL of 2.5 mg/kg bw/day in the dog study is the basis for the ADI.

Carcinogenicity: There was an increase in hepatocellular adenomas and carcinomas in some strains of mice, but these were considered species-specific and not relevant to humans.

Genotoxicity: Carbendazim can cause aneuploidy as a result of interference with the formation of the mitotic spindle. It is negative for genotoxicity in other *in vitro* and *in vivo* studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: In a reproduction study in rats, carbendazim produced testicular degeneration and reduced fertility at 50 mg/kg bw/d. Developmental studies conducted via gavage in rats (but not dietary studies) demonstrated skeletal malformations at 30-90 mg/kg bw/day in the absence of any maternal toxicity. In rabbits, gavage studies produced embryotoxicity at doses at and above 20 mg/kg bw/day and teratogenicity at 125 mg/kg bw/day.

Neurotoxicity: There was no evidence of delayed neurotoxicity.

Poison Schedule: Carbendazim is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Thiophanate-methyl is included in Schedule 5 or 6 depending on the concentration. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.09 mg/L for carbendazim/thiophanate-methyl was determined as follows:

$$0.09 \text{ mg/L} = \frac{2.5 \text{ mg/kg bodyweight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.5 mg/kg bw/day is a NOEL based on a long-term (2-year) dog study with carbendazim.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Crescenzi C, Di Corcia A, Guerriero E, Samperi R (1997). Development of a multiresidue method for analyzing pesticide traces in water based on solid-phase extraction and electrospray liquid chromatography mass spectrometry. *Environmental Science and Technology,* 31(2):479-488.

Deng HS, Xiang BR, Xie SF, Zhou XH (2007). Trace determination of carbendazim and thiabendazole in drinking water by liquid chromatography and using linear modulated stochastic resonance algorithm. *Arzneimittel-Forschung-Drug Research,* 57(11):717-722.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Di Corcia A, Nazzari M, Rao R, Samperi R, Sebastiani E (2000). Simultaneous determination of acidic and non-acidic pesticides in natural waters by liquid chromatography-mass spectrometry. *Journal of Chromatography A,* 878(1):87-98.

Hogendoorn EA, Westhuis K, Dijkman E, Heusinkveld EAG, Chamraskul P, Biadul P, Baumann RA, Cornelese AA, van der Linden MA (2000). Determination of carbendazim in water, soil and sediment samples by RPLC with and without column switching and various spectrometric detection modes. *International Journal of Environmental Analytical Chemistry,* 78(1):67-85.

NOTE: Important general information is contained in PART II, Chapter 6

JunkerBuchheit A, Witzenbacher M (1995). Pesticide monitoring of drinking water with the help of solid-phase extraction and high-performance liquid chromatography. In: *5th Workshop on Chemistry and Fate of Modern Pesticides Paris*, France, pp 67-74.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Van Hoof F, Van Wiele P, Acobas F, Guinamant JL, Bruchet A, Schmitz I, Bobeldijk I, Sacher F, Ventura F, Boleda R (2002). Multiresidue determination of pesticides in drinking and related waters by solid-phase extraction and liquid chromatography with ultraviolet detection: Interlaboratory study. *Journal of AOAC International,* 85(2):375-383.

NOTE: Important general information is contained in PART II, Chapter 6

Carbofuran

(endorsed 2011)

GUIDELINE

Based on human health concerns, carbofuran in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Carbofuran (CAS 1563-66-2) belongs to the carbamate class of chemicals. Other pesticides in this class include aldicarb, bendiocarb, carbaryl, methiocarb, methomyl, pirimicarb and propoxur (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, carbofuran would not be a health concern unless the concentration exceeded 0.01 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Carbofuran is a broad-spectrum insecticide and nematicide used to control worms and other pests in a variety of crops.

There are registered products containing carbofuran in Australia. These are intended for professional use and are available in concentrated solutions or as granular formulations that are applied directly to crops and soil. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to carbofuran is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of carbofuran may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of this compound in Australian drinking water could be found and, given its physicochemical properties and rapid degradation, it is unlikely to persist in waters. Carbofuran was only detected in one sample of 678 samples in a survey of Canadian municipal and private water supplies (Health and Welfare Canada 1991). It has also been detected in streams in the USA (Kimbrough and Litke 1996).

TREATMENT OF DRINKING WATER

Carbofuran has been shown to be nearly completely (99.9%) removed when water undergoes advanced oxidation with iron-catalysed ultraviolet irradiation and peroxide (Fenton reaction) (Huston and Pignatello 1999). Conventional clarification/chlorination has been demonstrated to be unreliable for the removal of carbofuran from water, although softening clarification improves removal through conventional plants substantially (CARAT 2000).

MEASUREMENT

Carbofuran in water is commonly analysed in Australian laboratories using high-performance liquid chromatography with pre-column derivitisation with orthophthalaldehyde and fluorescence detection of the derivative. The detection limit is usually around 1 µg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for carbofuran is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.33 mg/kg bw/day from a 1-year dietary study in dogs. This NOEL is based on inhibition of brain cholinesterase and histopathological effects. The ADI incorporates a safety factor of 100 and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Carbofuran is rapidly absorbed via the gastrointestinal tract and undergoes rapid metabolism. The metabolites are formed by oxidative and hydrolytic pathways and are rapidly excreted, mainly in the urine.

Acute effects: Carbofuran has high acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: A 6-week intraperitoneal study in mice reported reduced cholinesterase activity.

Long-term effects: In long-term dietary studies in mice, rats and dogs, the most sensitive effect observed was inhibition of brain cholinesterase levels. The lowest dose at which this was observed was 0.66 mg/kg bw/day in dogs. Histopathological alterations in testicular tissue, lungs, liver and thyroid were also observed at this dose in dogs.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for carbofuran.

Genotoxicity: Carbofuran is not considered genotoxic, based on *in vitro* and *in vivo* short-term tests.

Reproductive and developmental effects: Two multigeneration reproduction studies in rats and developmental toxicity studies in mice, rats and rabbits reported no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Carbofuran is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for carbofuran was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.33 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.33 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization established a health-based guideline value of 0.007 mg/L for carbofuran in 1998 (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

CARAT (Committee to Advise on Reassessment and Transition) (2000). Summary of Pesticide Removal/Transformation Efficiencies from Various Drinking Water Treatment Processes. United States Environmental Protection Agency & United States. Department of Agriculture Committee to Advise on Reassessment and Transition.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Health and Welfare Canada (1991). *Guidelines for Canadian drinking water quality.* Ottawa, Ontario.

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction. *Water Research*, 33(5):1238-1246.

Kimbrough RA, Litke DW (1996). Pesticides in streams draining agricultural and urban areas in Colorado. *Environmental Science and Technology*, 30:908–916.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva.

NOTE: Important general information is contained in PART II, Chapter 6

Carbon tetrachloride

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of carbon tetrachloride in drinking water should not exceed 0.003 mg/L.

GENERAL DESCRIPTION

Carbon tetrachloride is not produced in drinking water as a by-product of chlorination, but it may be present in chlorine used for disinfection. It has occasionally been found overseas as a contaminant in drinking water supplies at concentrations less than 0.003 mg/L.

The major use of carbon tetrachloride is in the commercial production of chlorofluorocarbons which are used as refrigerants, foam-blowing agents and solvents. It is also used in the manufacture of paint and plastics.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Preliminary data indicate that concentrations of carbon tetrachloride in major Australian reticulated supplies are significantly less than 0.001 mg/L.

TREATMENT OF DRINKING WATER

Carbon tetrachloride can be removed from drinking water by adsorption onto granular activated carbon.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of carbon tetrachloride (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and carbon tetrachloride extracted using methyl tert-butyl ether. The extract is then analysed using gas chromatography with an electron capture detector. The limit of determination is approximately 0.000004 mg/L (4 ng/L).

HEALTH CONSIDERATIONS

Carbon tetrachloride is absorbed readily from the gastrointestinal tract, the respiratory tract and the skin. It is distributed to all major organs, with highest concentrations in fatty tissues. It is metabolised in the liver to chloroform and other products, and excreted in breath, urine and faeces.

In humans, acute inhalation can result in central nervous system depression, and kidney and liver toxicity. Occupational exposure to carbon tetrachloride by inhalation has been associated with cancer of several organs but the evidence is inconclusive. No data are available on the effects of long-term ingestion of carbon tetrachloride. In animals, the effects of long-term exposure include toxicity to the liver and kidney. Liver tumours have been reported in studies with mice, rats and hamsters, but at doses higher than those that cause liver toxicity.

Carbon tetrachloride does not exhibit any evidence of mutagenic activity in tests with bacteria or cultured liver cells.

The International Agency for Research on Cancer has concluded that carbon tetrachloride is possibly carcinogenic to humans (Group 2B, inadequate evidence in humans but sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value of 0.003 mg/L for carbon tetrachloride was determined as follows:

$$0.003 \text{ mg/L} = \frac{1.2 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000} \times \frac{5}{7}$$

where:

- 1.2 mg/kg body weight per day is the no-effect level based on a 90-day gavage study using mice (Condie ***et al***. 1986).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for less than lifetime study). An additional factor of 10 for carcinogenicity was not applied as tumours occur at doses that have already resulted in liver toxicity.
- 5/7 is used to convert data based on a 5 day per week gavage study to a 7-day week equivalent.

The World Health Organization guideline value of 0.002 mg/L was based on an adult body weight of 60 kg. The difference in the guideline values is not significant.

REFERENCES

Condie LW, Laurie RD, Mills T, Robinson M, Bercz JP (1986). Effect of gavage vehicle on hepatotoxicity of carbon tetrachloride in CD-1 mice: Corn oil versus Tween-60 aqueous emulsion. *Fundamental and Applied Toxicology*, 7:199–206.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

USEPA Draft Method 551 (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Carboxin

(endorsed 2011)

GUIDELINE

Based on human health concerns, carboxin in drinking water should not exceed 0.3 mg/L.

RELATED CHEMICALS

Carboxin (CAS 5234-68-4) belongs to the carboxamide class of chemicals. Other pesticides in this class include oxycarboxin (a metabolite of carboxin) (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population to carboxin is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, carboxin would not be a health concern unless the concentration exceeded 0.3 mg/L. Excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Carboxin is a systemic fungicide for the control of smut and bunt in seeds (wheat, barley, oats and maize) prior to planting.

There are currently products registered in Australia that contain carboxin. These products are intended for professional use and are available as concentrated solutions to be applied directly or in diluted form as a seed dressing. A dust formulation is also available. The seed dressing is applied prior to sowing. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to carboxin and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of carboxin may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on carboxin in Australian waters. In the USA, modelling by the United States Environmental Protection Agency suggests maximum concentrations of 0.63 µg/L and 0.095 µg/L in surface water and groundwater, respectively (USEPA 2004).

TREATMENT OF DRINKING WATER

No specific data on the treatment of carboxin in drinking water have been identified.

MEASUREMENT

Carboxin in water can be measured by gas chromatography–mass spectrometry, with a method detection limit of 1.4 µg/L (USEPA 1995).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for carboxin is 0.08 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 8.5 mg/kg bw/day from a long-term (2-year) dietary study. The NOEL is based on liver effects in mice. The ADI incorporates a safety factor of 100 and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was also 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Carboxin is readily absorbed via the gastrointestinal tract. It is extensively metabolised, and is excreted in the urine and faeces almost completely within 96 hours. A major metabolite is oxycarboxin, which also has fungicidal activity.

Acute effects: Carboxin has low to very low acute oral and dermal toxicity. Carboxin is not a skin sensitiser.

Short-term effects: A four-week dietary study in rats reported liver effects at dose levels above 90 mg/kg bw/day. These effects were still present (although not as severe) 3 weeks after cessation of treatment.

Two 90-day dietary studies in rats reported effects on both the liver and kidney at dose levels above 10 mg/kg bw/day.

Long-term effects: Long-term dietary studies in mice reported decreased survival in females and effects on the lung at 750 mg/kg bw/day, and effects on the liver at 375 mg/kg bw/day and above. The NOEL based on changes in the liver was 8.5 mg/kg bw/day.

Long-term dietary studies have also been performed on rats and dogs. Effects reported in rats include decreased bodyweight, decreased food consumption and decreased survival (males only) at 30 mg/kg bw/day. There was no evidence of toxicity in dogs at 15 mg/kg bw/day.

Genotoxicity: Carboxin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproduction and developmental effects: A 3-generation reproduction study and a developmental study in rats did not produce any evidence of reproductive or teratogenic effects. A developmental study in rabbits reported effects only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Carboxin is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.3 mg/L for carboxin was determined as follows:

$$0.3 \text{ mg/L} = \frac{8.5 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 8.5 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies variation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1995). Method 525.2. Determination of organic compounds in drinking water by liquid-solid extraction and capillary column gas chromatography/mass spectrometry. Revision 2.0.

USEPA (United States Environmental Protection Agency) (2004) – Reregistration Eligibility Decision for Carboxin. Online at: http://www.epa.gov/pesticides/reregistration/REDs/0012red_carboxin.pdf

NOTE: Important general information is contained in PART II, Chapter 6

arfentrazone-ethyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, carfentrazone-ethyl in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Carfentrazone-ethyl (CAS 128639-02-1) belongs to the triazolinone class of chemicals. Another pesticide in this class is amicarbazone (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, carfentrazone-ethyl would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Carfentrazone-ethyl is a herbicide used alone, or in combination with other herbicides, for the control of certain broad-leaf weeds prior to establishment of a variety of crops including winter cereals, pyrethrum, fruit trees, nuts, cotton and grapevines. It is also used to control weeds in commercial and industrial settings, in public areas, and around agricultural buildings and yards. In addition, carfentrazone-ethyl is used to control aquatic weeds in rice and for desuckering grapevines.

There are registered products that contain carfentrazone-ethyl in Australia. These products are dry flowables, emulsifiable concentrates or micro-encapsulated formulations, and are intended for professional use. They are most commonly applied by broadcast methods (boom spray). Back-pack hand spray is also used, with aerial application recommended for some products for cotton desiccation only. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to carfentrazone-ethyl is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of carfentrazone-ethyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater. In addition, carfentrazone-ethyl can also be applied directly into water bays of rice crops.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of carfentrazone-ethyl in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No reports of the treatment of carfentrazone-ethyl in drinking water have been identified. However, it has been reported that carfentrazone-ethyl is rapidly converted to carfentrazone-chloropropionic acid and then gradually degraded under aerobic aquatic conditions (Elmarakby *et al*. 2001).

MEASUREMENT

Solid-phase extraction (SPE) and gas chromatography with electron-capture detection has been used for sensitive, simple, and reliable analysis of carfentrazone-ethyl residues in water. Recent research has demonstrated that the use of multiwalled carbon nanotubes as a SPE adsorbent for analysis of carfentrazone-ethyl can achieve a limit of quantitation of 0.03 μg/L (Dong *et al*. 2009).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for carfentrazone-ethyl is 0.03 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 3 mg/kg bw/day from a long-term (2-year) study in rats. The NOEL is based on red fluorescence seen in the female liver at the next highest dose of 12 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was established in 1998.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Carfentrazone-ethyl is readily absorbed via the gastrointestinal tract in mice and rats. It is extensively metabolised, the main metabolites being carfentrazone-ethyl-chloropropionic acid and 3-hydroxymethyl-carfentrazone-ethyl-chloropropionic acid. Most of the administered dose is excreted as metabolites in the urine within 24 hours.

Acute effects: Carfentrazone-ethyl has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pigs.

Short-term effects: Repeat-dose studies indicate that the primary targets for carfentrazone-ethyl toxicity are the red blood cells (RBC) and the liver. In 28-day and 90-day dietary studies in mice, rats and dogs, effects were noted on blood parameters at very high dose levels only (in excess of 500 mg/kg bw/day).

Long-term effects: An 80-week dietary study in mice reported some reduction in RBCs at the lowest dose tested (10 mg/kg bw/day), and more significant effects (reduced packed cell volume and haemoglobin, increased mean corpuscular volume at 100 mg/kg bw/day. In long-term dietary studies in rats, there was a slight increase in urinary porphyrins, together with red fluorescence in the liver, correlating to porphyrin deposits, at 12 mg/kg bw/day. The lowest NOEL was 3 mg/kg bw/day in the rat study and this is the basis for the current ADI. A 52-week oral study in dogs showed an increased level of urinary porphyrins from 150 mg/kg bw/day.

Carcinogenicity: Based on long-term exposure studies in mice, rats and dogs, there is no evidence of carcinogenicity for carfentrazone-ethyl.

Genotoxicity*:* Carfentrazone-ethyl is not considered to be genotoxic, based on *in vitro* and in vivo short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive performance or on foetal development at below maternotoxic dose levels.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Carfentrazone-ethyl is considered not to require control by scheduling due to its low toxicity and is therefore in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for carfentrazone-ethyl was determined as follows:

$$0.1 \text{ mg/L} = \frac{3 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 3 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra http://www.comlaw.gov.au/Details/F2010L02386/Download.

Dong MF, Ma YQ, Liu FM, Qian CF, Han LJ, Jiang SR (2009). Use of multiwalled carbon nanotubes as a SPE adsorbent for analysis of carfentrazone-ethyl in water. *Chromatographia,* 69(1-2):73-77.

Elmarakby SA, Supplee D, Cook R (2001). Degradation of [C-14]carfentrazone-ethyl under aerobic aquatic conditions. *Journal of Agricultural and Food Chemistry,* 49(11):5285-5293.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Chloral hydrate

(Trichloroacetaldehyde)

CAS NO 302-17-0

(endorsed 2014)

GUIDELINE

Based on health considerations, the concentration of chloral hydrate in drinking water should not exceed 0.1 mg/L.

Action to reduce chloral hydrate is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chloral hydrate.

GENERAL DESCRIPTION

Chloral hydrate may be formed as a byproduct during chlorination of water containing naturally occurring organic material. Contamination of drinking water due to industrial spills is unlikely in Australia but has occurred overseas. In the United States, chloral hydrate has been detected in a small number of supplies, with concentrations ranging from 0.00001 mg/L (10 ng/L) to 0.1 mg/L.

Chloral hydrate has been used as a sedative and hypnotic drug in humans at oral doses up to 14 mg/kg body weight. A typical adult dose as a sedative is 250 mg, three times per day (WHO 2005). However, therapeutic use is generally short-term.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Testing undertaken of drinking water in South Australia between 2000 and 2012 detected up to 0.088 mg/L chloral hydrate (Amis 2012) while a survey conducted in Victoria in 2010 detected up to 0.04 mg/L (Department of Health 2011). Another study found concentrations of chloral hydrate in Australian drinking waters ranged from 0.0002 to 0.019 mg/L (Simpson and Hayes 1998).

LIMITING FORMATION IN DRINKING WATER

The presence of chloral hydrate in drinking water can be minimised by removing naturally occurring organic matter from the source water, by reducing the amount of chlorine added, or by the use of alternative disinfectants.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of chloral hydrate (USEPA Method 551.1 1995). Chloral hydrate is extracted using methyl tert-butyl ether and analysed using gas chromatography with an electron capture detector. The limit of determination is approximately 0.000005 mg/L (5 ng/L). Standard Method 5710 D of the 21st edition of the ***Standard Methods for the Examination of Water and Wastewater*** can be used to analyse chloral hydrate as well as trihalomethanes (APHA ***et al***. 2012).

HEALTH CONSIDERATIONS

Chloral hydrate is known to be rapidly absorbed in humans and quickly oxidised to trichloroacetic acid or reduced to trichloroethanol.

In its wide use as a sedative or hypnotic drug in humans, concentrated solutions have proved quite

irritating to the gastrointestinal tract, and have caused nausea and vomiting. Side effects of the drug have included central nervous system depression, minor sensitivity reactions, and central nervous system excitement. Chronic use may result in development of tolerance, physical dependence and addiction. Addicts have been reported to take as much as 12 grams per day.

There have been a number of animal toxicity studies using rats and mice varying in duration from a few days to 2 years. In a 90-day drinking water study using mice, some enlargement of the liver was reported at doses from 16 mg/kg body weight per day. Other studies have reported that higher doses cause some liver toxicity.

A number of chronic studies have provided equivocal evidence for carcinogenicity (WHO 2005; Health Canada 2008). In a 2-year drinking water study in mice, the incidence of proliferative lesions in the liver was increased at concentrations of 120 mg/L (13.5 mg/kg/d, LOAEL) and above.

Chloral hydrate was mutagenic in tests with some strains of bacteria but did not bind to mouse liver DNA. It increased the frequency of chromosome aberrations in cultured cells and of bone marrow micronuclei in mice.

DERIVATION OF GUIDELINE

The guideline value for chloral hydrate in drinking water was determined as follows:

$$0.1 \text{ mg/L} = \frac{13.5 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.8}{2 \text{ L/day} \times 3000}$$

where:

- 13.5 mg/kg body weight per day is the lowest effect level based on a 2-year drinking water study using mice where the incidence of liver proliferative lesions was increased at the lowest dose (George *et al*. 1982).
- 70 kg is the average weight of an adult.
- 0.8 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 3000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variation, 10 for intraspecies variation, 10 for the use of a lowest effect level instead of a no-effect level, and 3 for limitations in the data relating to carcinogenicity).

The World Health Organization has no formal guideline value but notes a value of 0.1 mg/L could be calculated (WHO 2011).

An allocation factor of 80% is used as, except for therapeutic use, exposure is predominantly from chlorinated drinking water. This is the same approach taken by WHO (2011) and Health Canada (2008).

REFERENCES

Amis A (2012). SA Water Drinking Water Quality January 2000-July 2012. Friends of the Earth, Melbourne.

APHA 5710-D Formation of other Disinfection By-Products (DBPs) (2012). Standard methods for the examination of water and wastewater. 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC. p. 5.70.

NOTE: Important general information is contained in PART II, Chapter 6

Department of Health (2011). Section 22 notifications under the ***Safe Drinking Water Act 2003*** for disinfection byproducts not regulated under schedule 2 of the ***Safe Drinking Water Regulations 2005***. Drinking Water Regulation Guidance Note No.13, 2011. Victorian Government.

George, M., Moore, T., Kilburn, S., Olson, G.R. and DeAngelo, A.B. (2000). Carcinogenicity of chloral hydrate administered in drinking water to the male F344/N rat and male B6C3F1 mouse. ***Toxicologic Pathology***, 28:610–618.

Health Canada (2008). Guidance on Chloral Hydrate in Drinking Water http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/chloral_hydrate-chloral/index-eng.php#b2_4 (accessed 15 July 2013)

Simpson KL and Hayes KP (1998). Drinking water disinfection by-products: an Australian perspective. ***Water Research***, 32 (5): 1522-1528.

U.S. EPA (United States Environmental Protection Agency) (1995). Methods for the determination of organic compounds in drinking water — Supplement III. U.S. EPA, Washington, DC (EPA-600/R-95/131).

Water Corporation of Western Australia (2013). Routine testing data (Richard Walker, personal communication)

WHO (2005). Chloral Hydrate in Drinking-water Background document for development of WHO ***Guidelines for Drinking-water Quality***. World Health Organisation, Geneva.

World Health Organization (2011). Guidelines for drinking-water quality, fourth edition.

Chlorantraniliprole

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlorantraniliprole in drinking water should not exceed 6 mg/L.

RELATED CHEMICALS

Chlorantraniliprole (CAS 500008-45-7) is in the anthranilic diamide class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chlorantraniliprole would not be a health concern unless the concentration exceeded 6 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlorantraniliprole is an insecticide used to control pests on turf, cotton and a variety of fruits and vegetables. It is also used on residential lawns.

There are registered products containing chlorantraniliprole in Australia. These products are for professional and home garden use and are applied using ground boom sprayers, hand-held sprayers or aerial spraying. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to chlorantraniliprole are the use of the home garden products, and residues in food. Residue levels in crops grown according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of sources waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No sufficient monitoring data in drinking water are available. Modelling incorporating data on environmental fate and physico-chemical characteristics has been used to estimate concentrations of chlorantraniliprole. Environmental concentrations of chlorantraniliprole from long-term exposure are estimated to be 3.65 µg/L for surface water and 1.06 µg/L for groundwater in the USA (USEPA 2008a) and 63 µg/L for groundwater in Canada (Health Canada 2008). There are no published reports on chlorantraniliprole occurrence in Australian drinking water supplies.

TREATMENT OF DRINKING WATER

There are no published reports on methods of removal of chlorantraniliprole from drinking water. However, granular activated carbon, reverse osmosis and advanced oxidation process would be probably effective. Photodegradation and alkaline-catalysed hydrolysis may also reduce chlorantraniliprole concentrations in water (USEPA 2008a)

MEASUREMENT

High performance liquid chromatography–ultraviolet detection, gas chromatography/electron capture detection, and liquid chromatography with tandem mass spectrometry (LC-MS-MS) have all been reported for the determination of chlorantraniliprole in water matrices (USEPA 2008b). LC-MS-MS is the most commonly used method. The LC-MS-MS DuPont-11374 method can achieve a limit of quantitation of 0.01 mg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for chlorantraniliprole is 1.58 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 158 mg/kg bw/day in an 18-month dietary study in mice. The NOEL is based on the appearance of eosinophilic foci accompanied by hepatocellular hypertrophy and increased liver weight. The ADI incorporates a safety factor of 100 and it was established in 2008.

An *Australian Drinking Water Guidelines* health value has not been previously set for chlorantraniliprole.

HEALTH CONSIDERATIONS

Metabolism: Chlorantraniliprole is rapidly absorbed (5-12 hours) from the gastrointestinal tract and is widely distributed throughout the body. Metabolism is extensive and there is a low potential for accumulation. Excretion is substantially complete by 48-72 hours, mainly via bile and faeces.

Acute effects: Chlorantraniliprole has low acute oral and dermal toxicity. It is a slight eye irritant but is not a skin irritant or a skin sensitiser.

Short-term and long-term effects: Short-term and long-term dietary studies in mice, rats and dogs produced adaptive liver changes including increases in liver weights (at ≥658 mg/kg bw/day in mice; at ≥128 mg/kg bw/day in rats; and at ≥1163 mg/kg bw/day in dogs) and cytochrome P450 levels (at ≥658 mg/kg bw/day in female mice) and microvesiculation of the adrenal cortex (at ≥7.71 mg/kg bw/day in rats). Additional studies demonstrated that this microvesiculation did not adversely affect adrenal gland function. Long-term studies also revealed eosinophilic foci accompanied by hepatocellular hypertrophy at high dose levels. The NOEL was 158 mg/kg bw/day, and this NOEL is the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for chlorantraniliprole.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Chlorantraniliprole is not considered to be genotoxic, based on short-term *in vitro* and *in vivo* studies.

Reproductive and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Neurotoxicity, immunotoxicity: In medium-term studies in mice and rats, there was no evidence of neurotoxicity. In 28-day studies in mice and rats, there was no evidence of effects on the immune system.

Poison Schedule: Chlorantraniliprole is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 6 mg/L for chlorantraniliprole was determined as follows:

$$6 \text{ mg/L} = \frac{158 \text{ mg/kg bodyweight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 158 mg/kg bw/day is a NOEL based on a long-term (18-month) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the average maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada Pest Management Regulatory Agency (2008). Evaluation Report Chlorantraniliprole ER2008-03. [www.pmra-arla.gc.ca/english/pdf/erc/er2008-03-eng.pdf]

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2008a). Chlorantraniliprole Pesticide Fact Sheet. Office of Prevention, Pesticides and Toxic Substances, http://www.epa.gov/opprd001/factsheets/chloran.html

USEPA (United States Environmental Protection Agency) (2008b). Chlorantraniliprole: proposed time-limited pesticide tolerance. Federal Register: October 1, 73(191):57040-57046.

NOTE: Important general information is contained in PART II, Chapter 6

Chlordane

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlordane in drinking water should not exceed 0.002 mg/L.

RELATED CHEMICALS

Chlordane (CAS 57-74-9) belongs to the cyclodiene organochlorine class of chemicals. A currently used pesticide in this class is endosulfan. Chlordane is also classified as a persistent organic pollutant (POP). Other cyclodiene organochlorines that were previously used as pesticides include aldrin, dieldrin and heptachlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present as a contaminant in drinking water, chlordane would not be a health concern unless the concentration exceeded 0.002 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlordane was used previously as an insecticide for the control of termites and soil insects.

There are no registered products that contain chlordane in Australia, but de-registered compounds may still be detected in water.

Exposure sources: The general public may be exposed to low levels of chlordane and its metabolites through residues in food and/or contaminated source waters from previous insecticidal use of chlordane.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No specific reports of chlordane in Australian drinking waters have been identified. However, chlordane is a relatively commonly identified water contaminant in many parts of the world (Yamashita ***et al***. 2000, Fatoki and Awofolu 2004, Singh ***et al.*** 2007, Kumari et al. 2008, Mmualefe ***et al***. 2009).

TREATMENT OF DRINKING WATER

No specific data on the treatment of chlordane in drinking water have been identified. It is expected that treatment by activated carbon should be effective under optimal conditions (WHO 2004).

MEASUREMENT

Chlordane is commonly measured in drinking waters by gas chromatography–electron capture detection, with a limit of detection of 0.014 µg/L (WHO 2004).

HISTORY OF THE HEALTH VALUES

The current tolerable daily intake (TDI) for chlordane is 0.0005 mg per kg bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.045 mg/kg bw/day from a 130-week dietary rat study. The NOEL is based on effects in the liver. The TDI incorporates a safety factor of 100, and was established in 2003.

When chlordane was previously used, the acceptable daily intake (ADI) was 0.0005 mg/kg bw/day, based on a NOEL of 0.045 mg/kg bw/day from the same long-term dietary study.

The previous ADWG health value was 0.001 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Chlordane is readily absorbed via the gastrointestinal tract and is extensively metabolised to oxychlordane and heptachlor epoxide. It is mainly excreted in the faeces.

Acute effects: Chlordane has low/moderate acute oral toxicity and moderate dermal toxicity. It is not a skin sensitiser.

Short-term effects: A 30-day dietary study in mice reported histopathological changes in the liver at 1.4 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs showed the liver to be the target organ of toxicity. Effects on the liver were observed at 0.13 mg/kg bw/day in mice and 0.27 mg/kg bw/day in dogs. In a 130-week study in rats, increased absolute and relative liver weights, liver enlargement, hepatocellular swelling and necrosis were seen at dose levels above 0.045 mg/kg bw/day. The NOEL of 0.045 mg/kg bw/day is the basis for the current TDI.

Carcinogenicity: Carcinogenicity studies were conducted in mice and rats. There was evidence of liver tumours in mice at dose levels of 0.65 mg/kg bw/day and above. There was no evidence of carcinogenicity in rats.

Genotoxicity: Chlordane is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Reproduction studies in rats and mice reported reduced viability of offspring during weaning at 3 mg/kg bw/day and 7.5 mg/kg bw/day, respectively. Developmental studies in rabbits showed no effects on foetal development at doses up to 50 mg/kg bw/day.

Poisons Schedule: Chlordane is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.002 mg/L for chlordane was determined as follows:

$$0.002 \text{ mg/L} = \frac{0.045 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.045 mg/kg bw/day is the NOEL based on a long-term (130-week) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the TDI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization established a health-based guideline value of 0.0002 mg/L for chlordane in 2003 (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Fatoki OS, Awofolu OR (2004). Levels of organochlorine pesticide residues in marine-, surface-, ground- and drinking waters from the Eastern Cape Province of South Africa. *Journal of Environmental Science and Health Part B – Pesticides, Food Contaminants and Agricultural Wastes,* 39(1):101-114.

Kumari B, Madan VK, Kathpal TS (2008). Status of insecticide contamination of soil and water in Haryana, India. *Environmental Monitoring and Assessment,* 136(1-3):239-244.

Mmualefe LC, Torto N, Huntsman-Mapila P, Mbongwe B (2009). Headspace solid phase microextraction in the determination of pesticides in water samples from the Okavango Delta with gas chromatography-electron capture detection and time-of-flight mass spectrometry. *Microchemical Journal,* 91(2):239-244.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Singh KP, Malik A, Sinha S (2007). Persistent organochlorine pesticide residues in soil and surface water of northern Indo-Gangetic alluvial plains. *Environmental Monitoring and Assessment,* 125(1-3):147-155.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

Yamashita N, Urushigawa Y, Masunaga S, Walash MI, Miyazaki A (2000). Organochlorine pesticides in water, sediment and fish from the Nile River and Manzala Lake in Egypt. *International Journal of Environmental Analytical Chemistry,* 77(4):289-303.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorfenvinphos

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlorfenvinphos in drinking water should not exceed 0.002 mg/L.

RELATED CHEMICALS

Chlorfenvinphos (CAS 470-90-2) belongs to the organophosphate class of chemicals. There are many other pesticides in this class including fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chlorfenvinphos would not be a health concern unless the concentration exceeded 0.002 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on both short-term and long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlorfenvinphos is an insecticide that is used primarily as a parasiticide treatment for cattle, sheep, horses and to a lesser extent, deer, goats and working dogs. Agricultural use of chlorfenvinphos was cancelled and veterinary use restricted following a review in 2000 (AVPMA 2000).

There are registered products containing chlorfenvinphos in Australia. These products are intended for veterinarian or farm worker use and are applied as a topical suspension or spray, or as a dip for cattle. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to chlorfenvinphos is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The veterinary use of chlorfenvinphos provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on the concentrations of chlorfenvinphos in Australian drinking water.

TREATMENT OF DRINKING WATER

Relatively high removal rates of chlorfenvinphos have been achieved using conventional flocculation, adsorption onto activated carbon and ozonation (Ormad *et al.* 2008).

MEASUREMENT

Several methods have been reported for the analysis of chlorfenvinphos in water including solid phase extraction with gas chromatography–tandem mass spectrometry, with a limit of detection (LOD) of 4 ng/L (Ruiz-Gill *et al.* 2008), solid phase extraction with liquid chromatography–tandem mass spectrometry (LOD 10 ng/L, Greulich *et al.* 2008) and stir-bar sorptive extraction with gas chromatography–mass spectrometry (LOD 4.3 ng/L, Ochiai *et al.* 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for chlorfenvinphos is 0.0005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.05 mg/kg bw/day from a 4-week dietary study in rats, a 2-year dietary study in rats and a 2-generation reproduction study in rats. This NOEL is based on plasma and/or brain cholinesterase inhibition. The ADI incorporates a safety factor of 100 and was established in 1998.

The previous Australian ADI for chlorfenvinphos was 0.002 mg/kg bw, based on a NOEL of 0.15 mg/kg bw/day for plasma cholinesterase inhibition seen in a 2-year rat dietary study and using a 100-fold safety factor.

The acute reference dose (ARfD) of 0.02 mg/kg bw/day for chlorfenvinphos was established in 2000, based on a NOEL of 1.9 mg/kg bw/day from a 14-day mouse study for inhibition of red blood cell cholinesterase activity. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Chlorfenvinphos is absorbed readily and extensively via the gastrointestinal tract. The rate of metabolism of chlorfenvinphos is species-specific, with dogs much higher than rats. In a human volunteer study, there was rapid excretion in the urine (94% in 26 hours). The main metabolite was desethyl chlorfenvinphos.

Acute effects: Chlorfenvinphos has low to high acute toxicity depending on the species. In rats, the acute oral and dermal toxicity was high. It was not a skin sensitiser. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes. These have been observed in humans as well as animals.

Short-term and long-term effects: Short-term and long-term dietary studies with chlorfenvinphos reported symptoms indicative of nervous system toxicity caused by depression of cholinesterase activity. Short-term studies in mice reported brain cholinesterase inhibition at 0.2 mg/kg bw/day and plasma cholinesterase inhibition at 1.9 mg/kg bw/day. In a 2-year mouse study, plasma cholinesterase levels were depressed at 3.9 mg/kg bw/day. Both plasma and brain cholinesterase had similar level of sensitivity in the rat. Four-week and 2-year studies in rats reported plasma cholinesterase inhibition at dose levels of 0.15 mg/kg bw/day and above. The NOEL of 0.05 mg/kg bw/day from the rat studies is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for chlorfenvinphos.

Genotoxicity: Chlorfenvinphos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: Multigeneration reproduction studies in rats reported decreased fertility and decreased pup survival, but only at dose levels causing cholinesterase inhibition. In developmental toxicity studies in rats and rabbits, foetal development was impaired only at dose levels caused significant cholinesterase inhibition.

Poisons Schedule: Chlorfenvinphos is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.002 mg/L for chlorfenvinphos was determined as follows:

$$0.002 \text{ mg/L} = \frac{0.05 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.05 mg/kg bw/day is the NOEL based on a short-term (4-week) dietary study in rats, a long-term (2-year) dietary study in rats, and a 2-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

APVMA (Australian Pesticides and Veterinary Medicines Association) (2000) The NRA Review of Chlorfenvinphos – Interim Report Volume 1 http://www.apvma.gov.au/products/review/current/chlorfenvinphos.php

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Greulich K, Alder L (2008). Fast multiresidue screening of 300 pesticides in water for human consumption by LC-MS/MS. *Analytical and Bioanalytical Chemistry*, 391:183-197.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ochiai N, Sasmoto K, Kanda H, Pfannkoch E (2008). Sequential stir bar sorptive extraction for uniform enrichment of trace amounts of organic pollutants in water samples. *Journal of Chromatography A*, 1200:72-79.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71, 97–106.

NOTE: Important general information is contained in PART II, Chapter 6

Ruiz-Gill L, Romero-Gonzalez R, Frenich AG, Vidal JLM (2008). Determination of pesticides in water samples by solid phase extraction and gas chromatography tandem mass spectrometry. *Journal of Separation Science*, 31:151-161.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Chloride

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, the chloride concentration in drinking water should not exceed 250 mg/L.

No health-based guideline value is proposed for chloride.

GENERAL DESCRIPTION

Chloride is present in natural waters from the dissolution of salt deposits, and contamination from effluent disposal.

Sodium chloride is widely used in the production of industrial chemicals such as caustic soda, chlorine, and sodium chlorite and hypochlorite. Potassium chloride is used in the production of fertilisers.

The taste threshold of chloride in water is dependent on the associated cation but is in the range 200–300 mg/L. The chloride content of water can affect corrosion of pipes and fittings. It can also affect the solubility of metal ions.

In surface water, the concentration of chloride is usually less than 100 mg/L and frequently below 10 mg/L. Groundwater can have higher concentrations, particularly if there is salt water intrusion.

Food is the major source of chloride intake. All plants and animals contain chloride. The addition of salt during processing or cooking can markedly increase the chloride content.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies chloride concentrations range up to 350 mg/L. Typical values depend to a large extent on local conditions but concentrations of 150 mg/L are not uncommon in some areas.

TREATMENT OF DRINKING WATER

Chloride cannot be removed from drinking water by conventional water treatment processes. It can be removed by distillation or reverse osmosis but these are expensive to operate.

MEASUREMENT

The chloride concentration in drinking water can be determined with titrimetric techniques using silver nitrate or mercuric nitrate and colorimetric or potentiometric end-point detection (APHA Method 4500-Cl^- Parts B or C 1992). The limit of determination is approximately 1 mg/L. Ion chromatography can also be used (APHA Method 4500-Cl^- Part F 1992), with a limit of determination of 0.1 mg/L.

HEALTH CONSIDERATIONS

Chloride is essential for humans and animals. It contributes to the osmotic activity of body fluids. A normal 70 kg human body contains approximately 80 g of chloride.

Chloride is absorbed almost completely by the gastrointestinal tract. Healthy individuals can tolerate the intake of large quantities of chloride provided there is a corresponding intake of fresh water.

Little is known about the prolonged intake of large amounts of chloride by humans. Large salt intake has been reported to increase blood pressure but this is attributed to the sodium content rather than chloride. Similar results have been reported in studies with animals, although long-term data are not available.

No data are available on carcinogenic or genotoxic effects for chloride.

DERIVATION OF GUIDELINE

The guideline value is based on the taste threshold in drinking water of approximately 250 mg/L.

There are no data to suggest that chloride causes health problems; hence, no guideline value based on health considerations is warranted.

REFERENCES

APHA Method 4500-Cl^- Part B (1992). Chloride: Argentometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 4500-Cl^- Part C (1992). Chloride: Mercuric nitrate method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 4500-Cl^- Part F (1992). Chloride: ion chromatography method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorinated furanones
3-chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX)

(endorsed 1996)

GUIDELINE

Data are inadequate to set a guideline value for MX in drinking water.

GENERAL DESCRIPTION

The organic compound known as MX can be formed by the reaction between chlorine and naturally occurring organic matter in water. It has been identified in chlorinated humic acid solutions, after the chlorination of pulp mill effluent, and chlorinated drinking water. No other sources of MX are known.

The stability of MX is dependent on pH. Below pH 7 it is relatively stable but above pH 7 it rapidly breaks down.

Studies in the United States, the United Kingdom and Finland have found extremely low MX concentrations in drinking water. Concentrations range up to 0.000067 mg/L (67 ng/L).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Preliminary investigations indicate that concentrations of MX in Australian drinking water are likely to be similar to those found overseas.

LIMITING FORMATION IN DRINKING WATER

The presence of MX in drinking water can be minimised by removing naturally occurring organic matter from the source water, by reducing the amount of chlorine added, by the use of alternative disinfectants, or by ensuring that the pH is kept above 7.

MEASUREMENT

MX is extremely difficult to detect because of the very low concentrations and the masking effects of other substances. Analysis is by extraction on XAD resin, methylation of the concentrate, and detection on a gas chromatography/mass spectrometer system employing selected ion monitoring techniques. The procedure is not suitable for routine analysis.

HEALTH CONSIDERATIONS

There are no data on the health effects of MX in humans, nor are there any long-term or lifetime toxicity data for animals.

Studies have shown that MX is an extremely potent mutagen when applied to some strains of bacteria, and about a third of the mutagenicity of chlorinated drinking water has been attributed to this compound. Genotoxic activity has also been observed *in vitro* using cultured mammalian cells, although *in vivo* experiments showed no evidence of genotoxic activity. No carcinogenicity data are available for MX.

Chlorine

CAS NO 7782-50-5

(endorsed 2014)

GUIDELINE

Based on health considerations, the guideline value for total chlorine in drinking water is 5 mg/L.

GENERAL DESCRIPTION

Chlorine dissociates in water to form free chlorine, which consists of aqueous molecular chlorine, hypochlorous acid and hypochlorite ion. Chlorine and hypochlorites are toxic to microorganisms and are used extensively as disinfectants for drinking water supplies. Chlorine is also used to disinfect sewage and wastewater, swimming pool water, in-plant supplies, and industrial cooling water.

Chlorine has an odour threshold in drinking water of about 0.6 mg/L, but some people are particularly sensitive and can detect amounts as low as 0.2 mg/L. Water authorities may need to exceed the odour threshold value of 0.6 mg/L in order to maintain an effective disinfectant residual.

In the food industry, chlorine and hypochlorites are used for general sanitation and for odour control. Large amounts of chlorine are used in the production of industrial and domestic disinfectants and bleaches, and it is used in the synthesis of a large range of chemical compounds.

Free chlorine reacts with ammonia and certain nitrogen compounds to form combined chlorine. With ammonia, chlorine forms chloramines (monochloramine, dichloramine and nitrogen trichloride or trichloramine) (APHA 2012). Chloramines are used for disinfection but are weaker oxidising agents than free chlorine.

Free chlorine and combined chlorine may be present simultaneously (APHA 2012). The term total chlorine refers to the sum of free chlorine and combined chlorine present in a sample.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

When used as a disinfectant, the free chlorine residual in major Australian reticulated supplies ranges from 0.1 mg/L to 4 mg/L, with typical concentrations in the reticulation of about 0.2 to 0.5 mg/L.

TREATMENT OF DRINKING WATER

Chlorine can be removed from drinking water by aeration, by exposure to sunlight, or by the addition of reducing agents such as sodium bisulfite.

MEASUREMENT

The concentration of chlorine in drinking water can be determined by several methods including the amperometric titration method (APHA Method 4500-Cl Part D 2012), DPD ferrous titrimetric method (APHA Method 4500-Cl Part F 2012) and the DPD colorimetric method (APHA Method 4500-Cl Part G 2012). The methods are subject to interferences and vary in complexity, sensitivity, precision and accuracy. Water utilities should consider Standard Methods when selecting a method (APHA 2012). The chlorine concentration should be determined immediately after sampling as chlorine is not stable in water.

HEALTH CONSIDERATIONS

Chlorine and hypochlorites are strong oxidising agents that readily react with organic molecules to produce a wide variety of chlorinated compounds. This reactivity makes it difficult to separate the effects of chlorine from those of its metabolites. In animal studies using a naturally occurring non-radioactive chlorine isotope, chlorine was rapidly absorbed by the gastrointestinal tract, and highest concentrations of the isotope were found in blood plasma.

It is assumed that the toxicities of aqueous solutions containing chlorine, hypochlorous acid or hypochlorite are similar since they are in dynamic equilibrium. Chlorine concentrations therefore refer to free available chlorine.

Very few toxic effects have been associated with drinking water containing high chlorine concentrations. In one report, 150 people drank water with 50 mg/L during a period of mains disinfection, with no adverse effects. Several instances have been reported where military personnel drank water with chlorine concentrations up to 32 mg/L for several months with no ill effects. Mouth irritation and momentary constriction of the throat were observed when the chlorine concentration exceeded 90 mg/L. Most people would refuse to drink water with a chlorine concentration over 25 mg/L (Muegge 1956).

A number of studies have suggested an association between water chlorination and various cancers or adverse reproductive outcomes. However, results of analytical epidemiological studies are insufficient to support a causal relationship for any of the observed associations (IPCS 2004). (See Section 6.3.2 for a discussion of disinfection by-products, and Section V - Fact Sheets on specific disinfection by-products.)

Long-term animal toxicity studies have shown no specific effects from the ingestion of chlorine. Chlorine, hypochlorous acid and hypochlorite did not act as carcinogens or tumour initiators.

Assessment of the mutagenicity of chlorine is complicated by the reactivity of chlorine. Hypochlorite was found to be mutagenic in tests with one strain of bacteria but not with another. Chromosome aberrations were reported in tests with mammalian cells.

The International Agency for Research on Cancer has concluded that hypochlorites are not classifiable as to their carcinogenicity in humans (Group 3, no human data and inadequate evidence in animals) (IARC 1991).

DERIVATION OF GUIDELINE

The guideline value for chlorine in drinking water was determined as follows:

$$5 \text{ mg/L} = \frac{15 \text{ mg/kg body weight per day} \times 70 \text{ kg}}{2 \text{ L/day} \times 100}$$

where

- 15 mg/kg body weight per day is the no-effect level from a 2-year drinking water study using rodents (NTP 1992).
- 70 kg is the average weight of an adult.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations).

It is assumed that all chlorine intake is from drinking water.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 4500-Cl Part A (2012). Chlorine (residual): Introduction. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

APHA Method 4500-Cl Part D (2012). Chlorine (residual): Amperometric titration method. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

APHA Method 4500-Cl Part F (2012). Chlorine (residual): DPD Ferrous Titrimetric method. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

APHA Method 4500-Cl Part G (2012). Chlorine (residual): DPD colorimetric method. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

IARC (International Agency for Research on Cancer) (1991). ***IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: chlorinated drinking water, chlorination by-products, some other halogenated compounds, cobalt and cobalt compounds***. World Health Organization, International Agency for Research on Cancer, p 52.

IPCS (International Program on Chemical Safety) (2004). ***Environmental Health Criteria 216 Disinfectants and disinfectant by-products***. World Health Organization, International Program on Chemical Safety
(http://whqlibdoc.who.int/ehc/WHO_EHC_216.pdf accessed 30 October 2008).

Muegge OJ (1956). Physiological effects of heavily chlorinated drinking water. ***Journal of the American Water Works Association***, 48:1507-9.

NTP (National Toxicology Program) (1992). ***Toxicology and carcinogenesis studies of chlorinated water and chloraminated water in F344/N and B6C3F1 mice (drinking water studies)***. National Toxicology Program, Technical Report No. 392, Publication No. 92-2847. United States Department of Health and Human Services, National Institute of Health.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorine dioxide

Chlorite

Chlorate

(endorsed 2011)

GUIDELINE

Chlorine dioxide: Based on aesthetic considerations, the concentration in drinking water should not exceed 0.4 mg/L.

Chlorite: Based on health considerations, the concentration in drinking water should not exceed 0.8 mg/L.

Chlorate: Data are insufficient to set a guideline value in drinking water.

Action to reduce chlorite is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than chlorite.

GENERAL DESCRIPTION

Chlorine dioxide is used as a disinfectant for drinking water supplies. When added to water, it dissociates into chlorite and, to a lesser extent, chlorate. It is usually generated on site due to handling and transportation difficulties.

Chlorine dioxide is used commercially as a bleaching agent in paper production, paper pulp, and cleaning and tanning of leather. Chlorite is used in the production of paper, textiles and straw products, and in the manufacture of waxes, shellacs and varnishes. Chlorates have been used as herbicides and defoliants, and in the manufacture of dyes, matches, and explosives. Chlorate is also generated by the dissociation of hypochlorite solutions, which are used for disinfection of drinking water. Use of such solutions has become more common in Australia in recent years as use of chlorine gas has declined due to occupational health and safety considerations. Chlorate levels can be minimised by restricting storage times for hypochlorite solution (7 days maximum storage is recommended), and storing the solution under cool dark conditions.

The taste and odour threshold for chlorine dioxide in water is 0.4 mg/L. No data are available on taste and odour thresholds for chlorite and chlorate.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Chlorine dioxide (chlorite) is rarely used as a disinfectant in Australian reticulated supplies. When used, the chlorite residual is generally maintained between 0.2 mg/L and 0.4 mg/L. It is particularly effective in the control of manganese-reducing bacteria. Few data are available on chlorate levels in Australian water supplies.

TREATMENT OF DRINKING WATER

Chlorine dioxide can be removed from drinking water by the addition of reducing agents such as sodium bisulfite (although some studies indicate that the chlorate concentration increases as a result), by exposure to sunlight, or by the use of granular activated carbon.

NOTE: Important general information is contained in PART II, Chapter 6

MEASUREMENT

Methods are available for the determination of chlorine dioxide, chlorite and chlorate and total available chlorine (APHA *et al.* 2005 a,b).

HEALTH CONSIDERATIONS

Chlorine dioxide, chlorite, and chlorate are all absorbed rapidly by the gastrointestinal tract into blood plasma and distributed to the major organs. All compounds appear to be rapidly metabolised.

Chlorine dioxide has been shown to impair neurobehavioural and neurological development in rats exposed before birth. Experimental studies with rats and monkeys exposed to chlorine dioxide in drinking water have shown some evidence of thyroid toxicity; however, because of the studies' limitations, it is difficult to draw firm conclusions (WHO 2005)

The primary concern with chlorite and chlorate is oxidative stress resulting in changes in red blood cells. This end point is seen in laboratory animals and, by analogy with chlorate, in humans exposed to high doses in poisoning incidents (WHO 2005).

In a study with human volunteers, no adverse effects were observed after drinking water with either chlorine dioxide or chlorite concentrations up to 5 mg/L for periods of 12 weeks (Lubbers *et al.* 1981).

The International Agency for Research on Cancer has concluded that chlorite is not classifiable as to its carcinogenicity in humans (Group 3, no human data and inadequate evidence in animals) (IARC 1991).

DERIVATION OF GUIDELINES

The guideline values were determined as follows:

i) Chlorine dioxide:

A health based guideline value has not been established for chlorine dioxide because of its rapid hydrolysis to chlorite and chlorate. The guideline for chlorite is adequately protective for potential toxicity from chlorine dioxide (the no-observed-adverse-effect level [NOAEL] of 2.9 mg/kg bw/day used to derive the tolerable daily intake for chlorite is similar to the lowest NOAELs observed for effects of chlorine dioxide on neurobehavioral and neurological development and on thyroid hormone levels). The taste and odour threshold for chlorine dioxide is 0.4 mg/L.

ii) Chlorite:

$$0.8 \text{ mg/L} = \frac{2.9 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.8}{2 \text{ L/day} \times 100}$$

where

- 2.9 mg/kg body weight per day is the no-effect level from a two-generation study using rats (CMA 1997, TERA 1998).
- 0.8 is the proportion of total daily intake attributable to the consumption of water, based on the occasional use of chlorite in the food industry.
- 100 is the safety factor applied in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations).

The World Health Organization (WHO) guideline value of 0.7 mg/L was determined using an adult body weight of 60 kg (WHO 2005). The difference is not significant.

NOTE: Important general information is contained in PART II, Chapter 6

iii) Chlorate:

Data are currently considered insufficient to set a guideline value for chlorate in Australian drinking water supplies. A provisional guideline value for chlorate of 0.7 mg/L was published by WHO in 2004 based on limited data from human volunteer studies and a short-term study in rats. Data from a long-term study in rats was subsequently published (NTP 2005) and has been used to derive a new TDI value (JEFCA 2007). Given the importance of maintaining adequate disinfection of water supplies and limited options for reducing chlorate levels in supplies treated with hypochlorite, further information on the occurrence and sources of chlorate in Australian waters is needed before a guideline value can be developed.

REFERENCES

APHA, AWWA, WEF (American Public Health Association, American Water Works Association, Water Environment Federation) (2005a). Method 4500, chlorine. In: *Standard Methods for the examination of water and wastewater, 21st Edition*. Washington, DC, American Public Health Association, American Water Works Association, Water Environment Federation, pp 4.56-4.69.

APHA, AWWA, WEF (American Public Health Association, American Water Works Association, Water Environment Federation) (2005b) Method 4500, chlorine dioxide. In: *Standard Methods for the examination of water and wastewater, 21st Edition*. Washington, DC, American Public Health Association, American Water Works Association, Water Environment Federation, pp 4.77-4.81.

CMA (Chemical Manufacturers Association) (1997). *Sodium Chlorite: drinking water rat two-generation reproductive toxicity study.* Washington, DC (Quintiles Report CMA/17/96).

IARC (International Agency for Research on Cancer) (1991). *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: chlorinated drinking water, chlorination by-products, some other halogenated compounds, cobalt and cobalt compounds.* World Health Organization, International Agency for Research on Cancer, p 52.

JEFCA (Joint FAO/ WHO Expert Committee on Food Additives) (2007). Evaluation of certain food additives and contaminants. WHO Technical Report Series 947, WHO.

Lubbers JR, Chauhan S, Bianchine JR (1981). Controlled clinical evaluations of chlorine dioxide, chlorite and chlorate in man. *Fundamentals and Applied Toxicology*, 1:334-338.

NTP (National Toxicology Program) (2005). *Technical Report on the Toxicology and Carcinogenesis Studies of Sodium Chlorate in F344/N Rats and B6C3F1 Mice*. National Institute of Health.

TERA (Toxicology Excellence for Risk Assessment) (1998). *Health Risk Assessment / Characterisation of the Drinking Water Disinfection By-products Chlorine Dioxide and Chlorite*. Cincinnati OH,

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

World Health Organization (2005). Chlorite and Chlorate in Drinking-water. Background document for development of WHO guidelines for drinking-water Quality.

NOTE: Important general information is contained in PART II, Chapter 6

Chloroacetic acids
chloroacetic acid
dichloroacetic acid (DCA)
trichloroacetic acid (TCA)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentrations of chloroacetic acids in drinking water should not exceed the following values:

chloroacetic acid	***0.15 mg/L***
dichloroacetic acid	***0.1 mg/L***
trichloroacetic acid	***0.1 mg/L***

GENERAL DESCRIPTION

Chloroacetic acids are produced in drinking water as by-products of the reaction between chlorine and naturally occurring humic and fulvic acids. Concentrations reported overseas range up to 0.16 mg/L, and are typically about half the chloroform concentration.

The chloroacetic acids are used commercially as reagents or intermediates in the preparation of a wide variety of chemicals. Monochloroacetic acid can be used as a pre-emergent herbicide, dichloroacetic acid as an ingredient in some pharmaceutical products, and trichloroacetic acid as a herbicide, soil sterilant and antiseptic.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Based on preliminary data, concentrations of chloroacetic acids in Australian drinking waters range from 0.01 mg/L to 0.1 mg/L for chloroacetic acid, from 0.003 mg/L to 0.05 mg/L for dichloroacetic acid, and from 0.001 mg/L to 0.1 mg/L for trichloroacetic acid.

LIMITING FORMATION IN DRINKING WATER

The formation of chloroacetic acids in drinking water can be minimised by removing naturally occurring organic matter from the source water, reducing the amount of chlorine added, or using alternative disinfectants.

MEASUREMENT

The chloroacetic acids can be analysed by a liquid–liquid extraction procedure (USEPA Draft Method 552 1990). In this method the sample is adjusted to pH 11.5 and extracted with methyl tert-butyl ether (MTBE) to remove neutral and basic compounds. The sample is then acidified to pH 0.5 and the chloroacetic acids extracted into MTBE. The dried extracts are methylated, and the esters analysed by gas chromatography using electron capture detection. Limits of determination are lower than 0.001 mg/L.

HEALTH CONSIDERATIONS

Chloroacetic acids would be expected to be absorbed after ingestion in view of their water solubility, but there are no data to confirm this assumption. Dichloroacetate is rapidly metabolised to glyoxylate and oxalate by the liver, but no data are available on how the other chloroacetic acids are metabolised.

Dichloroacetic acid has been used in humans to control blood sugar and cholesterol levels. There are no studies on the short- or long-term exposure of humans to chloroacetic acid or trichloroacetic acid.

In rats and mice fed chloroacetic acid for two years, survival was decreased in rats at doses of 15–30 mg/kg body weight per day, whereas in mice, survival was affected at 100 mg/kg body weight per day (males) but not at 50 mg/kg body weight per day. There was no evidence of carcinogenic activity.

Rats given dicholoroacetic acid by gavage at 3 months developed brain lesions and increases in mean liver, kidney and adrenal weight at doses from 125 mg/kg body weight per day. Similar effects were observed at 3 months in dogs fed encapsulated dichloroacetate at 50 mg/kg body weight per day. Mice receiving dichloroacetate in their drinking water for a year had decreased body weight at doses from 410 mg/kg body weight per day, increased liver weight at doses from 77 mg/kg body weight per day, and an increase in the incidence of hepatocellular carcinomas and adenomas at doses from 295 mg/kg body weight per day.

Trichloroacetic acid administered in drinking water to rats for 90 days significantly increased liver peroxisomal activity at a dose of 355 mg/kg body weight per day. A 12-month drinking water study in mice reported increases in liver weight and hepatocellular carcinomas at doses from 178 mg/kg body weight per day.

No data are available on the genotoxicity of dichloroacetic acid. Trichloroacetic acid and chloroacetic acid are not mutagenic in tests using bacteria, but have shown some mutagenic activity in some mammalian cells.

DERIVATION OF GUIDELINES

The guideline values for the chloroacetic acids in drinking water were determined as follows:

i) Chloroacetic acid:

$$0.15 \text{ mg/L} = \frac{15 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day} \times 500} \times \frac{5}{7}$$

where:

- 15 mg/kg body weight per day is the lowest effect level based on a 2-year feeding study using rats (NTP 1992).
- 70 kg is the average weight of an adult.
- 0.2 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 500 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 5 for use of the low-effect level, which is close to the no-effect level).
- 5/7 is used to convert data based on a 5-day week feeding study to a 7-day week equivalent.

The 2004 World Health Organization (WHO) Guidelines do not have a health-based guideline for chloroacetic acid.

NOTE: Important general information is contained in PART II, Chapter 6

ii) Dichloroacetic acid:

$$0.1 \text{ mg/L} = \frac{7.6 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day} \times 500}$$

where:

- 7.6 mg/kg body weight per day is the no-effect level based on a 90-day drinking water study using mice (DeAngelo ***et al***. 1991).
- 500 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 5 for limited evidence of carcinogenicity).
- Other factors are as above.

The 2004 WHO provisional guideline value of 0.05 mg/L includes a factor of 10 for carcinogenicity. On review this was considered to be excessive and a lower factor was used.

iii) Trichloroacetic acid:

$$0.1 \text{ mg/L} = \frac{36 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.2}{2 \text{ L/day} \times 2000}$$

where:

- 36 mg/kg body weight per day is the no-effect level based on a 90-day drinking water study using male rats (Mather ***et al***. 1990).
- 2000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations, 10 for evidence of carcinogenicity in animals and 2 because a less than lifetime study was used but chronic studies are available).
- Other factors are as above.

The 2004 WHO has a health-based guideline value of 0.2 mg/L for trichloroacetic acid.

REFERENCES

DeAngelo AB, Daniel FB, Stober JA, Olsen GR (1991). The carcinogenicity of dichloroacetic acid in the male B6C3F mouse. *Fundamental and Applied Toxicology*, 16:337–347.

Mather GC, Exon JH, Koller LD (1990). Subchronic 90 day toxicity of dichloroacetic acid and trichloroacetic acid in rats. *Toxicology*, 64:71–80.

NTP (National Toxicology Program) (1992). Toxicology and carcinogenesis studies of monochloroacetic acid in F344/N rats and B6C3F1 mice (gavage studies). NTP TR 396, NIH Publication No. 92-2851. United States Department of Health and Human Services, Public Health Service, National Institute of Health.

USEPA Draft Method 552 (1990). Determination of haloacetic acids in drinking water by liquid–liquid extraction, derivatization, and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, First Addendum. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorobenzene

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (taste), the concentration of chlorobenzene in drinking water should not exceed 0.01 mg/L.

Chlorobenzene would not be a health concern unless the concentration exceeded 0.3 mg/L.

GENERAL DESCRIPTION

Chlorobenzene is used as a solvent and may be present in drinking water through contamination of water sources by spills or discharges. It has occasionally been detected in drinking water supplies in Canada and the United States at concentrations up to 0.005 mg/L. Inhalation from the atmosphere is believed to be the major route of environmental exposure.

Chlorobenzene has a low taste and odour threshold in water of about 0.01 mg/L.

It is used primarily as a solvent for pesticide formulations, in di-isocyanate manufacture, as a degreasing agent for mechanical parts, and in the production of nitrochlorobenzene. It is also used in the production of other halogenated organic compounds.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Chlorobenzene has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Aeration or adsorption onto granular activated carbon will remove chlorobenzene from water.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and chlorobenzene trapped on an adsorbent. The adsorbent is then heated and chlorobenzene analysed using gas chromatography with electron capture detection. The limit of determination is 0.0002 mg/L.

HEALTH CONSIDERATIONS

In humans, chlorobenzene is absorbed after ingestion or inhalation, and distributed primarily to adipose tissue and to the liver and kidney. It is metabolised into 4-chlorocatechol, which is excreted in urine.

An extensive review and summary of the human and animal toxicity data for chlorobenzenes is available (IPCS 1991).

There are few data on the effects of chlorobenzene on humans, and those that are available are of poor quality. They consist mainly of cases of poisoning and occupational exposure, with the principal effect being disturbances to the central nervous system.

Studies over 2 years using rats and mice reported adverse effects to the liver, kidneys, and blood-cell formation at high doses (250 mg/kg body weight per day). There is evidence of an increase of liver

NOTE: Important general information is contained in PART II, Chapter 6

tumours in male rats fed doses of 120 mg/kg body weight per day of monchlorobenzene for 2 years. No increases were observed in female rats, or in male and female mice. Chlorobenzene was not mutagenic in tests with bacteria, but may bind to RNA and DNA.

DERIVATION OF GUIDELINE

The health-based guideline value for chlorobenzene in drinking water was determined as follows:

$$0.3 \text{ mg/L} = \frac{60 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 500} \times \frac{5}{7}$$

where:

- 60 mg/kg body weight per day is the no-effect level from a 2-year gavage study using rats (NTP 1985).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 500 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 5 for limited evidence of carcinogenicity).
- 5/7 is used to convert data based on a 5 day per week feeding study to a 7-day week equivalent.

This health-based guideline value is greater than the taste and odour threshold of 0.01 mg/L.

REFERENCES

IPCS (International Programme on Chemical Safety) (1991). Chlorobenzenes other than hexachlorobenzene. Environmental Health Criteria, 128. World Health Organization, IPCS.

NTP (National Toxicology Program) (1985). Toxicology and carcinogenesis studies of chlorobenzene in F344/N rats and B6C3F1 mice (gavage studies). NTP Report No. 261, Publication No. 86-2517. United States Department of Health and Human Services, National Institutes of Health.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Chloroketones
1,1-dichloropropanone (dichloroacetone)
1,3-dichloropropanone
1,1,1-trichloropropanone (trichloroacetone)
1,1,3-trichloropropanone

(endorsed 1996)

GUIDELINE

Data are inadequate to set guideline values for chloroketones in drinking water.

GENERAL DESCRIPTION

The chloroketones are produced in drinking water as by-products of the reaction between naturally occurring organic matter and chlorine. No data are available on other sources or uses for these compounds.

Concentrations of chloroketones in drinking water reported overseas are very low and are estimated at less than 0.01 mg/L.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies 1,1,1-trichloropropanone has been recorded in concentrations up to 0.02 mg/L, but it is usually below the limit of determination of 0.0005 mg/L. No data are available for other chloroketones.

LIMITING FORMATION IN DRINKING WATER

The presence of chloroketones in drinking water can be minimised by removing naturally occurring organic matter from the source water, by reducing the amount of chlorine added, or by the use of alternative disinfectants.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of chloroketones (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and the chloroketones extracted using methyl tert-butyl ether. The extracts are then analysed using gas chromatography with an electron capture detector. Limits of determination are less than 0.0005 mg/L.

HEALTH CONSIDERATIONS

No data are available on absorption from the gastrointestinal tract, metabolism or health effects in humans.

Acute oral toxicity studies in mice using 1,1-dichloropropanone and 1,3-dichloropropanone have found no toxic effects with single doses of 130 mg/kg and 20 mg/kg respectively. No long-term toxicity studies have been reported.

Both 1,1-dichloropropanone and 1,3-dichloropropanone were direct-acting mutagens in tests with bacteria. There was some evidence that 1,3-dichloropranone initiated skin tumours in mice when applied at 50 mg/kg body weight per day for two weeks. There was no evidence that either 1,1-dichloropropanone or 1,1,1-trichloropropanone acted in this way.

The NHMRC Standing Committee on Toxicity reviewed the available data for chloroketones in 1991.

It was concluded that data were insufficient to set no-effect levels for these compounds.

REFERENCE

USEPA Draft Method 551, (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorophenols
2-chlorophenol
2,4-dichlorophenol
2,4,6-trichlorophenol

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, the concentration of chlorophenols in drinking water should not exceed the following values.

Chlorophenols would not be a health concern unless concentrations exceeded the health values listed.

	Health value	*Aesthetic value (odour and taste)*
2-chlorophenol	***0.3 mg/L***	***0.0001 mg/L***
2,4-dichlorophenol	***0.2 mg/L***	***0.0003 mg/L***
2,4,6-trichlorophenol	***0.02 mg/L***	***0.002 mg/L***

GENERAL DESCRIPTION

Chlorophenols may be present in drinking water as a result of chlorination of water that contains phenol or lower chlorophenols, or from contamination of water sources. Chlorination of water containing natural organic compounds can produce very low concentrations of chlorophenols. Degradation of phenoxy herbicides such as 2,4,5-T and 2,4-D also generates chlorophenols. The limited data available from overseas studies indicate that concentrations in drinking water are very low.

Chlorophenols have taste and odour thresholds in the range 0.0001 mg/L to 0.002 mg/L, with a characteristic antiseptic smell.

Chlorophenols are used commercially as preservatives, moth-proofing agents, germicides and anti-mildew agents. Exposure to chlorophenols via tap water has been estimated to be less than 10% of total dietary exposure.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on concentrations of chlorophenols in Australian drinking waters. If present at all, it is likely that concentrations would be extremely low.

TREATMENT OF DRINKING WATER

In pilot studies, granular activated carbon has successfully removed over 90% of 2-chlorophenol from water. It would probably be similarly effective in removing the other chlorophenols.

MEASUREMENT

Sensitive and isomer-specific procedures for the analysis of chlorophenols are available (USEPA Method 604 1986). The chlorophenols are derivatised with pentafluorobenzyl ether, and analysed using gas chromatography with electron capture detection. Limits of determination are 0.01 mg/L for monochlorophenol, 0.0005 mg/L for dichlorophenol and 0.00001 mg/L (10 ng/L) for trichlorophenol.

HEALTH CONSIDERATIONS

Chlorophenols are known to be efficiently absorbed and metabolised when administered orally to laboratory animals. Highest concentrations occur in the liver, brain and fat.

An extensive review and summary of the human and animal toxicity data for chlorophenols is available (IPCS 1989).

People occupationally exposed to chlorophenols often complain of irritation to the skin, mucous membranes and respiratory tract as a result of direct airborne contact. In addition, chronic ailments, skin lesions and ulcerations (particularly chloracne), and clinical indications of liver damage and neurological effects have also been reported, particularly in association with high exposures.

There have been a number of studies on the toxic effects of chlorophenols in rats and mice. Short-term exposure to high doses results in an increased respiration rate, motor weakness, tremors, convulsion, coma and death. Long-term studies over 2 years could not determine any specific dose-related effects using either 2-chlorophenol or 2,4-dichlorophenol, but 2,4,6-trichlorophenol induced leukaemia and lymphomas in male rats, and liver cancer in male and female mice.

No information is available on the mutagenic effects of 2-chlorophenol. Mutagenic tests on bacteria were negative for 2,4-dichlorophenol. Separate tests gave weakly positive and negative results for 2,4,6-trichlorophenol.

The International Agency for Research on Cancer has concluded that 2,4,6-trichlorophenol is possibly carcinogenic to humans (group 2B, sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline values for the chlorophenols in drinking water, based on health considerations, were determined as follows:

i) 2-chlorophenol:

$$0.3 \text{ mg/L} = \frac{7.5 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 7.5 mg/kg body weight per day is the no-effect level based on a 2-year drinking water study using rats (Exon and Koller 1985).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in using the results of an animal study as a basis for human. exposure (10 for interspecies variations and 10 for intraspecies variations). The use of this safety factor was recommended by the NHMRC Standing Committee on Toxicity.

The World Health Organization (WHO) Guidelines do not have a health-based guideline for 2-chlorophenol.

ii) 2,4-dichlorophenol:

$$0.2 \text{ mg/L} = \frac{4.5 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 4.5 mg/kg body weight per day is the no-effect level based on a 2-year drinking water study using rats (Exon and Koller 1985). The use of this value was recommended by the NHMRC Standing Committee on Toxicity following a review of the available toxicity data for the chlorophenols.
- Other factors are as above.

The WHO Guidelines do not have a health-based guideline for 2,4-dichlorophenol.

iii) 2,4,6-trichlorophenol:

$$0.02 \text{ mg/L} = \frac{4.5 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 4.5 mg/kg body weight per day is the no-effect level based on a 2-year drinking water study using rats (Exon and Koller 1985). The use of this value was recommended by the NHMRC Standing Committee on Toxicity following a review of the available toxicity data for the chlorophenols.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for carcinogenic effects).
- Other factors are as above.

The WHO guideline value of 0.2 mg/L for 2,4,6-trichlorophenol was based on a calculation that estimated an additional lifetime risk of one fatal cancer per hundred thousand people (WHO 2006).

As the guideline values based on health considerations are greater than the taste thresholds for these compounds, the taste thresholds should be used as the guideline values.

REFERENCES

Exon JH, Koller LD (1985). Toxicity of 2-chlorophenol, 2,4-dichlorophenol and 2,4,6-trichlorophenol. Water Chlorination: Chemistry, Environmental Impact and Health Effects. Proceedings of the 5th Conference on Water Chlorination: Environmental Impact and Health Effects, Williamsburg, Virginia, June 3-8, 1984. Jolly RL *et al.* (editors), Chelsea, Michigan, Lewis Publisher Inc., 5, 307–330.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1989). Chlorophenols other than Pentachlorophenol. Environmental Health Criteria, 93. World Health Organization, IPCS.

USEPA Method 604 (1986). Phenols. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Chloropicrin

(endorsed 2011)

GUIDELINE

Data are inadequate to set a health-based guideline for chloropicrin in drinking water.

RELATED CHEMICALS

Chloropicrin (CAS 76-06-2) belongs to the fumigant class of chemicals. Other pesticides in this class include methyl bromide and sulfuryl fluoride (Tomlin 2006).

HUMAN RISK STATEMENT

There are currently insufficient data on which to base a human risk statement.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chloropicrin is a fumigant used in soil to control soil-borne fungi, diseases and nematodes. It is also used to fumigate stored products and to treat wood poles and timbers for internal decay by fungi and insects. Because of its pungent odour, it is also used as a warning agent in combination with other fumigants. Chloropicrin, or trichloronitromethane (Cl_3CNO_2), is a liquid that volatilises readily when released into the atmosphere.

There are registered products containing chlorpicrin in Australia. The products are all for professional use and are applied either by hand equipment or machinery. Currently, the only products containing chlorpicrin that are applied to stored food commodities also contain methyl bromide, which is in the phase-out stage (except for quarantine uses). Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure: Public exposure is unlikely as residues are not allowed in food and are at the level of detection in cereal grains. It is also unlikely to be found in drinking water due to its physicochemical properties.

Since chloropicrin is highly soluble in water and has low adsorption in soil, it can potentially leach into groundwater and to surface water through run-off under a flooded condition. The low octanol/water partition coefficient of chloropicrin also indicates that it is not likely to be bioconcentrated in tissues of aquatic organisms (USEPA 2008).

A major route of exposure to chloropicrin is likely to be inhalation from gaseous sources, such as leaking and venting from fumigation chambers, and gas escape when using the product.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on concentrations of chloropicrin in Australian drinking waters. Chloropicrin has been detected in drinking water supplies in the USA at concentrations of less than 0.005 mg/L (USEPA 1990) and in Korea at concentrations less than 0.003 mg/L (Lee *et al.* 2001).

TREATMENT OF DRINKING WATER

There are no reports of the treatment of chloropicrin in drinking water.

However, given that chloropicrin may be formed in water as a chlorination disinfection by-product, the presence of chloropicrin in drinking water can be minimised by removing naturally occurring organic matter from the source water, by optimising disinfection practices, or by using alternative disinfectants. Most importantly, adequate disinfection must remain the primary concern and should not be compromised in response to a perceived need to lower levels of chloropicrin that may be detected.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of chloropicrin (USEPA method 551 1990). Sodium chloride is added to the sample, and the chloropicrin extracted using methyl tert-butyl ether. The extract is then analysed using gas chromatography with an electron capture detector. The limit of detection is approximately 20 ng/L. Other methods include purge and trap-gas chromatography-mass spectrometry, with detection limits reported as 250 ng/L (Lee *et al*. 2001).

HISTORY OF THE HEALTH VALUES

No acceptable daily intake (ADI) or acute reference dose (ARfD) values have been established for chloropicrin.

The available toxicity data on chloropicrin have not been evaluated to establish a health value.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Chloropicrin has had only limited toxicological assessment by Australian authorities. The Australian Government Department of Health and Ageing proposed chloropicrin for a review by the Australian Pesticides and Veterinary Medicines Authority in 2008, based on the absence of data to determine a safe level of exposure, especially to workers.

The United States Environmental Protection Agency (USEPA) selected a reversible acute endpoint using a human sensory irritation study with a benchmark concentration level ($BMCL_{10}$) of 0.073 parts per million (0.073 mg/L). At this level, the USEPA does not expect eye or nose irritation, upper respiratory changes, or any other health effects (USEPA 2008).

Poisons Schedule: Chloropicrin is included in Schedule 6 or 7 and Appendix J (Conditions for availability and use of Schedule 7 poisons) of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on concentration Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

As a no-observed-effect level (NOEL) for repeat exposure to chloropicrin has not been determined, it is not possible to determine a health-based guideline value for chloropicrin in drinking water.

The World Health Organization has not established a guideline value for chloropicrin because of inadequate data (WHO 2004).

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lee JL, Kim BH, Hong JE, Pyo HS, Park S-J, Lee DW (2001). A study on the distribution of chlorination by-products (CBPs) in treated water in Korea. *Water Research,* 35(12):2861-2872.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1990). Draft Method 551: Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid-liquid extraction and gas chromatography with electron capture detection. USEPA, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

USEPA (United States Environmental Protection Agency) (2008). Office of Pesticide Programs RED Fact Sheet: Chloropicrin, 10th July 2008.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorothalonil

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlorothalonil in drinking water should not exceed 0.05 mg/L.

RELATED CHEMICALS

Chlorothalonil (CAS 1897-45-6) belongs to the chloronitrile class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chlorothalonil would not be a health concern unless the concentration exceeded 0.05 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlorothalonil is a fungicide used for the control of fungal diseases in fruits and vegetables, as well as in turf, ornamentals, freshly sawn *Pinus* spp timber, and in various tress and vine crops.

There are registered products that contain chlorothalonil in Australia. These products are intended for professional and for home garden use and are available as concentrated solutions to be applied in diluted form using ground or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to chlorothalonil and its metabolites are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of chlorothalonil may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of chlorothalonil in Australian drinking waters have been identified. However, chlorothalonil has been occasionally identified in drinking source waters in the USA (Walker *et al.* 2000).

TREATMENT OF DRINKING WATER

No specific data on the treatment of chlorothalonil in drinking water have been identified.

MEASUREMENT

Chlorothalonil can be measured in surface waters by liquid chromatography with ultraviolet (UV) detection, with a practical limit of quantitation of 25 ng/L (Ozhan and Alpertunga 2007). Similar detection limits may alternatively be achieved by solid-phase microextraction and gas chromatography coupled with electron-capture and mass spectrometric detection (Lambropoulou *et al.* 2000).

Chlorothalonil can be measured in surface waters by liquid chromatography with UV analysis

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for chlorothalonil is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.5 mg/kg bw/day from a long-term (2-year) dietary study in mice and dogs. The NOEL is based on lesions in the kidney and the stomach. The ADI incorporates a safety factor of 100 and was established in 1991.

The previous ADI was 0.4 mg/kg bw/day based on a NOEL of 800 mg/kg bw/day. The ADI incorporated a safety factor of 2000 and was set in 1973. In 1986, the Joint FAO/WHO Meeting on Pesticide Residues, recommended a temporary ADI of 0.0005 mg/kg bw based on a NOEL of 0.5 mg/kg bw and a safety factor of 1000. The NOEL was based on an increased incidence of renal tumours at high doses in a 2-year rat study. The transitional ADI was pending additional studies on carcinogenicity in mice and rats.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Chlorothalonil is absorbed via the gastrointestinal tract. It is largely eliminated in the faeces (95%) within 24 hours, with minor excretion of the parent compound or its metabolites in the urine. The major metabolite identified was 4-hydroxy-2,3,5-trichloroisophthalonitrile.

Acute effects: Chlorothalonil has low acute oral and dermal toxicity. Chlorothalonil is not a skin sensitiser.

Short-term effects: In a 90-day dietary study in mice and rats, kidney and liver weights were increased at doses of 71.4 and 75 mg/kg bw/day, respectively. The incidence of hyperplasia and hyperkeratosis of stomach epithelial cells was also increased at doses of 7.1 mg/kg bw/day and above in mice and at the lowest dose tested of 40 mg/kg bw/d and above (tested up to 1500 mg/kg bw/d) in rats. A separate medium-term dietary study in rats reported increased kidney weight at 3 mg/kg bw/day.

Long-term effects: Two-year dietary studies were conducted in mice, rats and dogs. The mouse study reported histopathological findings in the kidney at dose levels above 1.5 mg/kg bw. The rat study reported renal toxicity at 40 mg/kg bw/day. The dog study reported slight nephrotoxicity at 3 mg/kg bw. The overall NOEL was 1.5 mg/kg bw/day in the mouse and dog studies, and this was the basis for the ADI.

Carcinogenicity: There was evidence of carcinogenicity in both the mouse and rat studies. In mice, forestomach tumours occurred at 26 mg/kg bw/day as a result of increased hyperplasia and hyperkeratosis at 6 mg/kg bw/day and above. In rats, renal tumours and effects indicative of renal toxicity occurred at the lowest dose of 40 mg/kg bw/day. This dose level was considered well in excess of the likely level of human exposure.

Genotoxicity: There was equivocal evidence of genotoxicity from *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats did not produce any evidence of reproductive effects. In developmental toxicity studies in rats and rabbits, there was evidence of maternotoxicity in rats and foetotoxicity in rabbits at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Chlorothalonil is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.05 mg/L for chlorothalonil was determined as follows:

$$0.05 \text{ mg/L} = \frac{1.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.5 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice and dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for chlorothalonil and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lambropoulou DA, Konstantinou IK, Albanis TA (2000). Determination of fungicides in natural waters using solid-phase microextraction and gas chromatography coupled with electron-capture and mass spectrometric detection. *Journal of Chromatography A,* 893(1):143-156.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ozhan G, Alpertunga B (2007). Determination of various fungicides in water by LC-DAD analysis. *Fresenius Environmental Bulletin,* 16(8):880-886.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Walker AE, Holman RE, Leidy RB (2000). ELISA and GC/MS analysis of pesticide residues in North Carolina. *Journal of the American Water Resources Association,* 36(1):67-74.

NOTE: Important general information is contained in PART II, Chapter 6

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorpyrifos

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlorpyrifos in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Chlorpyrifos (CAS 39475-55-3) is in the organophosphate class of chemicals. There are many other pesticides in this class, including fenthion, parathion, profenofos and ethoprofos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chlorpyrifos would not be a health concern unless the concentration exceeded 0.01 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlorpyrifos is a broad-spectrum insecticide used to control a broad range of insect pests in many crops, the home and commercial sites.

There are approximately 100 products containing chlorpyrifos. These products are intended for professional and home garden use and are available in many different formulations for the large variety of use patterns. The most common application methods include vehicle-mounted equipment, hand-held equipment, and aircraft for spraying and soil injection. Aerial ultra-low volume (ULV) application is permitted.

Exposure sources: The main sources of public exposure to chlorpyrifos and its metabolites are home and garden uses, and residues in food. Residue levels in food produced according to good agricultural practice are anticipated to be generally low.

Agricultural use of chlorpyrifos may potentially lead to contamination of source waters through processes such as run-off, spray drift (especially from aerial application) or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Surface water contamination arising from agricultural uses of chlorpyrifos are generally below 1 µg/L on the rare occasions that chlorpyrifos is detected in Australian surface waters (APVMA 2000). Extensive monitoring has been conducted in the cotton areas of northern New South Wales and the irrigation areas in southern New South Wales. There are a few high outliers, reaching 26 µg/L in northern rivers and 25 µg/L in irrigation drainage adjacent to rice bays in southern New South Wales, but these appear to be isolated occurrences, with such levels seldom detected because of the limited aquatic persistence of chlorpyrifos (APVMA 2000).

TREATMENT OF DRINKING WATER

Chlorpyrifos has been shown to be completely removed from water by chlorination when the chlorine dose is adjusted to match chlorine demand. Nanofiltration has also been shown to be highly effective (Kiso *et al*, 2000).

Ozonation and activated carbon adsorption for chlorpyrifos removal has also been reported to have moderate success (Ormad *et al*. 2008). Conventional coagulation/flocculation has been shown to provide a relatively low removal rate.

More research is required to investigate the effectiveness of adsorption or oxidation methods. Jar testing to identify the effectiveness of various removal methods in specific waters is recommended if chlorpyrifos is detected.

MEASUREMENT

The practical limit of quantification for chlorpyrifos in water is 0.001 mg/L by liquid chromatography with tandem mass spectrometry (Alder *et al*. 2006).

HISTORY OF THE HEALTH VALUES

The acceptable daily intake (ADI) for chlorpyrifos is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.03 mg/kg bw/day. The NOEL is based on plasma cholinesterase inhibition in a 28-day human volunteer study. The ADI was established in 1998 and reaffirmed in 2000, and incorporates a safety factor of 10.

The acute reference dose (ARfD) of 0.1 mg/kg bw/day for chlorpyrifos was established in 2000, based on a NOEL of 1 mg/kg bw/day. The NOEL was based on inhibition of red blood cell acetylcholinesterase inhibition from a 3-day human volunteer oral study. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Chlorpyrifos is readily absorbed from the gastrointestinal tract, widely distributed throughout the body, and excreted rapidly in the urine and faeces. Only low tissue residues have been detected. The major urinary metabolite of chlorpyrifos identified was 3,5,6-trichloro-2-pyridinol (TCP).

Acute effects: Chlorpyrifos has moderate to high acute oral toxicity and low dermal toxicity. It is not a skin sensitiser. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes.

Short-term and long-term effects: Short-term and long-term studies in mice, rats, dogs and monkeys resulted in symptoms indicative of central nervous system toxicity. The most sensitive effect observed was inhibition of plasma acetylcholinesterase in rats at an oral dose of 0.03 mg/kg bw/day. Effects at higher doses included reductions in bodyweight, and increased adrenal and liver weights. In a 28-day human study, the NOEL based on cholinesterase inhibition was 0.03 mg/kg bw/day. This NOEL was the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for chlorpyrifos.

Genotoxicity: There was no evidence of genotoxicity in short-term *in vitro* or *in vivo* studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: There was no evidence of delayed neurotoxicity in chicken and rat studies, nor was there any evidence of developmental neurotoxicity in a developmental study in rats.

Poisons Schedule: Chlorpyrifos is included in Schedule 5 or 6 in the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) No.1 (2010), depending on the concentration, formulation type or use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for chlorpyrifos was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.03 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.03 mg/kg bw/day is the NOEL based on a short-term (28-day) volunteer study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is a safety factor applied to the NOEL derived from human studies to allow for intraspecies variation.

The World Health Organization has a guideline value of 0.03 mg/L for chlorpyrifos (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Alder L, Greulich K, Kempe G, Vieth B (2006). Residue analysis of 500 high priority pesticides: better by GC-MS or LC-MS/MS?. *Mass Spectrometry Reviews,* 25(6):838-65.

APVMA (2000) Chlorpyrifos Interim Review Report. Available from http://www.apvma.gov.au/products/review/current/chlorpyrifos.php

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Kiso Y, Nishimura Y, Kitao T, Nishimura K (2000). Rejection properties of non-phenylic pesticides with nanofiltration membranes. *Journal of Membrane Science*, 171:229-237.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71:97-106.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Chlorsulfuron

(endorsed 2011)

GUIDELINE

Based on human health concerns, chlorsulfuron in drinking water should not exceed 0.2 mg/L.

RELATED CHEMICALS

Chlorsulfuron (CAS 64902-72-3) belongs to the sulfonylurea herbicide class of chemicals. Other pesticides in this class include azimsulfuron, etametsulfuron-methyl, ethoxysulfuron, metsulfuron-methyl, halosulfuron-methyl, iodosulfuron methyl-sodium salt, sulfometuron methyl, sulfosulfuron, triasulfuron, tribenuron methyl and trifloxysulfuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chlorsulfuron would not be a health concern unless the concentration exceeded 0.2 mg/L. Excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Chlorsulfuron is a selective post-emergence herbicide used for control of broad-leaf weeds in cereal crops.

There are currently products containing chlorsulfuron registered in Australia. These products are intended for professional use and are available in wettable powder and granular formulations. Product labels indicate products are to be diluted and applied directly to crops and soil by ground and aerial spray application methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to chlorsulfuron and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of chlorsulfuron may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on chlorsulfuron in Australian waters. In the USA, modelling by the United States Environmental Protection Agency suggests maximum concentrations of 41.3 µg/L and 3.5 µg/L in surface water and groundwater, respectively (USEPA, 2005).

TREATMENT OF DRINKING WATER

No specific data on the treatment of chlorsulfuron in drinking water have been identified.

MEASUREMENT

Chlorsulfuron in water can be measured by high performance liquid chromatography with ultraviolet detector, with a limit of detection of 1 µg/L (Sarmah and Kookana 1999).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for chlorsulfuron is 0.05 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day from a long-term (2-year dietary) study. This NOEL is based on haematological changes observed in rats. The ADI incorporates a safety factor of 100 and was established in 1982.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Chlorsulfuron is not extensively metabolised, and is rapidly excreted mostly unchanged in the urine. A small portion is metabolised to a sulfonamide and a triazine residue.

Acute effects: Chlorsulfuron has low acute oral and dermal toxicity and is not a skin sensitiser. The minor amounts of sulfonamide and triazine metabolites formed also have low acute oral toxicity and are not skin sensitisers.

Short-term effects: Medium-term dietary studies in mice and rats reported haematological changes and decreased urine pH at doses above 8 mg/kg bw/day.

Short-term dietary studies in rats indicated that chlorsulfuron metabolites are less toxic than their parent compound.

Long-term effects: Long-term dietary studies in mice and rats reported reduced bodyweight as the major effect, occurring markedly at 120 mg/kg bw/day in rats, with the only effect noted in mice at 750 mg/kg bw/day. In addition, male rats had minor changes in haematological parameters (increased red blood cell volume, mean cell haemoglobin and haematocrit) in addition to reduced bodyweight from 25 mg/kg bw/day, but only at the 1-year interim. The NOEL for the rat study was 5 mg/kg bw/day, and this is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in rats, there is no evidence of carcinogenicity for chlorsulfoton

Genotoxicity: Chlorsulfuron and its metabolites are not considered genotoxic, based on a variety of *in vitro* and *in vivo* short-term tests.

Reproductive and developmental effects: Three-generation reproductive studies in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Chlorsulfuron is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.2 mg/L for chlorsulfuron was determined as follows:

$$0.2 \text{ mg/L} = \frac{5 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 5 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation. No additional safety was considered necessary.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health. This includes the NDPSC and PACC references below.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Sarmah AK, Kookana RS (1999). Simultaneous analysis of triasulfuron, metsulfuron-methyl and chlorsulfuron in water and alkaline soils by high-performance liquid chromatography. *Journal of Environmental Science and Health Part B*, 34(3):363-380.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (2005). Reregistration eligibility decision for chlorsulfuron, case no. 0631. 20 May 2005. United States Environmental Protection Agency.

Chromium

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of hexavalent chromium (Cr(VI)) in drinking water should not exceed 0.05 mg/L. If the concentration of total chromium exceeds this value then a separate analysis for hexavalent chromium should be undertaken.

GENERAL DESCRIPTION

Chromium is present in the environment in the trivalent (Cr(III)) and hexavalent (Cr(VI)) states.

Trivalent chromium is the most common naturally occurring state. Most soils and rocks contain small amounts of chromium oxide, and weathering, oxidation and bacterial action convert this insoluble compound into soluble Cr(III) salts.

Trivalent chromium salts are used in leather tanning, manufacture of catalysts, paint pigments, fungicides, and ceramic and glass manufacture.

Trivalent chromium is an essential trace element for humans, with food being the major source of intake.

Hexavalent chromium occurs infrequently in nature. Its presence in water is generally the result of industrial and domestic chromium waste discharges. Hexavalent chromium compounds are used in the metallurgical industry for chrome alloy and chrome metal production, and in the chemical industry as oxidising agents.

Hexavalent chromium is not considered to be an essential nutrient and harmful effects due to chromium have been attributed to this form.

Total chromium concentrations in drinking water are usually less than 0.005 mg/L although concentrations between 0.06 mg/L to 0.12 mg/L have been reported overseas.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies concentrations of total chromium range up to 0.03 mg/L, with typical concentrations usually less than 0.005 mg/L.

TREATMENT OF DRINKING WATER

Chromium can be removed from drinking water sources by coagulation/filtration, ion exchange, reverse osmosis and lime softening. Trivalent chromium can be oxidised to hexavalent chromium with disinfectants, particularly chlorine, chlorine dioxide and ozone.

MEASUREMENT

The total chromium concentration in drinking water can be determined by inductively coupled plasma emission spectroscopy or graphite furnace atomic absorption spectroscopy (APHA Method 3500-Cr Parts B or C 1992). The limit of determination is approximately 0.01 mg/L.

Hexavalent chromium (Cr(VI)) can be determined with a colorimetric method using diphenylcarbizide (APHA Method 3500-Cr part D 1992). The limit of determination is 0.005 mg/L.

HEALTH CONSIDERATIONS

The absorption of chromium after ingestion is low and depends on the valence state. Hexavalent chromium is more readily absorbed from the gastrointestinal tract than trivalent compounds. It is able to penetrate cell membranes, and within cells it is reduced to Cr(III) and forms complexes with proteins and genetic material.

An extensive review and summary of the human and animal toxicity data for chromium is available (IPCS 1988).

Epidemiological studies have found an association between inhalation of hexavalent chromium compounds and lung cancer, especially in humans occupationally exposed during chromate production. There is no evidence that organs other than the lung are affected or that ingestion of hexavalent chromium compounds can cause cancer.

There are sufficient animal data to indicate that many hexavalent chromium compounds are carcinogenic. Hexavalent chromium compounds also cause mutations and chromosome aberrations in a variety of test systems. The mutagenic activity can be decreased or abolished by reducing agents, such as gastric juice.

In animal studies, orally administered trivalent chromium compounds have not been shown to induce cancer or to induce mutations in genetic material.

The International Agency for Research on Cancer has concluded that hexavalent chromium is carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans); and that trivalent chromium is not classifiable as to its carcinogenicity to humans (Group 3, inadequate evidence in humans and inadequate evidence in animals) (IARC 1990).

DERIVATION OF GUIDELINE

The guideline value for chromium in drinking water is based on a World Health Organization assessment and should be reviewed when more toxicological data become available. It was adopted after consideration of the following points:

- The guideline value of 0.05 mg/L has been used in many countries for a number of years with no known cases of chromium toxicity.
- The value was originally set following a conservative assessment of studies on the toxicity of hexavalent chromium to rats (Mackenzie ***et al***. 1958).
- Trivalent chromium is essential for human health and has no known toxic effects.
- Data are insufficient to determine whether a higher value would be equally safe.

Analysis for the separate valence states of chromium is time consuming and hence the guideline value applies to total chromium. If concentrations of total chromium exceed the guideline value, it is recommended that separate analyses for Cr(VI) and Cr(III) be undertaken.

REFERENCES

APHA Method 3500-Cr Part B (1992). Chromium: Atomic Absorption method for total chromium. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Cr Part C (1992). Chromium: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Cr Part D (1992). Chromium: Colorimetric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

IARC (International Agency for Research on Cancer) (1990). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: chromium, nickel and welding. World Health Organization, IARC, 49.

IPCS (International Programme on Chemical Safety) (1988). Chromium. Environmental Health Criteria, 61. World Health Organization, IPCS.

MacKenzie RD, Byerrum RU, Decker CF, Hoppert CA, Langham RF (1958). Chronic toxicity studies: II. Hexavalent and trivalent chromium administered in drinking water to rats. *American Medical Association Archives of Industrial Health*, 18:232–234.

NOTE: Important general information is contained in PART II, Chapter 6

Clopyralid

(endorsed 2011)

GUIDELINE

Based on human health concerns, clopyralid in drinking water should not exceed 2 mg/L.

RELATED CHEMICALS

Clopyralid (CAS 1702-17-6) belongs to the pyridinecarboxylic acid class of chemicals. Other pesticides in this class include aminopyralid, fluroxypyr, picloram, picolinafen and triclopyr (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, clopyralid would not be a health concern unless the concentration exceeded 2 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Clopyralid is used as a herbicide for the control of broad-leaf weeds in turf, industrial situations and agricultural crops.

There are currently products registered in Australia that contain clopyralid, its potassium salt, its trisopropanolamine salt or its monoethanolamine salt. Clopyralid products are intended for professional use. Products are not intended for use in the home garden. Clopyralid is available as concentrated solutions, powder and granular formulations to be applied in diluted form using boom, aerial or hand-held spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to clopyralid and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of clopyralid may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of clopyralid in Australian drinking waters have been identified. However, an estimated mean annual calculated concentration of 24 ng/L and a maximum concentration of 1 µg/L has been reported in drinking water supplies of the northern Great Plains of the USA (Donald *et al.* 2007).

TREATMENT OF DRINKING WATER

No specific data on the treatment of clopyralid in drinking water have been identified.

MEASUREMENT

Clopyralid can be measured in drinking waters by solvent extraction followed by gas-chromatography with mass spectrometry detection. The practical limit of detection is 0.6 ng/L (Donald *et al*. 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for clopyralid is 0.5 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 50 mg/kg bw/day from a long-term (2-year dietary) study. The NOEL is based on decreased bodyweight gain and adverse effects in the stomach epithelium observed in rats. The ADI incorporates a safety factor of 100 and was established in 1982.

The previous *Australian Drinking Water Guidelines* health value was 1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

The toxicological database for clopyralid consists of toxicity studies on the parent compound.

Metabolism: No metabolism studies in animals are available for clopyralid. A short-term human volunteer study reported elimination of clopyralid to be rapid and occur mainly via the urine. Metabolites were not measured in this study.

Acute effects: Clopyralid has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Two- and four-week dietary studies in rats and dogs indicate the kidney and liver are target organs of clopyralid toxicity. Decreased bodyweight was reported at the highest dose level of 1500 mg/kg bw/day. The most sensitive toxicological effects observed were increased blood urea nitrogen levels and proliferative changes in the stomach epithelium at dose levels above 150 mg/kg bw/day in rats.

Medium-term (90-day) dietary studies were conducted in mice, rats and dogs. In rats, increased kidney and liver weights were observed at doses of 300 mg/kg bw/day. There were no treatment-related effects observed in dogs at the highest dose tested (150 mg/kg bw/day).

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, decreased bodyweight gain was observed at the high dose of 2000 mg/kg bw/day. In dogs, changes in haematological parameters and increased liver and kidney weights were observed above a dose level of 100 mg/kg bw/day. In rats, hyperplasia in stomach epithelium and decreased bodyweight gain were observed at a dose of 100 mg/kg bw/day. The NOEL based on decreased bodyweight gain and effects on the gastric epithelium was 50 mg/kg bw/day, and is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for clopyralid.

Genotoxicity: Clopyralid is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits produced no evidence of adverse effects on reproduction or the developing foetus.

Poisons Schedule: Clopyralid is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 2 mg/L for clopyralid was determined as follows:

$$2 \text{ mg/L} = \frac{50 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 50 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Donald DB, AJ Cessna, E Sverko, NE Glozier (2007). Pesticides in surface drinking-water supplies of the northern Great Plains (Research). *Environmental Health Perspectives*, 115(8):1183(9).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Colour (True)

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, true colour in drinking water should not exceed 15 HU.

GENERAL DESCRIPTION

Two terms are used to describe colour. 'True colour' is the colour after particulate matter has been removed (usually by filtration through a 0.45 micrometer pore size filter). 'Apparent colour' is what one actually sees; it is the colour resulting from the combined effect of true colour and any particulate matter, or turbidity. In turbid waters, the true colour is substantially less than the apparent colour.

In natural waters, colour is due mainly to the presence of dissolved organic matter including humic and fulvic acids, which originate from soil and decaying vegetable matter. Surface water can also be coloured by waste discharges, for example from dyeing operations in the textile industry, and paper manufacture.

The dissolution of metals in pipes and fittings can also discolour drinking water. Badly corroded iron pipes can produce a brownish colour whereas corrosion of copper pipes can produce a blue-green colouration on sanitary ware and a faint blue colour in water in extreme cases. The condition of household pipes can significantly influence water colour.

In bore water, 'red water' is a frequent problem, caused by the oxidation of iron. In addition, a black discolouration in reservoirs and distribution systems can result from the action of bacteria on dissolved manganese to produce insoluble oxides. Some of these compounds form fine suspensions, or are only partially dissolved, and so contribute to apparent rather than true colour. (See Section 5.6 *Nuisance organisms*.)

As a guide, tea has a colour of about 2500 Hazen units (HU, see below). A true colour of 15 HU can be detected in a glass of water, and a true colour of 5 HU can be seen in larger volumes of water, for instance in a white bath. Few people can detect a true colour level of 3 HU, and a true colour of up to 25 HU would probably be accepted by most people provided the turbidity was low. Some examples of drinking water with differing turbidity and colour are shown in Plate 1.

True colour is preferred analytically, as the measurement is more precise than for apparent colour, and not as dependent on site or time. If both true colour and turbidity are at the guideline values (i.e. true colour of 15 HU and turbidity of 5 NTU[Nephelometric Turbidity Units]), the apparent colour could be 20 HU. This is considered to be acceptable.

Variations in colour are likely to lead to more complaints than a high but consistent colour.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies true colour ranges from 1 HU to 25 HU for filtered or fully treated supplies, and from 1 HU to 85 HU for unfiltered supplies.

MEASUREMENT

Colour can be measured spectrophotometrically or using a visual comparator. In both cases, the standard unit of measurement is the Hazen unit (HU). (True colour is often quoted as True Colour Units, or TCU; however, the numerical values are identical.) Hazen units are defined in terms of a platinum–cobalt standard (APHA Method 2120B 1992). This standard was developed for the analysis of colour in natural

waters with a yellow-brown appearance, and is not applicable to waters with different colours.

It is advisable to record the pH with the colour measurement, as the colour of natural surface waters increases with pH.

Colour values obtained using a spectrophotometer are dependent on the wavelength used for the measurement. There is no standard wavelength used in Australia, but values ranging from 395 nm to 465 nm are generally used. In the absence of a suitable Australian Standard, the British Standard, which uses 436 nm (BSI Method BS6068 1986), is suitable.

TREATMENT OF DRINKING WATER

Constituents of natural colour derived from humic and fulvic acids can be reduced by coagulation followed by filtration (AWWA 1990). Oxidation by chlorine or ozone will also reduce colour but may produce undesirable by-products.

HEALTH CONSIDERATIONS

Colour is generally related to organic content, and while colour derived from natural sources such as humic and fulvic acids is not a health consideration, chlorination of such water can produce a variety of chlorinated organic compounds as by-products (see Section 6.3.2 on disinfection by-products). If the colour is high at the time of disinfection, then the water should be checked for disinfection by-products. It should be noted, however, that low colour at the time of disinfection does not necessarily mean that the concentration of disinfection by-products will be low.

Reactions between naturally occurring humic and fulvic material and water disinfectants (such as chlorine, ozone, chloramines and chlorine dioxide) can also cause difficulties in maintaining an adequate level of disinfectant, thus creating the opportunity for bacterial reinfection or regrowth.

The solubility of some organic pollutants can also be increased through complex formation with humic material.

Coloured water may prompt people to seek other, perhaps less safe, sources of drinking water.

DERIVATION OF GUIDELINE

The guideline value is based on the colour that is just noticeable in a glass of water. This is generally accepted as being 15 HU.

GUIDELINES IN OTHER COUNTRIES

The Canadian Guidelines and the 1984 World Health Organization (WHO) Guidelines both recommend a value of 15 HU. The 1993 WHO Guidelines indicate that a colour above 15 TCU may give rise to consumer complaints.

The United States EPA Secondary Drinking water Regulations have a maximum concentration for colour of 15 HU.

The European Economic Community Standards for colour are a maximum admissible value of 20 HU and a guideline value of 1 HU.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 2120B (1992). Colour: Visual comparison method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

AWWA (American Water Works Association) (1990). *Water Quality and Treatment: A handbook of community water supplies*. AWWA, 4th edition, McGraw-Hill Inc.

BSI Method BS6068 (1986). Examination and determination of colour. British Standards Institution, British Standard for Water Quality, Section 2.22.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

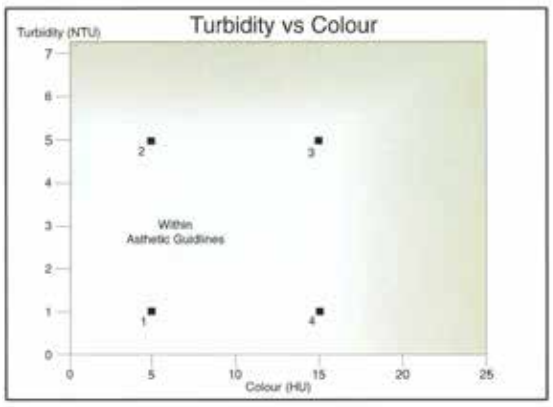
Colour and Turbidity

1. Colour = 5 HU
   Turbidity = 1 NTU

2. Colour = 5 HU
   Turbidity = 5 NTU

3. Colour = 15 HU
   Turbidity = 5 NTU

4. Colour = 15 HU
   Turbidity = 1 NTU



NOTE: Important general information is contained in PART II, Chapter 6

Copper

(endorsed 2001)

GUIDELINE

Based on health considerations, the concentration of copper in drinking water should not exceed 2 mg/L.

Based on aesthetic considerations, the concentration of copper in drinking water should not exceed 1 mg/L.

GENERAL DESCRIPTION

Copper is widely distributed in rocks and soils as carbonate and sulfide minerals.

Copper is relatively resistant to corrosion and is used in domestic water supply pipes and fittings. It is also used in the electroplating and chemical industries, and in many household goods. Copper sulfate is used extensively to control the growth of algae in water storages.

Copper is present in uncontaminated surface waters at very low concentrations, usually less than 0.01 mg/L. The concentration can rise substantially when water with a low pH and hardness remains in stagnant contact with copper pipes and fittings. Under these conditions, the concentration of copper can reach 5 mg/L or higher. In one extreme case overseas, a concentration of 22 mg/L was reported.

The taste threshold for copper is in the range 1–5 mg/L, depending on the water purity. Concentrations above 1 mg/L may cause blue or green stains on sanitary ware. Such stains may also be due to slowly leaking taps, where copper corrosion occurs over a long time, and are not necessarily due to high concentrations of copper in drinking water.

Food is the main source of copper intake. Intake from water would normally be less than 10% of total intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, total copper concentrations range up to 0.8 mg/L, with typical concentrations of about 0.05 mg/L.

TREATMENT OF DRINKING WATER

Copper can be removed from drinking water by increasing the pH, and then using the water treatment processes of coagulation followed by filtration. Aggressive water, which is likely to induce corrosion of copper pipes, should be stabilised with respect to pH and hardness as part of the treatment process prior to distribution in order to minimise copper leaching.

MEASUREMENT

The copper concentration can be determined by inductively coupled plasma emission spectroscopy (APHA Method 3500-Cu Part C 1992) with an estimated limit of determination of 0.01 mg/L. Alternatively, flame or graphite furnace atomic absorption spectroscopy can be used (APHA 3500-Cu Part B 1992) with limits of determination of 0.05 mg/L and 0.005 mg/L respectively.

HEALTH CONSIDERATIONS

Copper is an essential trace element for humans. It is estimated that adult requirements are about 2–3 mg per person per day. High doses of copper (above 50 mg/kg bodyweight) can be lethal.

The absorption of copper by the gastrointestinal tract is in the range of 25–60%, depending on a number of factors, including copper speciation and copper dietary status (Olivares *et al.* 1998). Copper is stored in the liver, brain and muscle tissue. High concentrations can also be found in the kidneys, heart and hair. Copper is eliminated from the body mainly in the bile.

Many cases of copper poisoning have been reported, including cases involving the poisoning of children who had their food prepared in copper or brass pots (Tanner 1998). Copper poisoning has resulted in cirrhosis of the liver and, in extreme cases, death. Other less severe symptoms associated with the consumption of water containing 3–5 mg/L copper (but not 1 mg/L) are gastrointestinal symptoms such as nausea, abdominal pain and vomiting (Pizarro *et al.* 1999). Infants are thought to be most susceptible, though in one study of 3-month-old infants given water containing 2 mg/L copper over 9 months there were no acute or chronic adverse consequences (Olivares *et al.* 1998). In the genetic disorders Wilson's disease and idiopathic copper toxicosis, sufferers are particularly susceptible to copper (Lönnerdal and Uauy 1998).

Apart from humans, sheep are the most susceptible animals to the toxic effects of copper, with a daily intake of 1–2 mg/kg body weight resulting in serious illness and death.

Copper was not found to be carcinogenic in tests with mice and dogs. The results of mutagenicity tests with different strains of bacteria were generally negative. Tests for mutagenicity using mammalian cells, both *in vitro* and *in vivo*, gave predominantly positive results.

DERIVATION OF GUIDELINE

The health-based guideline value of 2 mg/L (rounded up) for copper in drinking water was derived as follows:

$$2\ \text{mg/L} = \frac{0.5\ \text{mg/kg bodyweight per day} \times 70\ \text{kg} \times 0.1}{2\ \text{L/day}}$$

where:

- 0.5 mg/kg body weight per day is the provisional maximum tolerable daily intake for humans (WHO 1982).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

In this derivation, which others have also used and which is still endorsed by the World Health Organization (2006), there is considerable uncertainty (Fitzgerald 1998). Nevertheless, on the basis of recent copper investigation studies, the derived guideline value appears to be a safe level for infants and is just below a level where minor symptoms were observed in adults.

In premises with a history of copper corrosion, water that has been in stagnant contact (6 hours or more) with copper pipes and fittings should not be used in the preparation of food or drink. Copper levels can be effectively reduced by flushing the taps for 1 minute.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 3500-Cu Part B (1992). Copper: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Cu Part C (1992). Copper: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Fitzgerald DJ (1998). Safety guidelines for copper in water. *American Journal of Clinical Nutrition*, 67(suppl):1098S–1102S.

Lönnerdal B, Uauy R (1998). Genetic and environmental determinants of copper metabolism. *American Journal of Clinical Nutrition*, 67(5S).

Olivares M, Pizarro F, Speisky H, Lönnerdal B, Uauy R (1998). Copper in infant nutrition: Safety of World Health Organization provisional guideline value for copper content of drinking water. *Journal of Pediatric Gastroenterology and Nutrition*, 26:251–257.

Pizarro F, Olivares M, Uauy R, Contreras P, Rebelo A, Gidi V (1999). Acute gastrointestinal effects of graded levels of copper in drinking water. *Environmental Health Perspectives*, 107:117–121.

Tanner MS (1998). Role of copper in Indian childhood cirrhosis. *American Journal of Clinical Nutrition*, 67(suppl):1074S–1081S.

WHO (World Health Organization) (1982). Toxicological evaluation of certain food additives: copper. WHO, Joint Food and Agriculture Organization of the United Nations/WHO Expert Committee on Food Additives, 17, 265–296, Geneva, Switzerland.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Cyanide

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of cyanide in drinking water should not exceed 0.08 mg/L.

GENERAL DESCRIPTION

Cyanide can be present in drinking water through the contamination of source water, or through the natural decomposition of some plants that synthesise cyanoglycosides. Some microorganisms, such as the cyanobacterium *Anacystis nidulans* and the bacterium *Chromobacterium violaceum*, produce free cyanide. In uncontaminated water sources, free cyanide concentrations are usually less than 0.01 mg/L.

Sodium cyanide is used in the extraction of gold and silver from low-grade ores. It is also used in the electroplating, steel and chemical industries.

Some foods can contain quite high concentrations of cyanide. Green almonds and improperly treated cassava are of particular concern.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies cyanide concentrations range up to 0.05 mg/L, with typical concentrations usually less than 0.02 mg/L.

TREATMENT OF DRINKING WATER

There are no published reports on methods for the removal of cyanide from drinking water. Chlorine gas or hypochlorite will react with cyanide to form cyanate. Ozone is also an effective oxidant.

MEASUREMENT

The cyanide concentration in drinking water can be determined with a colorimetric method using chloramine-T (APHA Method 4500-CN Part E 1992). The limit of determination is 0.02 mg/L.

HEALTH CONSIDERATIONS

Cyanide is highly toxic. It is rapidly absorbed by the gastrointestinal tract and metabolised to thiocyanate.

In humans, long-term consumption of improperly prepared cassava in the tropics has been linked with effects on the thyroid gland and particularly the nervous system. Cyanide may deplete vitamin B12 and result in a deficiency that can cause goitre and cretinism. People most at risk are those with a nutritionally inadequate diet.

Animal studies indicate that pigs may be more sensitive than rats to the effects of long-term exposure to cyanide. In a six-month study using pigs, exposure to cyanide was reported to increase ambivalence (sic) and result in slower response times to stimuli. Behaviour demanding high energy appeared to be more readily affected by cyanide exposure than low-energy behaviour.

No data are available on the carcinogenic properties of cyanide. Tests for mutagenicity with different strains of bacteria have been mostly negative.

DERIVATION OF GUIDELINE

The guideline value for cyanide in drinking water was derived as follows:

$$0.08 \text{ mg/L} = \frac{1.2 \text{ mg/kg body weight per day } 70 \text{ kg} \times 0.2}{2 \text{ L/day} \times 100}$$

where:

- 1.2 mg/kg body weight per day is the no-effect level from 6-month feeding studies using pigs Jackson ***et al***. 1986, (Jackson 1988).
- 70 kg is the average weight of an adult.
- 0.2 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in applying the results of animal studies to humans (10 for interspecies variations and 10 for intraspecies variations).

The World Health Organization guideline of 0.07 mg/L was based on an adult weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

APHA Method 4500-CN Part E (1992). Cyanide: Colorimetric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Jackson LC, Chandler JP, Jackson RT (1986). Inhibition and adaptation of red cell glucose-6-phosphate dehydrogenase *in vivo* to chronic sublethal dietary cyanide in an animal model. *Human Biology*, 58:67–77.

Jackson LC (1988). Behavioural effects of chronic sub-lethal dietary cyanide in an animal model: Implications for humans consuming cassava (*Manihot esculenta*). *Human Biology*, 60:597–614.

NOTE: Important general information is contained in PART II, Chapter 6

Cyanogen chloride

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of total cyanogenic compounds in drinking water should not exceed 0.08 mg/L.

GENERAL DESCRIPTION

Cyanogen chloride is a by-product of chloramination. It can be formed as a by-product of the reaction between organic precursors with hypochlorous acid in the presence of the ammonium ion. Concentrations reported overseas in chloraminated supplies are typically 0.004 mg/L.

Cyanogen chloride may be used commercially in chemical synthesis, and for fumigation.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on concentrations of cyanogen chloride in Australian drinking waters.

LIMITING FORMATION IN DRINKING WATER

The presence of cyanogen chloride in drinking water can be minimised by removing naturally occurring organic matter from the source water, by reducing the amount of chloramine added, or by the use of alternative disinfectants.

MEASUREMENT

A suitable method for analysis involves extraction from water using the purge and trap technique followed by gas chromatography/mass spectrometry (USEPA Draft Method 524.2 1986). The limit of determination is 0.0003 mg/L.

HEALTH CONSIDERATIONS

Cyanogen chloride is highly irritant and very poisonous, as it is rapidly metabolised to cyanide in the body and has similar toxicity.

Effects of ingested cyanogen chloride in humans have not been reported. A concentration of 1 ppm in air causes irritation on inhalation.

Only acute toxicity data are available on the health effects of cyanogen chloride in animals.

No data are available on the carcinogenicity or mutagenicity of cyanogen chloride.

The NHMRC Standing Committee on Toxicity reviewed available toxicity data for cyanogen chloride in 1991. It was considered that data were insufficient to set a no-effect level.

DERIVATION OF GUIDELINE

As cyanogen chloride is rapidly converted to cyanide by the body, the guideline value is based on the cyanide value of 0.08 mg/L (see also Fact Sheet on Cyanide).

REFERENCE

USEPA Draft Method 524.2 (1986). Volatile organic compounds in water by purge and trap capillary column gas chromatography/mass spectrometry. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Cyfluthrin, Beta-cyfluthrin

(endorsed 2011)

GUIDELINE

Based on human health concerns, cyfluthrin or beta-cyfluthrin in drinking water should not exceed 0.05 mg/L.

RELATED CHEMICALS

Cyfluthrin and beta-cyfluthrin (CAS 68359-37-5) are in the pyrethroid class of chemicals. Cyfluthrin is a mixture of 8 isomers, comprising 4 diastereoisomeric pairs. Beta-cyfluthrin contains the two active diastereoisomers. Other pesticides in this class include cypermethrin, alpha-cypermethrin, deltamethrin and permethrin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, cyfluthrin or beta-cyfluthrin would not be a health concern unless the concentration exceeded 0.05 mg/L. Minor excursions above this level even for a short period are of concern, as the health-based guideline is based on short- to medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Cyfluthrin and beta-cyfluthrin are broad-spectrum insecticides used for the control of spiders, ants, fleas, flies, silverfish, cockroaches, bedbugs and mosquitoes.

There are registered products containing and registered products containing beta-cyfluthrin in Australia. The majority of those containing cyfluthrinvare household insecticide sprays, of both the knock-down and surface spray varieties. Some products are also used to impregnate mosquito nets. Cyfluthrin is not applied to crops. Products containing beta-cyfluthrin are used on a variety of fruits, vegetables, cereals and pastures. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure are the use of household insecticide sprays, and residues in food. Residue levels in crops grown according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of sources waters through processes such as run-off, spray drift, or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of cyfluthrin or beta-cyfluthrin in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No reports of the treatment of cyfluthrin or beta-cyfluthrin in drinking water have been identified.

MEASUREMENT

Several methods have been reported for the analysis of cyfluthrin and beta-cyfluthrin in water, including gas chromatography with micro-electron capture detection (limit of detection [LOD] 0.85 ng/L, Casas *et al.* 2006; LOD 2 ng/L, Mekebri *et al.* 2008) and gas chromatography with high resolution mass spectrometry (LOD 0.10 ng/L, Woudneh and Oras 2006).

HISTORY OF THE HEALTH VALUES

For cyfluthrin, the current acceptable daily intake (ADI) is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.5 mg/kg bw/day from a 2-year dietary study in rats. The NOEL is based on decreased bodyweight gain and a small increase in fluorine content of bones. The ADI incorporates a safety factor of 100 and was established in 1985.

For beta-cyfluthrin, the current ADI is 0.01 mg/kg bw, based on a NOEL of 1.5 mg/kg bw/day from a 13-week dietary study in dogs. The NOEL is based on vomiting, diarrhoea and effects on motor function. The ADI incorporates a safety factor of 100 and was established in 1990.

An *Australian Drinking Water Guidelines* value has not previously been established for cyfluthrin or beta-cyfluthrin.

HEALTH CONSIDERATIONS

Metabolism: Absorption of cyfluthrin and beta-cyfluthrin via the gastrointestinal tract is rapid and extensive (86-100%). Metabolism occurs via ester hydrolysis, oxidation of the alcohol moiety, hydroxylation and conjugation. The metabolites are excreted in urine (66%) and faeces (33%).

Acute effects: Cyfluthrin has low to moderate acute oral toxicity and low acute dermal toxicity. It does not cause skin sensitisation. Experience in the use of cyfluthrin has shown that skin irritation and peripheral sensory nerve excitation can result from human exposure, but these effects are reversible.

Beta-cyfluthrin has moderate acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: In a 28-day dietary study in rats, cyfluthrin significantly decreased weight gain, elevated serum alanine amino transferase levels, increased absolute and relative liver and adrenal weights, and caused increased mortality at 80 mg/kg bw/day.

Medium-term dietary studies with cyfluthrin were conducted in rats and dogs. In the 3-month rat study, no toxicity was observed up to the highest dose of 25 mg/kg bw/day. A 6-month dog study reported decreased weight gain at 6.2 mg/kg bw/day, and vomiting, diarrhoea, trembling and poor motor co-ordination at 18.5 mg/kg bw/day.

A 28-day dietary study in rats with beta-cyfluthrin reported behavioural changes and increased mortality at 4 mg/kg bw/day and above, and salivation, apathy, changes in gait, respiratory distress and body rolling at 16 mg/kg bw/day.

Medium-term dietary studies were conducted in rats and dogs with beta-cyfluthrin. In a 90-day rat study, head and neck necrosis, uncoordinated gait, poor general condition, reduced bodyweight gain, and decreased cholesterol and triglyceride levels were observed at 50 mg/kg bw/day. A 13-week study in dogs reported toxic effects on motor function, vomiting and diarrhoea at 9 mg/kg bw/day. The NOEL of 1.5 mg/kg bw/day in this dog study is the basis for the current ADI for beta-cyfluthrin.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: Long-term studies with cyfluthrin in mice, rats and dogs produced decreased bodyweight gain at dose levels of 220 mg/kg bw/day, 6 mg/kg bw/day and 24 mg/kg bw/day, respectively. A 2-year rat study reported a small increase in fluoride concentration in bones at 6 mg/kg bw/day. In a 12-month dietary study in dogs, clinical signs of toxicity were observed at a dose of 24 mg/kg bw/day. The NOEL of 2.5 mg/kg bw/day in the rat study is the basis for the current ADI for cyfluthrin.

Carcinogenicity: Based on long-term studies in rodents, there is no evidence of carcinogenicity for cyfluthrin.

Genotoxicity: Cyfluthrin and beta-cyfluthrin are not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats with cyfluthrin showed evidence of reduced fertility and pup survival at dose levels of 6 mg/kg bw/day and above. A developmental toxicity study in rats did not produce any evidence of effects on foetal development.

Neurotoxicity: There was no evidence of delayed neurotoxicity with cyfluthrin in studies in hens.

Poisons Schedule: Cyfluthrin is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Beta-cyfluthrin is included in Schedules 5, 6 and 7 , depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.05 mg/L for beta-cyfluthrin was determined as follows:

$$0.05 \text{ mg/L} = \frac{1.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.5 mg/kg bw/day is a NOEL based on a medium-term (13-week) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the average maximum amount of water consumed by an adult
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Casas V, Llompart M, Garcia-Jares C, Cela R, Dagnac T (2006). Multivariate optimisation of the factors influencing the solid-phase microextraction of pyrethroid pesticides in water. *Journal of Chromatography A*, 1124:148-156.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NOTE: Important general information is contained in PART II, Chapter 6

Mekebri A, Crane DB, Blondina GJ, Oros DR, Rocca JL (2008). Extraction and analysis methods for the determination of pyrethroid insecticides in surface water, sediments and biological tissues at environmentally relevant concentrations. *Bulletin of Environmnetal Contamination and Toxicology*, 80:455-460.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Woudneh MB, Oros DR (2008). Quantitative determination of pyrethroids, pyrethrins and piperonyl butoxide in surface water by high-resolution gas chromatography/high resolution mass spectrometry. *Journal of Agricultural and Food Chemistry*, 54(19):6957-6962.

NOTE: Important general information is contained in PART II, Chapter 6

Cypermethrin isomers

(endorsed 2011)

GUIDELINE

Based on human health concerns, cypermethrin in drinking water should not exceed 0.2 mg/L.

RELATED CHEMICALS

Cypermethrin (CAS 52315-07-8) belongs to the pyrethroid class of chemicals. Cypermethrin is a mixture of 8 isomers, comprising 4 diastereoisomeric pairs. Alpha-, beta- or zeta-cypermethrin each contain different amounts of the isomers. There are many other pesticides in this class including allethrin, bifenthrin, deltamethrin, permethrin, phenothrin and tetramethrin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, cypermethrin would not be a health concern unless the concentration exceeded 0.2 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on medium- to long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Cypermethrin is an insecticide used to control a broad range of insect and rodent pests in domestic, commercial and industrial areas, and in crops. It is also used as an ectoparasiticide in cattle and domestic pets, and as a fungicide in crop seeds.

There are many registered products containing cypermethrin in Australia; some contain alpha-cypermethrin, and a small number contain beta-cyperemethrin or zeta-cypermethrin. Cypermethrin products are intended for professional and home garden use, and include insecticide dusts, sprays and bait, rodent pellets, sheep drenches, cattle ear tags and pet shampoos. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to cypermethrin are the use of household products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of cypermethrin may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

The veterinary use of cypermethrin provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of cypermethrin in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No reports of the treatment of cypermethrin in drinking water have been identified.

MEASUREMENT

Several methods have been reported for the analysis of cypermethrin in water, including gas chromatography with micro-electron capture detection (limit of detection [LOD] 1 ng/L, Casas *et al*. 2006; LOD 2 ng/L, Mekebri *et al*, 2008) and gas chromatography with high resolution mass spectrometry (LOD 0.1 ng/L, Woudneh and Oras 2006).

HISTORY OF THE HEALTH VALUES

The acceptable daily intake (ADI) for cypermethrin, alpha-cypermethrin, and beta-cypermethrin is 0.05 mg per kg of bodyweight (mg/kg bw). For cypermethrin, the ADI is based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day from a 2-year rat study. This NOEL is based on increased liver weights, and haematological and biochemical effects. For alpha-cypermethrin, the ADI is based on a NOEL of 4.7 mg/kg bw/day from a 13-week dog study. This NOEL is based on neurological effects. The ADIs for both cypermethrin and alpha-cypermethrin incorporate a safety factor of 100 and were established in 1988 and 1994, respectively.

For beta-cypermethrin, the 2-year rat study used for establishing the cypermethrin ADI was considered appropriate to use for the beta-cypermethrin ADI.

For zeta-cypermethrin, the ADI is 0.07 mg/kg bw, based on a NOEL of 7 mg/kg bw/day from a multigeneration reproduction study in rats. The NOEL is based on clinical signs of toxicity and evidence of neurotoxicity. The ADI incorporates a safety factor of 100 and was established in 1996.

An acute reference dose (ARfD) of 0.05 mg/kg bw/day for beta-cypermethrin was established in 2002, based on a NOEL of 4.7 mg/kg bw/day from a 3-month dog study. The NOEL was based on neurological effects. The ARfD incorporates a safety factor of 100.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Cypermethrin and alpha-cypermethrin are both well absorbed via the gastrointestinal tract and readily metabolised in rats and humans. Excretion is rapid and mainly via the urine. In humans, cyclopropane carboxylic acid is the major urinary metabolite.

Acute effects: Cypermethrin has moderate acute oral toxicity and low acute dermal toxicity. There is some evidence of skin sensitisation. Alpha-cypermethrin has moderate to high acute oral toxicity. Beta-cypermethrin has low acute oral and dermal toxicity. Zeta-cypermethrin has moderate acute oral toxicity and low dermal toxicity.

Short-term effects: In 13-week dietary studies in rats and dogs with cypermethrin, there was nervous system toxicity in both species at doses above 7.5 mg/kg bw/day. Changes indicative of kidney and liver toxicity were also observed in rats at higher dose levels.

In 5- and 13-week dietary studies in rats and dogs with alpha-cypermethrin, there were clinical signs indicative of neurotoxicity (hypersensitivity to noise and abnormal gait) as well as increased organ weights for brain, liver and kidney at 21.9 mg/kg bw/day and above in rats and at 14 mg/kg bw/day in dogs. The NOEL of 4.7 mg/kg bw/day in this dog study is the basis for the current alpha-cypermethrin ADI.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: Long-term (2-year) studies in mice, rats and dogs with cypermethrin reported increased organ weights and haematological and biochemical changes in all species. The rat was the most sensitive species, with effects observed at 50 mg/kg bw/day and above. The NOEL of 5 mg/kg bw/day in this rat study is the basis for the current cypermethrin ADI.

There were no long-term studies conducted on alpha-cypermethrin or other isomers; however, the data for cypermethrin are considered representative of other isomers in relation to potential long-term effects.

Carcinogenicity: On the basis of long-term studies in mice and rats, there is no evidence of carcinogenicity for cypermethrin.

Genotoxicity: Cypermethrin and alpha-cypermethrin are not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A multigenerational reproduction study in rats and developmental studies in rats and rabbits with cypermethrin reported no evidence of effects on reproductive parameters or on foetal development. There are no reproduction or developmental studies available for the other cypermethrin isomers; however, the data for cypermethrin are considered representative of other isomers for these endpoints.

Neurotoxicity/immunotoxicity: Studies in rats and hamsters with cypermethrin reported no evidence of delayed neurotoxicity or toxicity to the immune system.

Poisons Schedule: Cypermethrin and alpha-, beta- and zeta-cypermethrin are in Schedules 5, 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard) (DoHA 2010), depending on the concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.2 mg/L for cypermethrin was determined as follows:

$$0.2 \text{ mg/L} = \frac{5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 5 mg/kg bw/day is the NOEL for cypermethrin and alpha-cypermethrin based on a long-term (2-year) study in rats and a medium-term (13-week) study in dogs, respectively. This NOEL is considered to be protective for other cypermethrin isomers.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has included cypermethrin in the list of chemicals from agricultural activities excluded from guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Casas V, Llompart M, Garcia-Jares C, Cela R, Dagnac T (2006). Multivariate optimisation of the factors influencing the solid-phase microextraction of pyrethroid pesticides in water, *Journal of Chromatography A*, 1124, 148-156.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Mekebri A, Crane DB, Blondina GJ, Oros DR, Rocca JL (2008) Extraction and analysis methods for the determination of pyrethroid insecticides in surface water, sediments and biological tissues at environmentally relevant concentrations, *Bulletin of Environmnetal Contamination and Toxicology*, 80, 455-460.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

Woudneh MB, Oros DR (2008). Quantitative determination of pyrethroids, pyrethrins and piperonyl butoxide in surface water by high-resolution gas chromatography/high resolution mass spectrometry. *Journal of Agricultural and Food Chemistry*, 54(19):6957-6962.

NOTE: Important general information is contained in PART II, Chapter 6

Cyprodinil

(endorsed 2011)

GUIDELINE

Based on human health concerns, cyprodinil in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Cyprodinil (CAS 121552-61-2) belongs to the anilinopyrimidine class of chemicals. Another pesticide in this class is pyrimethanil (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, cyprodinil would not be a health concern unless the concentration exceeded 0.09 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Cyprodinil is a fungicide for the control of black spot in apples and pears; the control of blossom blight and brown rot in apricots, peaches, plums and nectarines; and, in combination with fludioxonil, the control of grey mould in grapes.

There are registered products that contain cyprodinil in Australia. These are intended for professional use and are available as water-dispersible granules to be applied as concentrated or dilute solutions, commonly using air blast, misters or hand-held equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to cyprodinil is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of cyprodinil may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for cyprodinil in Australian waters were found.

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficiency were found for cyprodinil.

MEASUREMENT

Cyprodinil in water can be analysed by adapting a solid-phase microextraction gas chromatrography mass spectrometry method developed for white wine (Otero *et al.* 2002), with a limit of detection of 0.2 µg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for cyprodinil is 0.02 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.7 mg/kg bw/day from a long-term (2-year) dietary study in rats. The NOEL is based on an increased incidence of liver lesions in males at the next highest dose of 36 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was established in 1994.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Cyprodinil is rapidly absorbed via the gastrointestinal tract in rats. The parent compound is completely metabolised and excreted mainly as conjugated derivatives. These are excreted mainly in the urine within 48 hours.

Acute effects: Cyprodinil has low acute oral and dermal toxicity. It is a skin sensitiser in guinea pigs.

Short-term effects: In 1- to 3-month studies in mice, rats and dogs, the liver was the main target of toxicity, although most effects occurred only at very high dose levels. In mice, liver necrosis was observed at 257 mg/kg bw/day. In dogs, increased liver weights were observed at 46 mg/kg bw/day. Rats showed increased plasma cholesterol and phospholipid levels at 3 mg/kg bw/day, and effects on the thyroid and kidney at 19 mg/kg bw/day.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs showed the liver to be the main target organ of toxicity. Effects on the liver were noted at 600 mg/kg bw/day in mice (increased relative liver weight), at 36 mg/kg bw/day in male rats (spongiosis hepatitis) and at 446 mg/kg bw/day in dogs (pigmentation of hepatocytes). The lowest NOEL was 2.7 mg/kg bw/day in the rat study and this is the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for cyprodinil.

Genotoxicity: Cyprodinil is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats did not produce any evidence of reproductive effects. Developmental studies in rats and rabbits showed effects on foetal development in rats only, at dose levels above the maternotoxic dose, which are well in excess of the likely level of human exposure.

Poisons Schedule: Cyprodinil is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.09 mg/L for cyprodinil was determined as follows:

$$0.09 \text{ mg/L} = \frac{2.7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 2.7 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Otero RR, Ruiz CY, Grande BC, Gandara JS (2002). Solid-phase microextraction-gas chromatographic-mass spectrometric method for the determination of the fungicides cyprodinil and fludioxonil in white wines. *Journal of Chromatography A*, 942(1-2):41-52.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

2,4-D [(2,4-Dichlorophenoxy) acetic acid]

(endorsed 2011)

GUIDELINE

Based on human health concerns, 2,4-D in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

2,4-D (CAS 94-75-7) belongs to the phenoxycarboxylic class of chemicals. Other pesticides in this class include dicloprop-p, MCPA, and mecoprop-p (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, 2,4-D would not be a health concern unless the concentration exceeded 0.03 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking-water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: 2,4-D is a systemic herbicide used for the control of broad-leaf and aquatic weeds.

There are registered products containing 2,4-D in its various forms (free acid, alkali and amine salts and esters) in Australia. These products are intended for professional and home garden use and are used in food crops, in forestry, on turf and on non-crop land including industrial/commercial areas. They may be applied to these uses by a boom spray and aircraft. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to 2,4-D is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The agricultural use of 2,4-D can involve direct application into waterways or sewage systems, which may then enter source waters for drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Reported concentrations of 2,4-D in Queensland potable water supplies range from 0.2 to 0.0004 mg/L (0.4 µg/L) (Mitchell *et al.* 2005), and in Victoria range from 0.00002 to 0.34 mg/L (0.02 to 34 µg/L) (Amis 2008). Other river systems for which data exist have reported concentrations of up to 0.0156 mg/L (15.6 µg/L) (Hunter 2001).

NOTE: Important general information is contained in PART II, Chapter 6

TREATMENT OF DRINKING WATER

Common water treatment processes are not effective in removing 2,4-D from water. Activated carbon adsorption, either powdered or granulated, is the method of choice for removing 2,4-D from drinking water supplies (Health Canada 1991). Powdered activated carbon removed 90% of an initial dose of 1.0 mg/L (Canadian Department of National Health and Welfare 1993). Laboratory tests or expert advice should be sought to ensure that an effective activated carbon is selected for use. Membrane filtration treatments, ultraviolet (UV) irradiation, ozone, and the combination of UV and peroxide are also effective in removing 2,4-D (Benitez *et al*. 2004).

MEASUREMENT

Residues of 2,4-D and its salts and esters in water are commonly measured by solid-phase extraction, chemical derivatisation, separation by liquid or gas chromatography and electron capture detection (WHO 2004). Analytical detection limits for this approach range from 0.00005 to 0.0005 mg/L (0.05 to 0.5 µg/L). High performance liquid chromatography with ultraviolet detector is also commonly used and can achieve a limit of detection (LOD) of 0.001 mg/L (1 µg/L). High performance liquid chromatography–mass spectrometry can reach a lower LOD (0.00001 mg/L [0.01 µg/L]). Measurement of 2,4-D is also described in EPA methods 515.1, 555.2, 555.3, 55.4, and 555 and ASTM International methods D5317-93 and D5317-98 (USEPA 2008). Enzyme-linked immunosorbent assay (ELISA) has been also used for the quantification of 2,4-D and its salts and typical reported LODs from immunoassays are 0.0001 to 0.0007 mg/L (0.1 to 0.7 µg/L) (USEPA 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for 2,4-D is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observation-effect level (NOEL) of 1 mg/kg bw/day from a long-term (2-year) rat study. The NOEL is based on effects on the kidney. The ADI incorporates a safety factor of 100 and it was initially established in 1989 and re-confirmed in 2006.

An acute reference dose (ARfD) of 0.8 mg/kg bw/day was established for 2,4-D in 2006 based on a NOEL of 75 mg/kg bw/day derived from an acute exposure study in rats which reported effects on the nervous system. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC & NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Both the free acid and the salt form of 2,4-D are rapidly absorbed from the gastrointestinal tract and distributed to tissues, with highest concentrations in kidney, liver and brain. There is no evidence for accumulation in fat. More than 70% of the oral dose was recovered in urine in animals and humans.

Acute effects: 2,4-D has low acute oral and dermal toxicity, and is not a skin sensitizer.

Short-term effects: Three-month dietary studies in mice, rats and dogs reported effects on the kidney at dose levels of 45 mg/kg bw/day, 5 mg/kg bw/day and 3 mg/kg bw/day, respectively.

Long-term effects: Long-term dietary studies in rats reported abnormal kidney histopathology at doses of 5 mg/kg bw/day and above. Effects on the rat thyroid were also noted at 45 mg/kg bw/day. The NOEL of 1 mg/kg bw/day is the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for 2,4-D.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: 2,4-D is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a 2-generation reproduction study in rats, decreased offspring viability and malformations were seen only at maternotoxic doses. Developmental studies in rats and rabbits did not produce any evidence of effects on foetal development.

Neurotoxicity: In a 15-day dietary study, 2,4-D induced neurological effects that included changes in gait, coordination and decreased motor activity at doses of 250 mg/kg bw/day. The NOEL was 75 mg/kg bw/day and was the basis for the ARfD. These effects were not noted in other acute and/or repeat-dose studies in rats and dogs.

Poisons Schedule: 2,4-D is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010)., depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for 2,4-D was determined as follows:

$$0.03 \text{ mg/L} = \frac{1.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.0 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

This guideline value applies to 2,4-D as well as salts and esters of 2,4-D, as these are rapidly hydrolysed to the free acid in water.

The World Health Organization has established a health-based guideline value of 0.03 mg/L for 2,4-D in 1993 (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Amis A (2008). *Survey of Pesticides in Surface Waters of Domestic Water Supplies – Victorian Water Authorities 1998–2007*. Melbourne, Friends of the Earth Melbourne and National Toxics Network.

Benitez FJ, Acero JL, Real FJ, Roman S (2004). Oxidation of MCPA and 2,4-D by UV radiation, ozone, and the combinations UV/H_2O_2 and O_3/H_2O_2. *Journal of Environmental Science and Health, Part B*, 39(3):393-409.

Canadian Department of National Health and Welfare (1993). Water treatment principles and applications: a manual for the production of drinking water. Canadian Water and Wastewater Association, Ottawa.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NOTE: Important general information is contained in PART II, Chapter 6

Health Canada (1991). Guidelines for Canadian Drinking Water Quality – Technical Documents: 2,4-Dichlorophenoxyacetic Acid. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/dichlorophenoxyacetic_acid/]

Hunter H, Sologinkin S, Choy S, Hooper A, Allen W, Raymond M, Peeters J (2001). *Water management in the Johnstone Basin*. Queensland Department of Natural Resources and Mines, Brisbane.

Mitchell C, Brodie J, White I (2005). Sediments, nutrients and pesticide residues in event flow conditions in streams of the Mackay Whitsunday Region, Australia. *Marine Pollution Bulletin*, 51(1-4):23-36.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (2008). Analytical Methods Approved for Drinking Water Compliance Monitoring of Organic Contaminants. http://www.epa.gov/safewater/methods/pdfs/methods/organic_080521b.pdf

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

DDT (1,1,1-trichloro-di-(4-chlorophenyl) ethane)

(endorsed 2011)

GUIDELINE

Based on human health concerns, DDT in drinking water should not exceed 0.009 mg/L.

RELATED CHEMICALS

DDT (CAS 50-29-3(p.p')/CAS 789-02-6 (o.p')) belongs to the organochlorine class of chemicals and is classified as a persistant organic pollutant (POP). Other POPs that were previously used as pesticides include aldrin, dieldrin and heptachlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present as a contaminant in drinking water, DDT would not be a health concern unless the concentration exceeded 0.009 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: DDT is a contact insecticide used in some parts of the world for the control of insects, including those carrying diseases that are infectious to humans such as malaria.

There are no registered products that contain DDT in Australia, but de-registered compounds may still be detected in water.

Exposure sources: DDT persists in the environment as a result of previous use as an insecticide. The general public may be exposed to low levels of DDT through residues in food and/or contaminated source waters.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Since banning in the 1980s, DDT has not been commonly reported in Australian drinking waters. Evidence suggests that the concentrations of DDT in Australian rivers have progressively declined, as indicated by water in the Brisbane River, which consistently fell from maximum concentrations of about 1.7 µg/L in 1972-1973 to not detectable in 1986-1987 (Connell *et al.* 2001).

TREATMENT OF DRINKING WATER

DDT can be effectively removed from drinking waters by activated carbon treatment (Thacker *et al.* 1997, Ormad *et al.* 2008). Flocculation-coagulation can also be effective under suitable conditions (Ormad *et al.* 2008).

MEASUREMENT

DDT can be measured in drinking water using gas chromatography with electron caption detection (WHO 2004). The limit of detection for this technique is 0.01 µg/L.

HISTORY OF THE HEALTH VALUES

The current tolerable daily intake (TDI) for DDT is 0.002 mg per kg bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day from an epidemiological study. The NOEL is based on the absence of toxicological effects at this dose. The TDI incorporates a safety factor of 100, comprising 10 for intraspecies variation and 10 to take into account the uncertainty due to lack of detail in the study. The TDI was established in 2003.

When DDT was used previously, the ADI was 0.02 mg/kg bw based on a NOEL of 0.25 mg/kg bw/day from the same epidemiological study. The ADI was not maintained, as DDT is no longer used in agricultural practice.

The previous ADWG health value was 0.02 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: DDT is absorbed moderately well via the gastrointestinal tract in rats, and absorption is enhanced in the presence of fat. DDT and its metabolite DDE accumulate and are stored in adipose tissue and in organs containing large amounts of fat. DDT is also able to cross the placental barrier in rats and rabbits and accumulate in the foetus. It is excreted mainly unchanged in the faeces, with some excretion of the metabolite DDE occurring in urine and bile. DDT and its metabolites are also excreted in the milk of lactating animals. DDT residues have been found in human breastmilk.

Acute effects: DDT has moderate acute oral toxicity in mice, rats, guinea pigs and rabbits. Data are not available on its potential dermal toxicity or skin sensitisation.

Short-term effects: A short-term oral study in rabbits reported liver and kidney damage, and increased mortality, at 50 mg/kg bw/day. A 1-year oral study in monkeys reported hyperglycaemia, liver enlargement and hepatitis at 0.2 mg/kg bw/day. A 21-month oral study in human volunteers at 0.5 mg/kg bw/day of DDT did not produce any obvious signs of toxicity.

Long-term effects: Long-term oral studies were conducted in mice, rats, guinea pigs, dogs and monkeys. The liver was the main target organ of toxicity, with mice and rats showing particular sensitivity. In mice, there were increased benign and malignant liver tumours at dose levels above 3 mg/kg bw/day. In rats, there was hypertrophy, formation of lipospheres and cell proliferation at 0.25 mg/kg bw/day, and benign hepatocellular tumours at 12.5 mg/kg bw/day. In dogs, jaundice and haemorrhagic symptoms were reported at 50 mg/kg bw/day. In monkeys, loss of appetite, decreased bodyweight gain and convulsions were reported at 55 mg/kg bw/day.

In an epidemiological study of workers exposed to 0.25 mg/kg bw/day for 25 years, there was no association between adverse health outcomes and exposure to DDT. The NOEL of 0.25 mg/kg bw/day from this study is the basis for the current TDI. Two other epidemiological studies reported an association between the incidence of liver and pancreatic cancer and exposure to DDT, however, there were significant confounding factors in these studies.

Carcinogenicity: Long-term dietary studies in mice and rats reported evidence of liver cancer following exposure to DDT. In lifetime studies in hamsters and monkeys, there was no evidence of cancer from exposure to DDT. Epidemiological studies in humans produced only limited evidence of liver and pancreatic cancers following exposure to DDT.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: DDT is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2- and 3-generation reproduction study in rats and dogs respectively did not produce evidence of adverse effects on reproductive parameters at 10 mg/kg bw. Developmental studies in mice and rabbits have produced conflicting results but the weight of evidence indicates no effects on foetal development, except at high dose levels, well in excess of the likely level of human exposure.

Poisons Schedule: DDT is included in Appendix C of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). This appendix is for 'Substances, other than those included in schedule 9, of such danger to health as to warrant prohibition of sale, supply and use'. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.009 mg/L for DDT was determined as follows:

$$0.009 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.25 mg/kg bw/day is the NOEL based on a 25-year study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the
- TDI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for intraspecies variation and 10 for the uncertainty arising from the lack of detail in the epidemiological study.

The World Health Organization has a health-based guideline value of 0.001 mg/L for DDT (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Connell DW, Miller G, Anderson S (2001). Chlorohydrocarbon pesticides in the Australian marine environment after banning in the period from the 1970s to 1980s. In: *3rd International Conference on Marine Pollution and Ecotoxicology,* Pergamon-Elsevier Science Ltd, Hong Kong, Peoples Republic of China, pp 78-83.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71(1):97-106.

NOTE: Important general information is contained in PART II, Chapter 6

Thacker NP, Vaidya MV, Sipani M, Kalra A (1997). Removal technology for pesticide contaminants in potable water. *Journal of Environmental Science and Health Part B – Pesticides, Food Contaminants and Agricultural Wastes,* 32(4):483-496.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Deltamethrin

(endorsed 2011)

GUIDELINE

Based on human health concerns, deltamethrin in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Deltamethrin (CAS 52918-63-5) is in the pyrethroid class of chemicals. Other pesticides in this class include cyfluthrin, cypermethrin, alpha-cypermethrin and permethrin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, deltamethrin would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Deltamethrin is an insecticide and parasiticide used for the control of a range of insects in various situations.

There are 70 products containing deltamethrin. They are intended for both professional and home garden use. For professional use, deltamethrin may be applied either as a pour-on, or as a spray, using ground or aerial methods of application. For home garden use, deltamethrin is generally available as pre-prepared spray or aerosol.

Exposure sources: The main source of public exposure to deltamethrin is residues in food. Residues levels in food produced according to good agricultural practice are generally low.

Agricultural use of deltamethrin may potentially lead to the contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on the concentrations of deltamethrin in Australian drinking waters or in drinking water overseas.

TREATMENT OF DRINKING WATER

There are no reports of the treatment of deltamethrin in drinking water.

MEASUREMENT

Several methods have been reported for the analysis of deltamethrin in water including liquid chromatography with electrospray ionisation mass spectrometry, with a limit of detection (LOD) of 0.2 ng/L in groundwater and 0.3 ng/L in sea water (Gil-Garcia *et al*. 2006); gas chromatography with micro-electron capture detection, LOD 0.81 ng/L (Casas *et al*. 2006); and gas chromatography-high resolution mass spectrometry, LOD 0.74 ng/L (Woudneh and Oras 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for deltamethrin is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a long-term dog study. This NOEL is based on clinical effects, including dilation of the pupils, decreased weight gain, vomiting and diarrhoea. The ADI incorporates a safety factor of 100 and was established in 1980.

An *Australian Drinking Water Guidelines* health value has not previously been established.

HEALTH CONSIDERATIONS

Metabolism: Deltamethrin is rapidly absorbed via the gastrointestinal tract. It is extensively metabolised and excreted. The principal routes of metabolism are ester cleavage and oxidation at the 4-position of the alcohol moiety.

Acute effects: Deltamethrin has moderate to high acute oral toxicity and low acute dermal toxicity. It is not a skin sensitiser. Production and agricultural workers have reported irritation to the skin and mucous membranes, which last for several days.

Short-term effects: Medium-term dietary exposure in rats and dogs resulted in decreased bodyweight gain at the highest doses tested, namely, 2.5 mg/kg bw/day and 1 mg/kg bw/day, respectively. This was not accompanied by any pathological changes.

Long-term effects: Long-term dietary studies in mice, rat and dogs reported no significant toxic effects at dose levels of 15, 2.1 and 1.0 mg/kg bw/day, respectively.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for deltamethrin.

Genotoxicity: Deltamethrin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a multigenerational reproduction study in rats and in developmental studies in mice, rats and rabbits, there was no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Deltamethrin is included in Schedule 5, 6 or 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for deltamethrin was determined as follows:

$$0.04 \text{ mg/L} = \frac{1.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.0 mg/kg bw/day is the NOEL based on a long-term (2-year) toxicity dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

Deltamethrin is excluded from the World Health Organization drinking water guidelines because it is "unlikely to occur in drinking water" (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Casas V, Llompart M, Garcia-Jares C, Cela R, Dagnac T (2006). Multivariate optimisation of the factors influencing the solid-phase microextraction of pyrethroid pesticides in water. *Journal of Chromatography A*, 1124:148-156.

DoHA (2010) T***he Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Gil-Garcia MD, Barranco-Martinez D, Martinez-Galera M, Parrilla-Vazquez P (2006). Simple, rapid solid-phase extraction procedure for the determination of ultra-trace levels of pyrethroids in ground and sea water by liquid chromatography/electrospray ionisation mass spectrometry. *Rapid Communications in Mass Spectrometry*, 20:2395-2403.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, First Addendum, WHO, Geneva, Switzerland.

Woudneh MB, Oros DR (2006). Quantitative determination of pyrethroids, pyrethrins and piperonyl butoxide in surface water by high-resolution gas chromatography/high resolution mass spectrometry. *Journal of Agricultural and Food Chemistry*, 54(19):6957-6962.

NOTE: Important general information is contained in PART II, Chapter 6

Diazinon

(endorsed 2011)

GUIDELINE

Based on human health concerns, diazinon in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Diazinon (CAS 333-41-5) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including chlorpyrifos, malathion, and temephos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, diazinon would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Diazinon is a insecticide used on sheep and cattle and companion animals; on fruit, vegetables, mushrooms and field crops; and in horticulture for nursery plants and ornamentals, including quarantine use. It is also used on lawns and turf, and in commercial, public and domestic buildings and surrounds.

There are registered products containing diazinon in Australia. These are intended for professional use and are available as soluble concentrates to be diluted and applied by ground and aerial sprays to crops, or as impregnated flea collars (pets) and ear tags (cattle). Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to diazinon and its metabolites are its use in public or domestic buildings, in the garden, or on companion animals; and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural and veterinary use of diazinon may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of diazinon in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Diazinon can be readily removed by various oxidation processes including ultraviolet radiation, oxidation, advanced oxidation and ozone (Real *et al.* 2007). However, oxidation can result in the formation of unwanted by-products, therefore a by-product management plan is also recommended before implementing any oxidation processes.

Activated carbon is effective in the removal of diazinon (Ohno *et al.* 2008). However, in the presence of chlorine, unwanted diazinon by-products were shown to desorb from the activated carbon surface (Ohno *et al.* 2008).

Nanofiltration has also been shown to be effective in the removal of Diazinon. (Kiso *et al.* 2000).

MEASUREMENT

The practical limit of quantification for diazinon in water is 0.0001 µg/L by liquid chromatography–tandem mass spectrometry (Alder *et al.* 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for diazinon is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observable-effect level (NOEL) of 0.02 mg/kg bw/day from a short-term (37-43 days) human study. The NOEL was based on inhibition of plasma cholinesterease. The ADI incorporates a safety factor of 20 and was established in 1999.

The acute reference dose (ARfD) of 0.01 mg/kg bw/day for diazinon was established in 2002, based on a NOEL of 0.2 mg/kg bw/day from a human study. The NOEL was based on red blood cell cholinesterase inhibition after a single dose of diazinon. The ARfD incorporates a safety factor of 20.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Diazinon is rapidly absorbed from the gastrointestinal tract, extensively metabolised and rapidly excreted mainly in urine (97%) within 24 hours. The highest tissue concentrations are in fat, with no evidence of accumulation.

Acute effects: Diazinon has moderate acute oral and dermal toxicity, and is a skin sensitiser in guinea pigs. It can also cause delayed neuropathy 24-96 hours after acute exposure. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: Short-term dietary studies in rats reported inhibition of plasma cholinesterase at doses of 0.2 mg/kg bw/day. Inhibition of erythrocyte cholinesterase activity was seen at 2.5 mg/kg bw/day, while brain cholinesterase activity was inhibited at 23 mg/kg bw/day. In a 37-43 day dietary study in humans, the NOEL for cholinesterase inhibition was 0.02 mg/kg/ bw/day, and this is the basis for the ADI.

Long-term effects: In long-term dietary studies in rats, mice and dogs, plasma cholinesterase inhibition was seen at doses of 0.018 mg/kg bw/day and above (dogs). Decreased brain cholinesterase activity (rats) and decreased bodyweight gain (dogs) was seen at 5 mg/kg bw/day. Clinical symptoms of nervous system toxicity were seen at 15 mg/kg bw/day (mice). The overall lowest NOEL was 0.0037 mg/kg bw/day (dogs) in these studies.

NOTE: Important general information is contained in PART II, Chapter 6

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for diazinon.

Genotoxicity: Diazinon is not considered to be genotoxic, based on *in vitro* and *in vivo* short term studies.

Reproductive and developmental effects: Reproduction studies in rats and developmental studies in rats and rabbits produced no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Diazinon is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010)., depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.004 mg/L for diazinon was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.02 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 20}$$

where:

- 0.02 mg/kg bw/day is the NOEL based on a short-term (37-43 day) repeat-dose study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 20 is the safety factor applied to the NOEL from a study conducted in humans. The safety factor of 20 incorporates a factor of 10 for intraspecies variation and 2 for the closeness of the NOEL and LOEL.

The World Health Organization has not established a health-based guideline value for diazinon and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Alder L, Greulich K, Kempe G, Vieth B (2006). Residue analysis of 500 high priority pesticides: better by GC-MS or LC-MS/MS? *Mass Spectrometry Reviews*, 25(6):838-65.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Kiso Y, Nishimura Y, Kitao T, Nishimura K (2000). Rejection properties of non-phenylic pesticides with nanofiltration membranes. *Journal of Membrane Science*, 171, 229-237.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NOTE: Important general information is contained in PART II, Chapter 6

Ohno K, Minami T, Matsui Y, Magara Y (2008). Effects of chlorine on organophosphorus pesticides adsorbed on activated carbon: Desorption and oxon formation. *Water Research*, 42, 6-7, 1753-1759.

Real FJ, Benitez FJ, Acero JL, Gonzalez M (2007). Removal of diazinon by various advanced oxidation processes. *Journal of Chemical Technology and Biotechnology*, 82:566-574.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Dicamba

(endorsed 2011)

GUIDELINE

Based on human health concerns, dicamba in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Dicamba (CAS 1918-00-9) belongs to the chlorophenoxy chemical class. Other pesticides in this class include 2,4-D, MCPA and mecoprop (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, dicamba would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Dicamba is used as a herbicide for the control of annual and perennial broad-leaf weeds in home garden lawns, turf and agricultural crops.

There are currently products registered in Australia that contain dicamba, its acid derivative, its dimethylamine salt or its sodium salt. Dicamba products are intended for professional or home garden use. Products are available as concentrated solutions to be applied diluted and undiluted using boom spray, aerial spray, and hand-held sprays or watering cans. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to dicamba and its metabolites are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of dicamba may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Dicamba is a very polar herbicide and results of its analysis in water have traditionally been negative. This may be a factor in the absence of reliable data on the presence of dicamba in Australian waters. A survey of pesticides including dicamba in New South Wales drinking water reported in 1989 (Ang *et al.* 1989) did not find detectable levels of dicamba in any sample. A recent published paper (Tran *et al.* 2006)

reported use of state-of-the-art analytical techniques to determine the presence of a range of herbicides including dicamba in agricultural drainage waters in Australia. While a range of herbicides was detected, no dicamba was found in the samples.

One of the main studies on dicamba incidence in water was from Canada, to derive Canadian Water Quality Guidelines (Caux *et al*. 1993). Dicamba was detected in less than 8% of surface water samples, with a maximum concentration of 13 µg/L; and in groundwater, it was detected in 2% of samples but at concentrations to 517 µg/L.

TREATMENT OF DRINKING WATER

Dicamba has been shown to be completely removed when water undergoes advanced oxidation with iron-catalysed ultraviolet irradiation and peroxide (Fenton reaction) (Huston and Pignatello 1999).

MEASUREMENT

Dicamba is a very polar herbicide that is not amenable to direct analysis by gas chromatographic techniques. It is commonly extracted from water via solid phase extraction techniques and determined by high performance liquid chromatography using ultraviolet detection. This approach leads to limits of detection of approximately 1 µg/L (Krzyszowska and Vance 1994). This process has recently been refined in a study investigating a range of hydrophilic solid phase extraction techniques (Tran *et al*. 2006).

HISTORY OF THE HEALTH-BASED GUIDELINE

The current acceptable daily intake (ADI) for dicamba is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 3 mg/kg bw/day from a short-term (developmental toxicity) study. The NOEL is based on maternal toxicity (decreased bodyweight) in rabbits. The ADI incorporates a safety factor of 100 and was established in 1991.

The previous ADI for dicamba was 0.05 mg/kg bw/day. This ADI was established in 1986; however, there is no information indicating how it was derived.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Dicamba is readily and extensively absorbed via the gastrointestinal tract in all species studied. It does not undergo significant metabolism and is excreted rapidly, predominantly in urine, largely unchanged.

Acute effects: Dicamba has low acute oral and dermal toxicity. Dicamba is a moderate skin sensitiser in guinea-pigs.

Short-term effects: A 13-week dietary study in rats reported decreased bodyweight gain and elevated liver enzyme levels at 500 mg/kg bw/day.

Long-term effects: A 2-year dietary study in mice reported increased male mortality and reduced female bodyweight gain at the high dose (450 mg/kg bw/day). The NOEL for this study was 150 mg/kg bw/day.

Carcinogenicity: Based on long-term dietary studies in mice, there is no evidence of carcinogenicity for dicamba.

Genotoxicity: There is evidence of genotoxicity but only at dose levels well in excess of the likely level of human exposure.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats did not report any evidence of toxicity in adult rats or offspring at doses up to 25 mg/kg bw/day. In developmental studies in rats and rabbits, there was no evidence of delayed development or teratogenicity. Maternotoxicity (decreased bodyweight gain in rats and neurotoxicity in rabbits) was reported. The most sensitive NOEL for maternotoxicity was 3 mg/kg bw/day in rats, which was used to establish the current ADI.

Poisons Schedule: Dicamba is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for dicamba was determined as follows:

$$0.1 \text{ mg/L} = \frac{3 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 3 mg/kg bw/day is the NOEL based on a short-term (developmental) study in rabbits.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Ang C, Meleady K, Wallace L (1989). Pesticide residues in drinking water in the north coast region of New South Wales, Australia, 1986-87. *Bulletin of Environmental Contamination and Toxicology, 42:595-602.*

Caux PY, Kent RA, Tache M, Fan, GT, MacDonald DD (1993). Environmental fate and effects of Dicamba: A Canadian perspective. *Reviews of Environmental Contamination and Toxicology*, 133:1-58.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction. *Water Research,* 33(5):1238-1246.

Krzyszowska A, Vance G (1994). Solid phase extraction of dicamba and picloram from water and soil samples for HPLC analysis. *Journal of Agricultural and Food Chemistry,* 42:1693-1696.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Tran ATK, Hyne RV, Doble P (2006). Determination of commonly used polar herbicides in agricultural drainage waters in Australia by HPLC. *Chemosphere,* 67(5):944-953.

NOTE: Important general information is contained in PART II, Chapter 6

Dichlorobenzenes
1,2-dichlorobenzene (1,2-DCB)
1,3-dichlorobenzene (1,3-DCB)
1,4-dichlorobenzene (1,4-DCB)

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, the concentrations of dichlorobenzenes in drinking water should not exceed the values shown below.

Dichlorobenzenes would not be a health concern unless concentrations exceeded the health values shown below:

	Health value	*Aesthetic value*
1,2-dichlorobenzene	***1.5 mg/L***	***0.001 mg/L***
1,3-dichlorobenzene	***inadequate data***	***0.02 mg/L***
1,4-dichlorobenzene	***0.04 mg/L***	***0.0003 mg/L (300 ng/L)***

GENERAL DESCRIPTION

Dichlorobenzenes are widespread in the environment and may be present in drinking water though spills and discharges, from atmospheric deposition, or by contact with contaminated soils. Studies in Japan, England, Canada and the United States have reported concentrations in the range 0.0000005 mg/L (0.5 ng/L) to 0.013 mg/L. Most supplies tested are below 0.00001 mg/L (10 ng/L), with 1,2-DCB and 1,4-DCB the most widely detected isomers. Sources of human exposure to DCBs are mainly food and air.

The dichlorobenzenes impart an offensive taste and odour to water, with thresholds between 0.0003 mg/L and 0.02 mg/L.

1,4-DCB is used in toilet blocks to deodorise air, and as a moth repellent, and is widely diffused in the environment. 1,3-DCB is a minor fumigant and insecticide and can be formed from incomplete combustion of waste. 1,2-DCB is used primarily as a chemical intermediate for dyestuffs and pesticides.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

DCBs have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

It is unlikely that DCB concentrations are reduced significantly during conventional water treatment processes. Removal using packed tower aeration or by the use of granular activated carbon is more than 90% effective and it is likely that concentrations below 0.001 mg/L can be achieved using these methods.

NOTE: Important general information is contained in PART II, Chapter 6

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and the dichlorobenzenes trapped on an adsorbent. The adsorbent is then heated and the dichlorobenzenes analysed using gas chromatography with electron capture detection. The limit of determination is approximately 0.0002 mg/L.

HEALTH CONSIDERATIONS

DCBs are absorbed rapidly through the lungs and from the gastrointestinal tract, and then distributed to tissues, primarily to fat or fatty tissue, and the lungs and kidneys. They are metabolised by the liver to the respective chlorophenols and eliminated in urine.

An extensive review and summary of the human and animal toxicity data for chlorobenzenes is available (IPCS 1986).

In the various reported cases of human exposure to DCBs, inhalation is the primary route. Toxic effects include liver damage, blood disorders, and disturbances to the immune system, the central nervous system or the respiratory tract. Skin pigmentation and allergic dermatitis have followed skin contact.

In rodents, long-term gavage (measured force-feeding) studies showed high doses of 1,2-DCB (120 mg/kg body weight per day) to affect mainly the liver and kidney, but found no adverse effects at lower doses. On balance, the available evidence suggests that 1,2-DCB is neither mutagenic in tests with bacteria nor carcinogenic in rodents.

No data are available on chronic toxicity for 1,3-DCB. No mutagenic activity was seen in tests with bacteria.

Long-term gavage studies involving 1,4-DCB produced similar results to the 1,2-DCB studies. In addition, there is evidence that 1,4-DCB increases the incidence of kidney tumours in male rats and liver tumours in mice after long-term exposure. It did not exhibit mutagenic activity in tests with bacteria or mammalian cells.

The International Agency for Research on Cancer has concluded that 1,4-DCB is possibly carcinogenic to humans (Group 2B, inadequate evidence in humans but sufficient evidence in animals), but that 1,2-DCB is unclassifiable as to its carcinogenicity (Group 3, inadequate evidence in humans and animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline values for dichlorobenzenes in drinking water were determined as follows:

i) 1,2-dichlorobenzene:

The health-based guideline value of 1.5 mg/L was determined as follows:

$$1.5\ \text{mg/L} = \frac{60\ \text{mg/kg body weight per day} \times 70\ \text{kg} \times 0.1}{2\ \text{L/day} \times 100} \times \frac{5}{7}$$

where:

- 60 mg/kg body weight per day is the no-effect level from a 2-year gavage study using mice (NTP 1985).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

NOTE: Important general information is contained in PART II, Chapter 6

FACT SHEETS

- 100 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations).
- 5/7 is used to convert data based on a 5 day per week gavage study to a 7-day week equivalent.
- Other factors are as above.

This health-based guideline value exceeds the taste and odour threshold of 0.001 mg/L. The World Health Organization (WHO) guideline value of 1 mg/L was based on an adult body weight of 60 kg. The difference in the guideline values is not significant.

ii) 1,3-dichlorobenzene

There are insufficient long-term data to set a guideline value for 1,3-DCB in drinking water based on health considerations. The maximum concentration guideline of 0.02 mg/L is based on the aesthetic considerations of taste and odour.

iii) 1,4-dichlorobenzene

The health-based guideline value of 0.04 mg/L was determined as follows:

$$0.04\ \text{mg/L} = \frac{150\ \text{mg/kg body weight per day} \times 70\ \text{kg} \times 0.1}{2\ \text{L/day} \times 10{,}000} \times \frac{5}{7}$$

where:

- 150 mg/kg body weight per day is the lowest effect level based on the appearance of kidney tumours in a 2-year gavage study using rats (NTP 1987).
- 10,000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations, 10 because a lowest effect level was used instead of a no-effect level, and 10 because carcinogenic effects were observed at the lowest doses used).
- Other factors are as above.
- 5/7 is used to convert data based on a 5 day per week gavage study to a 7-day week equivalent.

This health-based value exceeds the taste and odour threshold of 0.0003 mg/L. The WHO guideline value of 0.3 mg/L did not include the additional factor of 10 for possible carcinogenic effects.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1991). Chlorobenzenes other than hexachlorobenzene. Environmental Health Criteria, 128. World Health Organization, IPCS.

NTP (National Toxicology Program) (1985). Toxicology and carcinogenesis studies of 1,2-dichlorobenzene (o-dichlorobenzene) in F344/N rats and B6C3F1 mice (gavage studies). NTP Technical Report No. 255, Publication No. 86-2511. United States Department of Health and Human Services, National Institute of Health.

NTP (National Toxicology Program) (1987). Toxicology and carcinogenesis gavage studies of 1,4-dichlorobenzene in F344/N rats and B6C3F1 mice. NTP, Technical Report Series No. 319, Publication No. 87-2575, National Institute of Health, Bethesda, United States.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Dichloroethanes
1,1-dichloroethane
1,2-dichloroethane

(endorsed 1996)

GUIDELINE

1,1-dichloroethane: data are inadequate to set a drinking water guideline value.

1,2-dichloroethane: based on health considerations, the concentration in drinking water should not exceed 0.003 mg/L.

GENERAL DESCRIPTION

Dichloroethanes are present in some industrial effluent and have occasionally been found in drinking water supplies in the United States at concentrations below 0.006 mg/L.

The major use for 1,2-dichloroethane is in the production of vinyl chloride. It is also used in the production of other solvents, and can be used as a lead scavenger in petrol. 1,1-dichloroethane is used in the commercial production of 1,1,1-trichloroethane, as a solvent in paints, and as a varnish and finish remover.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Dichloroethanes have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

The dichloroethanes can be removed from drinking water using packed tower aeration, or by adsorption onto granular activated carbon.

MEASUREMENT

The dichloroethanes can be analysed by the purge and trap method (USEPA Method 502.1 1986). In this method an inert gas is bubbled through the sample and the dichloroethanes trapped on an adsorbent. The adsorbent is then heated and the dichloroethanes analysed using gas chromatography with electron capture detection. The limit of determination is approximately 0.0002 mg/L.

HEALTH CONSIDERATIONS

1,2-dichloroethane is absorbed through the lungs and gastrointestinal tract. Highest concentrations occur in the kidney and liver where it is metabolised to 2-chloroethanol. There are few data for 1,1-dichloroethane but it could be absorbed faster as it is more lipophilic (fat soluble).

An extensive review and summary of the human and animal toxicity data for 1,2-dichloroethane is available (IPCS 1987).

A number of cases of poisoning following consumption of high doses of 1,2-dichloroethane have been reported. While not all cases have been fatal, death is attributed to circulatory and respiratory failure.

1,1-dichloroethane has been used as an anaesthetic. Its use was discontinued because of problems associated with heart rhythm.

A 13-week inhalation study with 1,1-dichloroethane reported elevated blood-urea nitrogen concentrations in cats but not in rats, rabbits or guinea pigs. No other adverse effects were observed. A 78-week feeding study reported a marginally significant increase in the incidence of tumours of the mammary glands of female rats. No statistically significant increase in tumours was observed in male rats, or male and female mice. 1,1-dichloroethane has exhibited mutagenic activity in tests with bacteria and mammalian cells.

A 13-week feeding and drinking water study with 1,2-dichloroethane using rats and mice reported increased kidney and liver weights at high doses (4000 mg/L). No increase in the incidence of tumours or lesions was observed in mice or male rats, but female rats exhibited an increase in the incidence of kidney lesions.

A significant increase in tumours of the fore-stomach and circulatory system was reported in male rats fed 1,2-dichloroethane five times per week for 78 weeks. The same study reported tumours of the mammary glands in female rats. 1,2-dichloroethane has exhibited mutagenic activity in tests with different strains of bacteria, and metabolites are known to be strongly mutagenic.

The International Agency for Research on Cancer has concluded that 1,2-dichloroethane is possibly carcinogenic to humans (Group 2B, no data in humans but sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The assessment of the toxicological data of these compounds by the World Health Organization (WHO) has been used without review; however, the guideline value has been adjusted to a risk level of one in one million.

i) 1,1-dichloroethane

There are insufficient long-term data to set a health-based guideline value for 1,1-dichloroethane in drinking water.

ii) 1,2-dichloroethane

The guideline value for 1,2-dichloroethane in drinking water has been set at 0.003 mg/L. The WHO has conservatively calculated, using an extrapolation model based on a 78 week study in rats (NCI 1978), that consumption of water containing 0.003 mg/L of 1,2-dichloroethane would pose a lifetime risk of one additional cancer per million people.

The guideline value should be reviewed when more data are available.

The WHO guideline value of 0.03 mg/L was based on a calculation that estimated an additional lifetime risk of one fatal cancer per 100,000 people.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1987). 1,2-Dichloroethane. Environmental Health Criteria, 62. World Health Organization, IPCS.

NCI (National Cancer Institute) (1978). Bioassay of 1,2-dichloroethane for possible carcinogenicity. NCI, Department Health Education and Welfare, Washington DC, Report NCI-CG-TR-55.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Dichloroethenes
1,1-dichloroethene (1,1-DCE)
1,2-dichloroethene (1,2-DCE)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentrations of dichloroethenes in drinking water should not exceed the following values:

1,1-dichloroethene	***0.03 mg/L***
1,2-dichloroethene	***0.06 mg/L***

GENERAL DESCRIPTION

Available data indicate that the dichloroethenes are rarely found in drinking water. Studies in the United States have very occasionally reported DCEs in groundwater, usually from wells heavily contaminated with other chlorinated solvents.

1,1-DCE is used as a chemical intermediate in the manufacture of chloroform and polyvinylidene (PVDE) polymers. 1,2-DCE is also used as an intermediate in the manufacture of chlorinated solvents, and as a solvent. It can occur as two isomers, the *cis* and *trans* forms.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

DCEs have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

The DCEs can be removed from drinking water by aeration, or by adsorption onto granular activated carbon.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and the dichloroethenes trapped on an adsorbent. The adsorbent is then heated and the dichloroethenes analysed using gas chromatography with electron capture detection. The limit of determination is approximately 0.0002 mg/L.

HEALTH CONSIDERATIONS

The DCEs can be readily absorbed through the lungs and the gastrointestinal tract. They are distributed primarily to the liver and kidneys, and are metabolised to chloroacetic acid, chloroacetyl chloride, dichloroacetaldehyde and reactive epoxides.

In humans, exposure to high concentrations in air can lead to central nervous system depression. The DCEs have been used as anaesthetics.

NOTE: Important general information is contained in PART II, Chapter 6

A long-term study where rats were exposed to 1,1-DCE in their drinking water for 2 years reported minimal swelling to liver cells but no other adverse effects. No changes were observed in tissues taken from dogs after 97 days of exposure. 1,1-DCE induced tumours in mice in one inhalation study, but was not carcinogenic in other studies, including one drinking water study. It has exhibited some mutagenic activity in tests with bacteria but not with cultured mammalian cells.

The International Agency for Research on Cancer has concluded that 1,1-DCE is not classifiable as to its carcinogenicity (Group 3, evidence inadequate in humans and limited in animals) (IARC 1987).

No long-term data are available for 1,2-DCE; however, a 90-day immunotoxicity study with mice using the *trans* isomer reported increases in glutathione levels and aniline hydroxylase activity. No data are available on carcinogenicity bioassays with animals. The *cis* isomer, but not the *trans* isomer, has exhibited some mutagenic activity *in vivo* in tests with bacteria. Neither isomer induced chromosomal aberrations in hamster lung cells *in vitro*.

DERIVATION OF GUIDELINE

The assessment of the toxicological data of these compounds by the World Health Organization (WHO) has been used without review. The guideline values were determined as follows:

i) 1,1-dichloroethene

$$0.03 \text{ mg/L} = \frac{9 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 70.1}{2 \text{ L/day} \times 1000}$$

where:

- 9 mg/kg body weight per day is the lowest effect level based on a 2-year drinking water study using rats (Quast *et al.* 1983).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 because a lowest effect level was used instead of a no-effect level).

ii) 1,2-dichloroethene

$$0.06 \text{ mg/L} = \frac{17 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 17 mg/kg body weight per day is the no-effect level based on a 90-day drinking water study using mice (Barnes *et al.* 1985).
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for the less than lifetime study).
- other factors apply as above.

The WHO guideline value of 0.05 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

Barnes DW, Sanders VM, White KL, Shopp GM, Munson AE (1985). Toxicology of trans-1,2-dichloroethylene in the mouse. *Drug and Chemical Toxicology*, 8:373–392.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

Quast JF, Humiston CG, Wade CE, Ballard J, Beyer JE, Schwetz RW, Norris JM (1983). A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. *Fundamental and Applied Toxicology*, 3:55–62.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Dichloromethane (methylene chloride)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of dichloromethane in drinking water should not exceed 0.004 mg/L.

GENERAL DESCRIPTION

Dichloromethane releases into the environment are substantial and widely dispersed. In overseas studies it has been found in the parts-per-trillion range in air and is a common contaminant of ground and surface waters, with higher concentrations found in groundwater. In surface waters it can volatilise into air and will degrade in the atmosphere.

Dichloromethane is a widely used organic solvent. It can be found in paints, insecticides, degreasing agents, cleaning fluids and paint strippers.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Dichloromethane has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Dichloromethane concentrations in drinking water can be reduced using aeration, or by adsorption onto granular activated carbon.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and dichloromethane trapped on an adsorbent. The adsorbent is then heated and dichloromethane analysed using gas chromatography with electron capture detection. The limit of determination is approximately 0.0003 mg/L.

HEALTH CONSIDERATIONS

Studies indicate that dichloromethane is completely absorbed after ingestion and distributed primarily to the liver. It is metabolised to carbon monoxide, carbon dioxide and formic acid.

An extensive review and summary of the human and animal toxicity data for dichloromethane is available (IPCS 1984).

Inhalation of high doses has induced narcosis in humans, and acute exposure has caused impairment of sensory and motor functions.

In animals, a 2-year drinking water study on rats reported some changes to the liver at doses from 52 mg/kg body weight per day. Studies have shown mice to be less sensitive than rats to the toxic effects of dichloromethane.

Epidemiological investigations have failed to demonstrate a correlation between dichloromethane exposure and increased cancer incidence.

Overall, carcinogenicity of dichloromethane given in water to rodents is borderline and not conclusive. By inhalation there is clear evidence of carcinogenicity in rodents (IARC 1987).

The International Agency for Research on Cancer has concluded that dichloromethane is possibly carcinogenic to humans (Group 2B, inadequate evidence in humans but sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value for dichloromethane in drinking water of 0.004 mg/L was determined as follows:

$$0.004 \text{ mg/L} = \frac{6 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 5000}$$

where:

- 6 mg/kg body weight per day is the lowest effect level based on a 2-year drinking water study using rats (Serota *et al.* 1986).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 5000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations, 10 for genotoxicity and 5 for lowest effect level).

The World Health Organization guideline of 0.02 mg/L did not include a safety factor for the use of a lowest effect level. The need to use this additional factor arose after a statistical evaluation of the data in the referenced study indicating that the end point was a lowest effect level, not a no-effect level.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1984). Methylene chloride. Environmental Health Criteria, 32. World Health Organization, IPCS.

Serota DG, Thakur AK, Ulland BM, Kirschman JC, Brown NM, Coots RH, Morgareidge K (1986). A two-year drinking water study of dichloromethane in rodents: I. Rats. *Food and Chemical Toxicology*, 24:951–958.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

1,3-Dichloropropene

(endorsed 2011)

GUIDELINE

Based on human health concerns, 1,3-dichloropropene in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

1,3-Dichloropropene (CAS 542-75-6) does not belong to a recognised class of chemicals. The commercial product is a mixture of the *cis* and *trans* isomers (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, 1,3-dichloropropene would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: 1,3-Dichloropropene is a nematocide for the control of soil pests including plant parasitic nematodes.

There are registered products that contain 1,3-dichloropropene in Australia. These products are intended for professional use only by authorised or licensed persons as a pre-plant soil fumigant. 1,3-dichloropropene is available as a concentrated solution to be applied in undiluted form using soil injection equipment, or in diluted form by drip irrigation, and applied as either a broadacre or row soil treatment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to 1,3-dichloropropene is contamination of water. No residues are expected in food.

Agricultural use of 1,3-dichloropropene may potentially lead to contamination of source waters through absorption into the soil moisture and subsequent leaching into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of 1,3-dichloropropene in Australian drinking waters have been identified. However, laboratory experiments indicate that leaching 1,3-dichloropropene residues through soil to water is likely to be significant (Guo *et al.* 2003). Therefore there is some risk of contamination of groundwaters in areas where 1,3-dichloropropene is applied to agricultural soils.

TREATMENT OF DRINKING WATER

No reports have been identified for the targeted treatment of 1,3-dichloropropene in drinking water supplies.

MEASUREMENT

Analysis of 1,3-dichloropropene by solid-phase micro-extraction followed by gas chromatography with electron capture detection has been reported, with a limit of quantitation of 0.5 μg/L (Fuster *et al.* 2005). Further optimisation of this approach has been reported to achieve method detection limits of 0.5 ng/L for cis-1,3-dichloropropene and 1.0 ng/L for trans-1,3-dichloropropene (Antelo *et al.* 2007). An on-line purge and trap gas chromatography mass spectrometry has been developed with a reported limit of quantitation of 0.1 μg/L (Frenich *et al.* 2009).

HISTORY OF THE HEALTH VALUES

Currently no acceptable daily intake (ADI) or acute reference dose (ARfD) values have been established for 1,3-dichloropropene, since these are required only for pesticides with residues in food. There are no detectable residues in crops when 1,3-dichloropropene is used as a pre-plant soil fumigant.

An *Australian Drinking Water Guidelines* health value has not previously been established.

HEALTH CONSIDERATIONS

Metabolism: 1,3-dichloropropene is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, and the metabolites are rapidly excreted in the urine, almost completely within 48 hours. The primary metabolite of 1,3-dichloropropene in the urine is N-acetyl-S-(3-chloroprop-2-enyl)cysteine.

Acute effects: 1,3-dichloropropene (both the *cis* isomer and a mixture of the *cis* and *trans* isomers) has moderate acute oral and dermal toxicity in rats, and is a skin sensitiser in guinea pigs.

Short-term effects: Short-term studies in mice and rats reported reduced bodyweight gain, decreased clinical chemistry parameters, hyperplasia of the gastric mucosa and decreased organ weights at 15 and 5 mg per kg body weight per day (mg/kg bw/day), respectively. At higher doses in mice, there was decreased vacuolation of the renal tubules at 175 mg/kg bw/day. In a short-term study in dogs, vomiting occurred at 10 mg/kg bw/day. The lowest overall no-observed-effect level (NOEL) was 3 mg/kg bw/day in rats.

Long-term effects: Two-year dietary studies in mice and rats reported reduced bodyweight gain, decreased serum triglycerides and decreased organ weights at 25 mg/kg bw/day and 12.5 mg/kg bw/day respectively. In rats, there was also an increased incidence of liver adenomas and hyperplasia of the gastric mucosa at 12.5 mg/kg bw/day. A 1-year dietary study in dogs reported anaemia, with increased haematopoiesis from 15 mg/kg bw/day. The lowest NOEL was 2.5 mg/kg bw/day in rats, mice and dogs.

Carcinogenicity: Tumour formation was observed in long-term studies in mice and rats. In rats, there was an increase in primary hepatocellular adenomas at 12.5 mg/kg bw/day, and squamous cell papillomas and some carcinomas of the forestomach at 50 mg/kg bw/day. In mice, there was an increase in bladder and lung tumours at 50 mg/kg bw/day. These effects occurred at doses well in excess of the likely level of human exposure through drinking water. In male mice exposed via inhalation to 60 ppm technical-grade 1,3-dichloropropene for 24 months, an increased incidence of bronchioalveolar adenomas was seen.

Genotoxicity: 1,3-dichloropropene was positive in some *in vitro* short-term assays but negative in most *in vivo* assays. Overall, it was not considered to be genotoxic.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: Reproduction studies and developmental toxicity studies via the inhalation route in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development. No dietary studies were available.

Poisons Schedule: 1,3-dichloropropene is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), with an Appendix J rider limiting availability to authorised or licensed persons. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for 1,3-dichloropropene was determined as follows:

$$0.1 \text{ mg/L} = \frac{2.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 1}{2 \text{ L/day} \times 1000}$$

where:

- 2.5 mg/kg bw/day is the NOEL based on long-term (1- and 2-year) studies in rats, mice and dogs.
- 70 kg is taken as the average weight of an adult.
- The proportionality factor is 1, since 1,3-dichoropropene has no residues in food and therefore the assumption is that 100% of the ADI (nominal in this case) will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation and an additional 10 for the uncertainty associated with the potential carcinogenic risk.

The World Health Organization has established a health-based guideline value of 0.02 mg/L for 1,3-dichloropropene in 1993 (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Antelo A, Lasa M, Millan E (2007). Use of experimental design to develop a method for analysis of 1,3-dichloropropene isomers in water by HS-SPME and GC-ECD. *Chromatographia,* 66(7-8): 555-563.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Frenich AG, Aguado SC, Vidal JLM, Arrebola FJ (2009). On-line purge and trap GC-MS for monitoring 1,3-dichloropropene in agricultural water and soil samples. *Journal of Chromatographic Science,* 47(1): 26-30.

Fuster S, Beltran J, Lopez FJ, Hernandez F (2005). Application of solid phase microextraction for the determination of soil fumigants in water and soil samples. *Journal of Separation Science,* 28(1):98-103.

Guo MX, Yates SR, Zheng W, Papiernik SK (2003). Leaching potential of persistent soil fumigant residues. *Environmental Science & Technology,* 37(22):5181-5185.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality,* 3rd Edition. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Dichlorprop/Dichlorprop-P

(endorsed 2011)

GUIDELINE

Based on human health concerns, dichlorprop in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Dichlorprop (CAS 7547-66-2/CAS 15165-67-0) is in the phenoxycarboxylic acid group of chemicals. Dichlorprop possesses a single asymmetric carbon and is a chiral molecule. Dichlorprop is a racemic mixture of stereoisomers and dichlorprop-P is the R-isomer of dichlorprop. Other pesticides in this group include 2,4-D, MCPA and mecoprop (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, dichlorprop would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level even for a relatively short period are of concern as the health-based guideline is based on short- to medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate

inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Dichlorprop is a herbicide used for the control of weeds and as a plant growth regulator for citrus fruits.

There are registered product containing either dichlorprop (as the potassium salt) or dichlorprop-P (as the 2-ethylhexyl ester) in Australia. These are for professional use and applied by spray (backpack, boom spray or helicopter) to citrus fruits or non-crop areas. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to dichlorprop is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are few available data for dichlorprop in Australian drinking water. In Australian treated sewage, dichlorprop was below 0.5 µg/L (supporting data, NRMMC/EPHC/NHMRC 2008). In Canada, the maximum concentration in drinking water was 0.10 µg/L (Donald *et al.* 2007).

TREATMENT OF DRINKING WATER

Although no empirical data are currently available, it is likely that activated carbon would provide efficient removal based on its chemical structure.

MEASUREMENT

Dichlorprop in water can be analysed by gas chromatography with mass spectrometry, with a detection limit of 0.42 ng/L (Donald *et al*. 2007).

HISTORY OF THE HEALTH VALUES

The acceptable daily intake (ADI) for dichlorprop (R and S isomers) is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 3.1 mg/kg bw/day from a 13-week study in dogs. The NOEL was based on changes in clinical chemistry and kidney discolouration. The ADI incorporates a safety factor of 100 and was established in 1998.

The ADI for dichlorprop-P is 0.03 mg/kg bw, based on a NOEL of 6 mg/kg bw/day in an 18-month mouse dietary study. The NOEL is based on chronic necropathy. The ADI incorporates a safety factor of 200 and was established in 2006.

An acute reference dose (ARfD) has not been set for dichlorprop. An acute reference dose (ARfD) of 0.20 mg/kg bw/day for dichlorprop-P was established based on a NOEL of 20 mg/kg bw/day from a developmental study in rats. The ARfD incorporates a safety factor of 100.

An *Australian Drinking Water Guidelines* health value has not been previously established for dichlorprop-P.

HEALTH CONSIDERATIONS

The toxicology data on dichlorprop-P largely form the basis for the public health standards for both of these chemicals.

Metabolism: Dichlorprop-P is rapidly and extensively absorbed via the gastrointestinal tract in rats. There is limited metabolism of dichlorprop and no evidence of tissue accumulation. It is largely excreted unchanged in the urine within 24-48 hours. Metabolism and toxicokinetic data are similar for the dichlorprop-P acid and 2-ethylhexylester forms.

Acute effects: Dichlorprop-P has low acute oral and dermal toxicity in rats. It is not a skin sensitiser.

Short-term effects: Short-term studies in mice reported effects on the liver and on blood cholesterol at very high dose levels only. Studies in rats and dogs also reported effects on the kidney and liver at high dose levels. Blood lipids were decreased in dogs at 17 mg/kg bw/day.

Long-term effects: Long-term dietary studies in mice and dogs reported the kidney as the main target organ. The lowest NOEL was 6 mg/kg bw/day in mice and this NOEL is the basis of the ADI.

Carcinogenicity: Based on long-term studies in mice, there is no evidence of carcinogenicity for dichlorprop-P.

Genotoxicity: Dichlorprop-P is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Neurotoxicity: In an oral single dose and a dietary repeat dose study in rats, there was no evidence of delayed neurotoxicity.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Dichlorprop and dichlorprop-P are included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for dichlorprop and dichlorprop-P was determined as follows:

$$0.1 \text{ mg/L} = \frac{6 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 6 mg/kg bw/day is the NOEL for dichlorprop-P based on a long-term (18-month) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is a safety factor applied to the NOEL derived from a study conducted in animals. The safety factor incorporates a factor of 10 for interspecies extrapolation, a factor of 10 for intraspecies variation, and an additional factor of 2 for uncertainty in the toxicology database.

The World Health Organization has established a health-based guideline value of 0.1 mg/L for dichlorprop, in 1993 (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Donald DB, Cessna AJ, Sverko E, Glozier NE (2007). Pesticides in surface drinking-water supplies of the Northern Great Plains. *Environmental Health Perspectives,* 115:1183-1191.

NRMMC (Natural Resource Management Ministerial Council), EPHC (Environment Protection and Heritage Council), NHMRC (National Health and Medical Research Council) (2008). *Australian Guidelines for Water Recycling: Managing health and environmental risks (Phase 2) - Augmentation of drinking water supplies*. NRMMC, EPHC, NHMRC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Dichlorvos

(endorsed 2011)

GUIDELINE

Based on human health concerns, dichlorvos in drinking water should not exceed 0.005 mg/L.

RELATED CHEMICALS

Dichlorvos (CAS 62-73-7) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including acephate, chlorpyrifos, diazinon, fenitrothion, profenofos and trichlorfon (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, dichlorvos would not be a health concern unless the concentration exceeded 0.005 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Dichlorvos is an insecticide and acaricide for the control of insects in a wide range of situations, including in domestic and public health situations, as well as in warehouses and storerooms and in animal houses. Dichlorvos is also used to control pests in a wide range of crops and is used as a veterinary anthelmintic.

There are registered products containing dichlorvos in Australia. These products are intended for professional and home garden use. The products used as insecticides are generally available as emulsifiable concentrates to be diluted and applied by spray. The veterinary products containing dichlorvos are formulated as an oral paste for veterinary use. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to dichlorvos are the use of home garden products and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of dichlorvos may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

The veterinary use of dichlorvos provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Dichlorvos has been routinely monitored by some water utilities in Australia. No detections above analytical limits of detection have been reported in the reviewed reports.

TREATMENT OF DRINKING WATER

Coagulation achieves only 10-20% removal of organophosphorus pesticides. Ozone treatment can achieve complete degradation of dichlorvos under experimental conditions (Liu et al. 2009). Kim et al. (2002) reported a complete reduction of dichlorvos (initial concentration of 220 mg/L) by batch treatment with ozone at 1 mg/L. Good photocatalytic degradation/oxidation of dichlorvos has been reported using ultraviolet (UV) irradiation (Lu 1994, Liu et al. 2009) and UV/titanium dioxide (Evgenidou et al. 2005, Senthilnathan et al. 2009). A challenger advanced oxidation process test using ozone and hydrogen peroxide was able to reduce by 49% the influent concentration of dichlorvos, and the combination of advance oxidation and activated carbon filter reduced dichlorvos by 99% (USEPA 2007).

Nanofiltration using different membranes gave 4–87% removal from a 1 mg/L solution; adsorption onto the membrane was an important contributor to removal (Ozaki et al. 2000). A concentration of 0.01 µg/L was reduced by 98–99% by different ultra-low reverse osmosis membranes (Hofman et al. 1998). Reverse osmosis challenger test with an initial concentration of 1300 µg/L was efficient in removing dichlorvos (95% removal) (USEPA 2005).

MEASUREMENT

Dichlorvos is included in the United States Environmental Protection Agency (USEPA) gas chromatographic Method 622. The extract is concentrated and analyzed by gas chromatography using a flame photometric or phosphorus/nitrogen detector (Pressley *et al.* 2002). Hollow fibre-liquid phase microextraction with gas chromatography by flame thermionic detection can achieve a limit of detection of 32 ng/L for dichorvos (Lambropoulou *et al.* 2005). Enzyme-linked immunosorbent assay (IC-ELISA) method can achieve a limit of quantitation of 0.048 µ/mL for dichlorvos (Tang *et al.* 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for dichlorvos is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.014 mg/kg bw/day from a short-term (28-day) human study. The NOEL is based on plasma cholinesterase inhibition. The ADI incorporates a safety factor of 10, and was established in 2004.

The previous ADI was 0.0005 mg/kg bw based on a NOEL of 0.05 mg/kg bw/day for cholinesterase inhibition in a dog study, and using a safety factor of 100.

The acute reference dose (ARfD) of 0.1 mg/kg bw for dichlorvos was established in 2004, based on a NOEL of 1 mg/kg bw/day from a single oral dose study in human males. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.001 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Dichlorvos is readily absorbed from the gastrointestinal tract. It is extensively metabolised and rapidly excreted in the urine and as exhaled CO_2 within 24 hours.

Acute effects: Dichlorvos has moderate to high acute oral and dermal toxicity in rats. It is a skin sensitiser in both humans and guinea pigs.

NOTE: Important general information is contained in PART II, Chapter 6

Short-term effects: In a 28-day dietary study in humans, there was inhibition of plasma cholinesterase activity at 0.021 mg/kg bw/day. The NOEL of 0.014 mg/kg bw/day in humans is the basis for the current ADI.

Inhibition of plasma and red blood cell cholinesterase activity was reported at 0.9 mg/kg bw/day and above in a 90-day dietary study in dogs, and at 1.5 mg/kg bw/day in a 13-week oral study in rats.

Long-term effects: In a 2-year rat study, inhibition of plasma and red blood cell cholinesterase activity was reported from 2.3 mg/kg bw/day. In a 2-year dog study, inhibition of red blood cell cholinesterase activity in males was reported at 0.08 mg/kg bw/day and above.

Carcinogenicity: The weight of evidence from long-term studies in rodents indicates that dichlorvos is not carcinogenic.

Genotoxicity: Dichlorvos was positive in some *in vitro* short-term assays, but not genotoxic *in vivo*.

Reproductive and developmental effects: A 2-generation reproduction study in rats reported effects on reproductive parameters at high dose levels, which were well in excess of the likely human exposure levels. Developmental studies in mice, rats and rabbits did not produce any evidence of effects on foetal development.

Neurotoxicity: Short-term oral studies in chickens and rats did not report any evidence of delayed neurotoxicity.

Poisons Schedule: Dichlorvos is included in Schedules 5, 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.005 mg/L for dichlorvos was determined as follows:

$$0.005 \text{ mg/L} = \frac{0.014 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.014 mg/kg bw/day is the NOEL based on a short-term (28-day) study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from human studies to allow for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Evgenidou E, Fytianos F, Poulios I (2005). Semiconductor-sensitized photodegradation of dichlorvos in water using TiO_2 and ZnO as catalysts. *Applied Catalysis B: Environmental,* 59(1/2):81-89.

NOTE: Important general information is contained in PART II, Chapter 6

Hofman JAMH, Beerendonk EF, Folmer HC, Kruithof JC (1998). Removal of pesticides and other micropollutants with cellulose-acetate, polyamide and ultra-low pressure reverse osmosis membranes. *Desalination,* 113(2-3):209-214.

Kim B, Fujita H, Sakai Y, Sakoda A, Suzuki M (2002). Catalytic ozonation of an organophosphorus pesticide using microporous silicate and its effect on total toxicity reduction. *Water Science and Technology,* 46(4/5):35-41.

Lambropoulou DA, Albanis TA (2005). Application of hollow fiber liquid phase microextraction for the determination of insecticides in water. *Journal of Chromatography A,* 1072(1):55-61.

Liu C, Qiang ZM, Tian F, Zhang T (2009). [Reactivity of several classes of pesticides with UV, ozone and permanganate]. *Huan Jing Ke Xue,* 30(1):127-33.

Lu M. C (1994). Photocatalytic oxidation of dichlorvos in the presence of hydrogen peroxide and ferrous iron. *Water Science and Technology,* 30(9):29-38.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ozaki H, Li H (2000). Rejection properties of non-phenylic pesticides with nanofiltration membranes. *Journal of Membrane Science,* 171(2):229-237.

Pressley T, Longbottom J (2002). *Method 622 Method 622. The Determination of Organophosphorus Pesticides in Municipal and Industrial Wastewater.* http://www.accustandard.com/asi/pdfs/epa_methods/622.pdf

Senthilnathan J, Philip L (2009). Removal of mixed pesticides from drinking water system by photodegradation using suspended and immobilized TiO_2. *Journal of Environmental Science and Health, Part B,* 44(3):262-70.

Tang J, Zhang M, Cheng G, Li A, Lu Y (2008). Development of IC-ELISA for detection of organophosphorus pesticides in water. *Journal of Environmental Science and Health, Part B,* 43(8):707-12.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA Method 622: The Determination of Organophosphorus Pesticides in Municipal and Industrial Wastewater, #600/4-82-008, NTIS #82-156027. Washington, DC, United States Environmental Protection Agency.

USEPA (United States Environmental Protection Agency) (2005). *Point of use drinking water treatment system application: Removal of chemical contaminants in drinking water: Reverse osmosis*. The Environmental Technology Verification Program, USEPA.

USEPA (United States Environmental Protection Agency) (2007). *Point of use drinking water treatment system application: Removal of chemical contaminants in drinking water: Advanced simultaneous oxidation process*. The Environmental Technology Verification Program, USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Diclofop-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, diclofop-methyl in drinking water should not exceed 0.005 mg/L.

RELATED CHEMICALS

Diclofop-methyl (CAS 51338-27-3) belongs to the aryloxyphenoxy propionate and chlorophenoxy class of chemicals. Other pesticides in these classes include dichlorprop, fenoprop, 2,4-D, 2,4,5-T and MCPA (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, diclofop-methyl would not be a health concern unless the concentration exceeded 0.005 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Diclofop-methyl is used as a post-emergence herbicide for the control of grass weeds in turf and agricultural crops.

There are currently products registered in Australia that contain diclofop-methyl. All of these are intended for professional use and not for use in the home garden. Diclofop-methyl is available in concentrated solutions to be applied in diluted form using aircraft, ground sprayers or knapsack spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to diclofop-methyl and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of diclofop-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of diclofop-methyl in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of diclofop in drinking water have been identified.

MEASUREMENT

Diclofop-methyl can be analysed in water by solid phase extraction followed by high performance liquid chromatography with diode array detection (Ozhan *et al*. 2005). The limit of detection for this method is 0.04 µg/L. Diclofop-methyl can also be analysed in environmental samples by gas chromatography–mass spectrometry (Tadeo *et al*. 1996).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for diclofop-methyl is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day from a long-term study. The NOEL is based on liver toxicity observed in a 2-year study in mice. The ADI incorporates a safety factor of 100 and was established in 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Diclofop-methyl is readily absorbed via the gastrointestinal tract. It is extensively metabolised and is excreted in the urine and faeces, within 96 hours.

Acute effects: Diclofop-methyl has low oral acute toxicity and moderate acute dermal toxicity. It is a skin sensitiser.

Short-term effects: Medium-term dietary studies in rats and dogs reported increases in liver weight and enlargement of hepatocytes, in addition to changes in blood chemistry at dose levels of 1.5 mg/kg bw/day in rats and at 4 mg/kg bw/day in dogs. Fatty infiltration of the adrenal cortex was reported at high doses in dogs.

Long-term effects: Long-term dietary studies in mice, rats and dogs demonstrated the most sensitive toxic effects to be increased liver, heart and kidney weights in addition to increased serum alkaline phosphatase levels in the mouse, at dose levels of 0.8 mg/kg bw/day. Decreased bodyweight and liver pathology and changes in blood chemistry were observed at high doses in all species. The NOEL of 0.25 mg/kg bw in mice is the basis for the current ADI.

Carcinogenicity: Two-year studies in mice reported an increase in hepatocellular adenomas at 2.5 mg/kg bw/day, considered to be related to liver toxicity. In the absence of positive genotoxicity, these tumours were considered to be a threshold effect.***Genotoxicity:*** Diclofop-methyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rabbits showed no adverse effects on reproduction or development

Poisons Schedule: Diclofop-methyl is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.005 mg/L for diclofop-methyl was determined as follows:

$$0.005 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 0.25 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise rom the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation and 2 for uncertainty due to potential for tumour promotion.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ozhan G, Ozden S, Alpertunga B (2005). Determination of commonly used herbicides in surface water using solid-phase extraction and dual-column HPLC-DAD. *Journal of Environmental Science and Health Part B – Pesticides Food Contaminants and Agricultural Wastes,* 40(6):827-840.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Tadeo JL, SanchezBrunete C, GarciaValcarcel AI, Martinez L, Perez RA (1996). Determination of cereal herbicide residues in environmental samples by gas chromatography. *Journal of Chromatography A,* 754(1-2):347-365.

NOTE: Important general information is contained in PART II, Chapter 6

Dicofol

(endorsed 2011)

GUIDELINE

Based on human health concerns, dicofol in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Dicofol (CAS 115-32-2) belongs to the organochlorine class of chemicals. Other previously used pesticides in this class include DDT, aldrin, dieldrin, chlordane and heptachlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, dicofol would not be a health concern unless the concentration exceeded 0.004 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Dicofol is an acaricide for the control of mites in agricultural, veterinary, and household settings.

There are registered products containing dicofol in Australia. These are intended for professional and home garden use. Use patterns include ground spray onto plants and onto soil for professional use, and by hand-held spray for home garden use. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to dicofol are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of dicofol may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on dicofol in Australian waters. Dicofol has low solubility and a very high sorption coefficient (K_{oc}) and is expected to adsorb rapidly to sediments and suspended matters rather than be present in water, except in exceptional circumstances. For example, dicofol was found at concentrations up to 0.0025 mg/L at Orestimba Creek in the USA following agricultural spraying in the area (Domagalski *et al.* 1996).

TREATMENT OF DRINKING WATER

Dicofol has been shown to be completely removed when water undergoes conventional clarification (with alum), powdered activated carbon dosing, followed by oxidation with ozone (Ormad *et al*. 2008). Adsorption onto powdered activated carbon has been performed with a relatively moderate-high level of removal. Chlorination/ozonation for dicofol removal has also been reported with moderate success.

MEASUREMENT

Dicofol can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for dicofol is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.12 mg/kg bw/day from a 1-year dietary study in dogs. This NOEL is based on evidence of toxicity in the pituitary and liver. The ADI incorporates a safety factor of 100 and was first established in 1990.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Dicofol is rapidly absorbed via the gastrointestinal tract with wide, uniform tissue distribution. It is slowly eliminated, mainly in the faeces, as benzophenone metabolites and de-chlorinated metabolites. Dicofol has a moderate potential for bioaccumulation in body fat.

Acute effects: Dicofol has moderate acute oral toxicity and low dermal toxicity. It is a skin sensitiser in guinea pigs.

Short-term and long-term effects: In short-term and long-term dietary studies in mice, rats and dogs, effects indicative of liver toxicity were reported in all species, and included changes in clinical chemistry parameters, enzyme levels and increased relative liver weight. Rats were the most sensitive species, with effects reported at dose levels of 0.7 mg/kg bw/day and above in a 3-month study and 2.5 mg/kg bw/day and above in a 2-year study. In dogs, pituitary toxicity in the form of cysts was noted at 0.72 mg/kg bw/day and above in a 1-year study. The lowest NOEL was 0.12 mg/kg bw/day and this is the basis of the ADI.

Carcinogenicity: Based on a long-term study in rats, there is no evidence of carcinogenicity for dicofol.

Genotoxicity: Dicofol is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A multigeneration reproduction study in rats and a developmental study in rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Dicofol is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.004 mg/L for dicofol was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.12 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.12 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Domagalski J (1996). Occurrence of dicofol in the San Joaquin River, California. *Bulletin of Environmental Contamination and Toxicology,* 57(2):284-291.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71:97–106.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Diflubenzuron

(endorsed 2011)

GUIDELINE

Based on human health concerns, diflubenzuron in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Diflubenzuron (CAS 35367-38-5) belongs to the benzoylurea class of chemicals. Other pesticides in this class include chlorfluazuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, diflubenzuron would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Diflubenzuron is an insecticide and a parasiticide for the control of blowfly and lice in cattle and sheep.

There are registered products that contain diflubenzuron in Australia. These products are intended for professional use and are available as topical solution/suspension formulations to be diluted and applied by dipping or hand jetting, or directly by pour-on along the midline of the back of sheep and cattle. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to diflubenzuron is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The veterinary use of diflubenzuron provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on diflubenzuron occurrence in Australian drinking water supplies were found. Exposure to diflubenzuron through drinking-water is expected to be negligible.

TREATMENT OF DRINKING WATER

No specific data on treatment for diflubenzuron have been found. However, the low aqueous solubility (0.08 mg/L) and relatively high log Kow of 3.7 suggest that it may be amenable to adsorption by activated carbon (WHO 2006). Reverse osmosis is also expected to be effective in removing diflurobenzuron, given its high molecular weight (310 g/mol).

MEASUREMENT

Diflubenzuron can be determined by high-performance liquid chromatography with ultraviolet detector. The method can achieve a limit of quantitation (LOQ) of 0.05 mg/L (Miliadis *et al.* 1999). A high-performance liquid chromatography with atmospheric pressure chemical ionisation and mass spectrometry method can achieve a LOQ of 0.025 mg/L (Barnes *et al.* 1995). Diflubenzuron in drinking water can also be determined by liquid chromatography with electrospray ionization and mass spectrometry, achieving a LOQ of 0.01 µg/L (Li *et al.* 2006). Automated solid-phase extraction and high-performance liquid chromatography with diode-array detection method can achieve a LOQ of 0.1 µg/L for diflubenzuron (Nouri *et al.* 1995). On-line pre-concentration method for the analysis of diflubenzuron in ground water samples using two C18 columns, and fluorescence detection after photochemical induced fluorescence post-column derivatization can achieve a LOQ of 0.01 µg/L (García *et al.* 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for diflubenzuron is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.0 mg/kg bw/day from long-term dietary studies in rats and dogs. The NOEL is based on haematotoxicity and liver damage. The ADI incorporates a safety factor of 100 and was established in 1985.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Diflubenzuron is relatively well absorbed from the gastrointestinal tract in rats (up to 50%). The two major metabolic routes are via hydroxylation of the aromatic rings (80%) and via scission of the ureido bridge (20%). Diflubenzuron is rapidly excreted in the urine and faeces, almost completely within 72 hours. There is no accumulation in body tissues.

Acute effects: Diflubenzuron has low acute oral and dermal toxicity. Its skin sensitisation potential has not been tested.

Short-term effects: Medium-term dietary studies were conducted in rats and dogs. In rats, there was an increase in relative adrenal weights and an increase in the incidence of necrotic foci in the liver at 2.5 mg/kg bw/day and above. Haematological changes indicative of anaemia were observed in males at 10 mg/kg bw/day. In dogs, methaemoglobin, sulfaemoglobin and spleen weights were elevated and there was evidence of liver toxicity at 6.6 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in rats and dogs. The 2-year rat study reported elevated methaemoglobin levels at 8 mg/kg bw/day. The 1-year dog study reported increased methaemoglobin and sulfaemoglobin levels, and increased pigmentation in macrophages and Kupffer cells of the liver at 10 mg/kg bw/day. Liver and spleen weights were significantly elevated at 50 and 250 mg/kg bw/day, respectively. The NOEL was 2 mg/kg bw/day in both the rat and dog studies, and is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for diflubenzuron.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Diflubenzuron was positive in some *in vitro* short-term assays, but negative in all *in vivo* studies. Overall, it is not considered to be genotoxic.

Reproductive and developmental effects: 1-, 2- and 3-generation reproduction studies in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development. Maternotoxicity occurred only at dose levels well in excess of the likely human exposure level.

Poisons Schedule: Diflubenzuron is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for diflubenzuron was determined as follows:

$$0.07 \text{ mg/L} = \frac{2.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.0 mg/kg bw/day is the NOEL based on long-term dietary studies in rats (2-years) and dogs (1-year).
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Barnes KA, Startin JR, Thorpe SA, Reynolds SL, Fussell RJ (1995). Determination of the pesticide diflubenzuron in mushrooms by high-performance liquid chromatography-atmospheric pressure chemical ionisation mass spectrometry. *Journal of Chromatography A,* 712(1):85-93.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

García MDG, Galera MM, Martínez DB, Gallego JG (2006). Determination of benzoylureas in ground water samples by fully automated on-line pre-concentration and liquid chromatography-fluorescence detection. *Journal of Chromatography A,* 1103(2):271-277.

Li Y, George JE, McCarty CL, Wendelken SC (2006). Compliance analysis of phenylurea and related compounds in drinking water by liquid chromatography/electrospray ionization/mass spectrometry coupled with solid-phase extraction. *Journal of Chromatography A,* 1134(1-2):170-6.

Miliadis GE, Tsiropoulos NG, Aplada-Sarlis PG (1999). High-performance liquid chromatographic determination of benzoylurea insecticides residues in grapes and wine using liquid and solid-phase extraction.. *Journal of Chromatgraphy A,* 835:113-120

NOTE: Important general information is contained in PART II, Chapter 6

Nouri B, Fouillet B, Toussaint G, Chambon P, Chambon R (1995). High-performance liquid chromatography with diode-array detection for the determination of pesticides in water using automated solid-phase extraction. *Analyst,* 120:1133-1136.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). Diflubenzuron in Drinking Water Use for Vector Control in Drinking Water Sources and Containers. Background document for development of WHO Guidelines for Drinking-Water Quality, WHO.

NOTE: Important general information is contained in PART II, Chapter 6

Dimethoate

(endorsed 2011)

GUIDELINE

Based on human health concerns, dimethoate in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Dimethoate (CAS 60-51-5) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes acephate, chlorpyrifos, diazinon, dichlorvos, fenitrothion, omethoate, profenofos and trichlorfon (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, dimethoate would not be a health concern unless the concentration exceeded 0.007 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Dimethoate is a broad use systemic insecticide and acaricide (miticide) for the control of insects and mites in the home garden and in agricultural crops.

There are registered products that contain dimethoate in Australia. These products are intended for professional and/or home garden use and are available in aerosol formulation or concentrated solutions. Agricultural products for professional use are intended for application as a dilute or concentrated spray using hand-held, ground boom, mist machine or aerial spray equipment, or application as a diluted seed dressing or concentrated pre-planting or post-harvest dip. Home garden products are intended for application as an aerosol spray or concentrated spray using hand-held equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to dimethoate are the use of home garden products and residues in food. Omethoate is an environmental degradant of dimethoate and thus the residue definition for dimethoate is “sum of dimethoate and omethoate, expressed as dimethoate”. Residue levels of dimethoate and omethoate in food produced according to good agricultural practice are generally low.

Agricultural use of dimethoate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on the occurrence of dimethoate in Australian waters could be found. In Canadian waters, dimethoate was not detected in drinking water (<0.6 µg/L) or in surface waters (<0.5 µg/L) (Health Canada 1986). The United States Environmental Protection Agency predicted surface water concentration after application is 0.44 to 33.4 µg/L, depending on use pattern (USEPA 2008).

TREATMENT OF DRINKING WATER

Dimethoate appears to be very well removed by chlorination but is likely to be transformed into omethoate in the process (Ormad *et al.* 2008). Activated carbon is partially effective for the removal of dimethoate (Ormad *et al.* 2008).

MEASUREMENT

Dimethoate can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for dimethoate is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a short-term (57-day) human volunteer study. The NOEL is based on cholinesterase inhibition. The ADI incorporates a safety factor of 10 and was established in 1988.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Since omethoate is an environmental degradant of dimethoate, the toxicity of omethoate needs to be considered in the context of the health impact of dimethoate in drinking water. A separate fact sheet is available for omethoate.

Metabolism: Dimethoate is well absorbed via the gastrointestinal tract and distributed rapidly to the tissues, accumulating in the liver and kidneys. It is slowly excreted as metabolites (85-91% of the dose within 5 days), primarily in the urine. The major metabolites were thiophosphate and phosphate esters, and omethoate (1-6%). The parent compound represented 1-2% of the excreted dose.

Acute effects: Dimethoate has moderate acute oral toxicity, and low acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: Short-term dietary studies in rats and dogs reported the main effect to be on the nervous system, with reduced erythrocyte and brain cholinesterase activity from the lowest dose of 2.48 mg/kg bw/day (rats) and 2.2 mg/kg bw/day (dogs). From 40 mg/kg bw/day, severe clinical signs consistent with cholinesterase inhibition were observed. In addition, increased mortality occurred in dogs at high doses.

Short-term (57-day) volunteer studies in humans reported inhibition of cholinesterase in whole blood at doses of 0.4 mg/kg bw/day and above. The NOEL from this study was 0.2 mg/kg bw/day and is the basis for the current ADI.

Long-term effects: Long-term dietary studies in mice, rats and dogs showed the most sensitive effects to be on the nervous system. In mice, erythrocyte cholinesterase inhibition was reported at 3.6 mg/kg bw/day and above. In rats, erythrocyte and brain cholinesterase were inhibited at 0.23 mg/kg bw/day

NOTE: Important general information is contained in PART II, Chapter 6

and above. In dogs, brain and erythrocyte cholinesterase were inhibited at 0.125 mg/kg bw/day and above. At higher dose levels, there were changes in haematological parameters and clinical chemistry, as well as organ weight changes. Clinical neurological signs consistent with cholinesterase inhibition were observed at 50 mg/kg bw/day.

Carcinogenicity: Based on long-term dietary studies in mice, rats and dogs, there was no evidence of carcinogenicity associated with dimethoate.

Genotoxicity: Dimethoate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A two-generation reproduction study in rats and developmental toxicity studies in rats and rabbits did not produce evidence of effects on reproductive parameters or foetal development. A developmental neurotoxicity study in rats reported increased post-natal mortality and cholinesterase inhibition in pups dosed *in utero* and post-natally at 0.5 mg/kg bw/day and above, in the absence of maternal toxicity. The NOEL from this study is 0.1 mg/kg bw/day. The potential effects of dimethoate on foetal development are the subject of a current review.

Poisons Schedule: Dimethoate is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for dimethoate was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a short-term (57-day) human volunteer study.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from human studies. This safety factor incorporates a factor of 10 for intraspecies (human) variation, with an additional safety factor of 10 to account for the uncertainty in the ADI (which is likely to be lower as a result of the current review).

The World Health Organization has a health-based guideline value of 0.006 mg/L for dimethoate (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1986). Guidelines for Canadian Drinking Water Quality: Technical documents – Dimethoate. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/dimethoate/]

NOTE: Important general information is contained in PART II, Chapter 6

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71:97–106.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Department of Health and Ageing (Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2008). Revised Interim Reregistration Eligibility Decision for Dimethoate. USEPA.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Diquat (ion), Diquat dibromide

(endorsed 2011)

GUIDELINE

Based on human health concerns, diquat in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Diquat (CAS 2764-72-9; 85-00-7) is a quaternary ammonium compound and belongs to the bipyridilium class of chemicals. The other pesticide in this class is paraquat (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, diquat would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Diquat is a contact herbicide for the control of aquatic weeds and weeds in agricultural crops. It is also used as a desiccant in seed crops and to clear weeds from bodies of water and sewerage systems.

There are currently products registered in Australia that contain diquat, mostly as diquat dibromide. Diquat products are intended for professional use and are available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to diquat and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The agricultural use of diquat involves direct application into water ways and sewerage systems, which may then enter source water for drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of diquat in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of diquat in drinking water have been identified.

MEASUREMENT

Diquat can be measured in drinking water by solid phase extraction followed by high performance liquid chromatography with mass spectrometry, with a method detection limit of 0.1 µg/L (Grey *et al.* 2002, Nunez *et al.* 2004, Rial-Otero *et al.* 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for diquat is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a long-term study. The NOEL is based on cataract formation in a 2-year rat dietary study. The ADI incorporates a safety factor of 100. It was established in 1985 and reaffirmed in 2002.

The acute reference dose (ARfD) of 0.05 mg/kg bw/day for diquat was established in 2002, based on a NOEL of 26.5 mg/kg bw/day from an acute dietary study in dogs. The ARfD incorporates a safety factor of 500.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Diquat is poorly absorbed via the gastrointestinal tract. The absorbed fraction undergoes minimal metabolism before being rapidly excreted in the urine, almost completely within 48 hours.

Acute effects: Diquat has a high acute oral and dermal toxicity. It is a skin sensitiser in guinea-pigs. Human poisoning incidents indicate the lowest lethal dose to be 6 g. Doses greater than 12 g are usually fatal.

Short-term effects: A 4-week dietary study in rats reported decreased bodyweight gain and food consumption as well as clinical biochemistry changes at doses of 17 mg/kg bw/day.

In a 3-week dermal study, rats were treated with diquat up to 80 mg/kg bw/day. The reported effects were irritation and tissue destruction at the application site at dose levels of 5 mg/kg bw/day. Systemic effects included decreased bodyweight gain, hypothermia and emaciation at the highest dose. Treatment-related increases in mortality were evident at 40 mg/kg bw/day.

Medium-term dietary studies were conducted in rats and dogs. In rats, there was increased inflammation of the tongue and palate and effects on the kidney. Cataract formation was reported at doses above 8.9 mg/kg bw/day. In dogs, the reported dose-related effects were tongue lesions, decreased appetite and decreased bodyweight gain.

Long-term effects: Two-year dietary studies were conducted in mice, rats and dogs. In mice, there was decreased bodyweight gain at doses of 4.2 mg/kg bw/day. In rats, there were effects in the kidney at doses of 10.7 mg/kg bw/day. Cataract formation was increased in both in rats and dogs at doses of 1 mg/kg bw/day and 1.7 mg/kg bw/day, respectively. The NOEL of 0.2 mg/kg bw forms the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for diquat.

Genotoxicity studies: Diquat gave positive results in some *in vitro* short-term assays, but overall, it is not considered to be genotoxic.

NOTE: Important general information is contained in PART II, Chapter 6

Reproduction and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects or teratogenicity. There were effects on maternotoxicity and foetal development only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Diquat is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE VALUE

The health-based guideline value of 0.007 mg/L for diquat was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for diquat based on the available evidence that the chemical is rarely found in drinking water, even though it may be used as an aquatic herbicide. A provisional guideline value of 0.01 mg/L was established based on the practical limit of detection in water (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Grey L, Nguyen B, Yang P (2002). Liquid chromatography-electrospray ionization isotope dilution mass spectrometry analysis of paraquat and diquat using conventional and multilayer solid-phase extraction cartridges. *Journal of Chromatography A,* 958(1-2):25-33.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Nunez O, Moyano E, Galceran MT (2004). Time-of-flight high resolution versus triple quadrupole tandem mass spectrometry for the analysis of quaternary ammonium herbicides in drinking water. *Analytica Chimica Acta,* 525(2):183-190.

Rial-Otero R, Cancho-Grande B, Perez-Lamela C, Simal-Gandara J, Arias-Estevez M (2006). Simultaneous determination of the herbicides diquat and paraquat in water. *Journal of Chromatographic Science,* 44(9):539-542.

NOTE: Important general information is contained in PART II, Chapter 6

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Dissolved oxygen

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, it is desirable that the dissolved oxygen concentration in drinking water be greater than 85% saturation.

No health-based guideline value has been set for dissolved oxygen.

GENERAL DESCRIPTION

Drinking water will generally contain an adequate concentration of dissolved oxygen; however, under some circumstances the oxygen concentration may be reduced. This may occur, for instance, where water has been drawn from deep storages, where there is considerable growth of microorganisms in a distribution system, or following prolonged periods of high water temperature.

Low oxygen concentrations or anoxic conditions enable nuisance anaerobic microorganisms to grow, producing by-products that affect the aesthetic quality of the water and increase corrosion of pipes and fittings.

There are a number of such nuisance microorganisms. Manganese-reducing bacteria produce black manganese deposits which can slough off pipes and soil laundry. Sulfate-reducing bacteria can produce hydrogen sulfide, giving drinking water a 'rotten egg' smell. Nitrate-reducing bacteria can produce nitrite. Iron-reducing bacteria can increase the concentration of ferrous ion in solution which will lead to the deposition of insoluble ferric salts when aeration is increased.

Localised pH changes associated with the growth of nuisance microorganisms can cause rapid corrosion in metal pipes.

Water from groundwater sources will generally have low oxygen concentrations and while this may cause no difficulties for most supplies, some supplies may need aeration to improve water quality (e.g. taste and odour).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies the dissolved oxygen concentration is generally greater than 85% saturation. Ground water supplies may have less dissolved oxygen.

TREATMENT OF DRINKING WATER

The dissolved oxygen concentration in drinking water can be increased by aeration or ozonation.

MEASUREMENT

The dissolved oxygen content of drinking water can be determined on site using an oxygen-sensitive membrane electrode (APHA Method 4500-O Part G 1992). Alternatively, the iodometric method (azide modification) can be used (APHA Method 4500-O Part C 1992).

HEALTH CONSIDERATIONS

There have been no direct health effects caused by low oxygen concentrations in drinking water. Indirect effects may result from the corrosion of fittings, which can give rise to higher concentrations of heavy metals such as lead, copper and cadmium, and by the anaerobic generation of hydrogen sulfide and nitrite.

DERIVATION OF GUIDELINE

The guideline value of more than 85% saturation is based on aesthetic considerations for taste, odour and prevention of corrosion of pipes and fittings. If the concentration is lower than 85%, an investigation should be carried out to determine the cause.

REFERENCES

APHA Method 4500-O Part C (1992). Oxygen: Azide modification. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 4500-O Part G (1992). Oxygen: Membrane electrode method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

Disulfoton

(endorsed 2011)

GUIDELINE

Based on human health concerns, disulfoton in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Disulfoton (CAS 298-04-4) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, disulfoton would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Disulfoton is an insecticide and acaricide (miticide) for the control of pest infestations in food crops.

There is at currently at least one registered product containing disulfoton in Australia. Disulfoton products are intended for use by professionals and are available as a granular formulation. The use pattern is to apply the granule at or below the soil level to lucerne, cotton, potato, pea, and bean crops in agricultural settings. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to disulfoton is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of disulfoton may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Information of the presence of disulfoton in Australian waters was not found. In extensive surveys in the USA, disulfoton was not found in surface waters or groundwaters (US-NLM 2009).

TREATMENT OF DRINKING WATER

Disulfoton has been shown to be completely removed when water undergoes advanced oxidation with iron-catalysed ultraviolet irradiation and peroxide (Fenton reaction) (Huston and Pignatello 1999).

MEASUREMENT

Disulfoton is commonly analysed in Australian laboratories after solvent extraction and gas chromatographic determination using a nitrogen-phosphorus detector or mass selective detector. Typical limits of quantitation (LOQ) are 1 µg/L. The use of solid phase microextraction can permit lower LOQs, down to 0.1 µg/L (Queiroz *et al.* 2001).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for disulfoton is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.01 mg/kg bw/day from a 30-day dietary study in humans. This NOEL is based on inhibition of cholinesterase activity. The ADI incorporates a safety factor of 10, and was established in 1988.

The previous ADI set in 1969 was 0.002 mg/kg bw, based on studies reviewed by WHO.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Disulfoton is rapidly absorbed from the gastrointestinal tract, with wide, uniform tissue distribution. It is rapidly eliminated, mainly in the urine as sulfoxide and sulfone metabolites.

Acute effects: Disulfoton has high acute oral toxicity and low acute dermal toxicity. The potential for skin sensitisation is unknown.

Short-term and long-term effects: In a 30-day dietary study in humans, disulfoton administered at a dose of 0.01 mg/kg bw/day did not produce a reduction in cholinesterase activity. This NOEL is the basis for the ADI.

In short- and long-term studies in mice, rats and dogs, all reported reduced cholinesterase activity together with symptoms indicative of nervous system toxicity. In 3-month studies, these effects were observed at 0.65 mg/kg bw/day in mice and 0.12 mg/kg bw/day and above in rats.

In long-term studies in rats and dogs, these effects were observed at 0.12 mg/kg bw/day in rats and 0.06 mg/kg bw/day in dogs.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for disulfoton.

Genotoxicity: Disulfoton is not considered to be genotoxic, on the basis of *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A multigeneration reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development. The only effect noted was decreased cholinesterase activity in parental animals at dose levels above 0.1 mg/kg bw/day.

Poisons Schedule: Disulfoton is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.004 mg/L for disulfoton was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.01 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.01 mg/kg bw/day is the NOEL from a short-term (30-day) dietary study in humans. No cholinesterase inhibition was observed at this dose.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from the human study. The safety factor incorporates a factor of 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download)

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction, *Water Research* 33(5):1238-1246.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queiroz M, Silva S, Carvalho D, Lancas F (2001). Comparison between solid-phase extraction methods for the chromatographic determination of organophosphorus pesticides in water. *Journal of Environmental Science and Health* Part B, 36(5):517-527.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

US-NLM (United States National Library of Medicine (2009). Hazardous Substances Database. http://toxnet.nlm.nih.gov/

Diuron

(endorsed 2011)

GUIDELINE

Based on human health concerns, diuron in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Diuron (CAS 330-54-1) belongs to the urea class of chemicals. Other pesticides in this class include fluometuron, linuron, methabenzthiazuron, siduron and tebuthiuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, diuron would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level even for a relatively short period are of concern as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Diuron is a herbicide used for the control of broad-leaf weeds and grasses on a variety of crops including citrus, pineapple, cereals and sugar cane, as well as around buildings and public rights of way. It is also used as a defoliant on cotton, in marine antifouling paints and in ponds and aquariums to control algae.

There are registered products containing diuron in Australia. These are intended for professional and home garden use and contain diuron alone or in combination with other active ingredients. The products are generally available as suspension concentrates and water dispersible granules to be mixed with water and applied by aerial or ground sprayers. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to diuron are the use of home garden products, entry into treated public areas, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of diuron may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater, or through the discharge of treated aquarium or pond water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of diuron in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Diuron has been shown to be completely removed from water by chlorination when the chlorine dose is adjusted to match chlorine demand (Ormad *et al.* 2008). Ozonation and activated carbon adsorption for diuron removal has also been reported with high-moderate success (Bozkaya-Schrotter *et al.* 2008, Ormad *et al.* 2008). Advanced oxidation using ultraviolet irradiation and peroxide has been demonstrated to achieve a moderate level of diuron removal (Kruithof *et al.* 2002). Conventional coagulation/floculation has been shown to provide a relatively low removal rate (30%).

MEASUREMENT

Diuron can be measured in drinking water by solid phase extraction followed by high performance liquid chromatography with tandem mass spectrometry. The limit of quantitation for this method is typically 0.01 µg/L (QHFSS 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for diuron is 0.007 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.7 mg/kg bw/day from a 6-month dietary study in rats. This NOEL is based on reduced haemoglobin concentrations and increased reticulocytes. The ADI incorporates a safety factor of 100 and it was established in 2005.

The previous ADI, established in 1987, was 0.006 mg/kg bw based on a NOEL of 0.6 mg/kg bw/day from a 2-year dog study. The NOEL was based on abnormal blood pigments (probably sulfaemoglobin).

The acute reference dose (ARfD) of 0.007 mg/kg bw/day for diuron was established in 2005, based on a NOEL of 0.7 mg/kg bw/day from a 6-month dietary study in rats, as indicated above. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L, with a note that this pesticide has either been detected on occasions in Australian drinking water or its likely use would indicate that it may occasionally be detected (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Diuron is rapidly and extensively absorbed from the gastrointestinal tract. It is efficiently metabolised and excreted within the first 24 hours. The main metabolites detected in blood and urine are 3-(3,4-dichlorophenyl)-1-methyl urea, 3-(3,4-dichlorophenyl) urea and 3,4-dichloroaniline.

Acute effects: Diuron has low acute oral and dermal toxicity in rats. It is not a skin sensitiser.

Short-term effects: Short-term dietary studies in rats reported changes to haematological parameters consistent with haemolytic anaemia at dose levels of 1.6 mg/kg bw/day in males and 1.8 mg/kg bw/day in females. Other effects included changes indicative of liver and kidney damage. The NOEL of 0.7 mg/kg bw/day in rats is the basis for the current ADI.

Long-term effects: Long-term dietary studies have been conducted on mice, rats and dogs. These studies reported findings of haemolytic anaemia at 3.1 mg/kg bw/day and above in a 2-year dog study. Other findings included liver enzyme level and organ weight increases and histological changes in liver, spleen and kidney, rats at 1 mg/kg bw/day and dogs at 7.5 mg/kg bw/day and above.

Carcinogenicity: The long-term study in rats reported an increased incidence of tumours in the bladder epitheliu; however, development of these tumours in rats appeared to be related to urinary pH changes as a result of feeding the rats a specific rat diet, rather than to diuron alone, and are therefore not considered to be relevant to humans.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Diuron is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats with diuron reported no changes in reproductive parameters. Developmental toxicity studies in rats and rabbits reported decreased foetal bodyweight only at above maternotoxic dose levels.

Poisons Schedule: Diuron is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for diuron was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.7 mg/kg bw/day is the NOEL based on a medium-term (6-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bozkaya-Schrotter B, Daines C, Lescourret A, Bignon A, Breant P, Schrotter J (2008). Treatment of trace organics in membrane concentrates I: pesticide elimination, *Water Science Technology: Water Supply*, 8(2):223-230.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Kruithof J, Kamp P, Belosevic M (2002). UV/H_2O_2 treatment: the ultimate solution for pesticide control and disinfection. *Water Science Technology: Water Supply*, 2(1):113-122.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71:97–106.

QHFSS (Queensland Health Forensic and Scientific Services) (2008). Personal Communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

2,2-DPA

(endorsed 2011)

GUIDELINE

Based on human health concerns, 2,2-DPA in drinking water should not exceed 0.5 mg/L.

RELATED CHEMICALS

2,2-DPA (2,2-dichloropropionic acid)(CAS 127-20-8) belongs to the class of halogenated aliphatic chemicals. Another pesticide in this class is iodomethane (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, 2,2-DPA would not be a health concern unless the concentration exceeded 0.5 mg/L. Excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking-water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: 2,2-DPA is a herbicide for the control of grasses and broad-leaf weeds in industrial sites, footpaths, domestic and public areas.

There are registered products that contain 2,2-DPA as its sodium salt in Australia. These products are intended for professional and home garden use. All are soluble powder formulations intended to be diluted and applied by hand spray or backpack spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to 2,2-DPA and its metabolites are the use of home garden products, and exposure from treated public places such as footpaths.

Use of 2,2-DPA in public spaces may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reported occurrences of 2,2-DPA in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No information regarding the effective treatment of 2,2-DPA in drinking water has been identified.

MEASUREMENT

2,2-DPA can be measured in drinking waters by liquid–liquid microextraction, acidic methanol derivatization, and gas chromatography–mass spectrometry. The reported method detection limit is 0.13 μg/L (Xie 2001).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for 2,2-DPA is 0.2 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 15 mg/kg bw/day from a long-term (2-year dietary) study. The NOEL is based on increased kidney weight in rats. The ADI incorporates a safety factor of 100 and it was established in 1989.

The previous *Australian Drinking Water Guidelines* health value was also 0.5 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: 2,2-DPA is rapidly absorbed from the gastrointestinal tract in humans. Peak levels in blood are reached by 2 to 3 hours, and decrease with a half life of 2-3 days. Metabolism is limited, with the majority of the 2,2-DPA excreted unchanged in urine.

Acute effects: 2,2-DPA has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Three-month dietary studies were conducted in rats and dogs. In rats, kidney weights were increased at doses of 50 mg/kg bw/day, and at higher doses there were effects on the liver. In dogs, there was no evidence of toxicity at doses up to 1000 mg/kg bw/day, however the study was of a poor quality.

Long-term effects: Long-term dietary studies in mice, rats and dogs reported increased kidney weights at 50 mg/kg bw/day and above. The lowest overall NOEL was 15 mg/kg bw/day in the rat study.

Carcinogenicity: Benign adenomas in lacrimal glands were noted in mice at 200 mg/kg bw/day, however, these tumours were not considered relevant to low-dose human exposure.

Reproductive and developmental effects: A 3-generation reproduction study in rats and a developmental study in rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Genotoxicity: 2,2-DPA is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Poisons Schedule: 2,2-DPA is considered not to require control by scheduling due to its low toxicity and is therefore in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.5 mg/L for 2,2-DPA was determined as follows:

$$0.5 \text{ mg/L} = \frac{15 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 15 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation. An additional safety factor is unnecessary.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Xie Y (2001). Analyzing haloacetic acids using gas chromatography/mass spectrometry. *Water Research*, 35(6):1599-1602.

Endosulfan

(endorsed 2011)

GUIDELINE

Based on human health concerns, endosulfan in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Endosulfan (CAS 115-29-7) is in the cyclodiene organochlorine class of chemicals. The other pesticide in this class is trinexapac-ethyl (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, endosulfan would not be a health concern unless the concentration exceeded 0.02 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Endosulfan is an insecticide used for the control a range of insects and mites on a broad spectrum of crops, as well as on cotton.

There are registered products containing endosulfan in Australia. The products are for professional use only and are available as emulsifiable concentrates to be diluted before use. Application is by aircraft, boom spray, or hand spray from the ground for crops other than cotton, and by aircraft for cotton crops. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to endosulfan is residues in food. The residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are few available data on endosulfan concentrations in Australian drinking water. Treated sewage in Australia contains less than 0.5 µg/L (supporting data, NRMMC/EPHC/NHMRC 2008). Internationally, endosulfan contamination does not appear to be widespread in the aquatic environment, but has been found in agricultural run-off and rivers in industrialised areas where it is manufactured or formulated (IPCS 1984). Endosulfan (one or both of its isomers) has been identified in 24 surface water samples and 103 groundwater samples collected from 164 hazardous waste sites in the USA. Surface water samples in the USA generally contain less than 1 µg/L (ATSDR 2000).

TREATMENT OF DRINKING WATER

Like many other pesticides, endosulfan is efficiently removed by activated charcoal (94% removal; Mishra and Patel 2007) and ozonation (94-97%; Yazgan and Kinaci 2003, Yazgan *et al*. 2003).

MEASUREMENT

The method of choice for the determination of endosulfan involves extraction from water with methylene chloride followed by gas chromatography combined with electron capture detection (WHO 2004a). In considering residue levels, the sum of the α- and β-somers plus the endosulfan sulfate metabolite, which is similar in toxicity to the parent compound, have to be considered. Detection limits are 0.015 µg/L for α-endosulfan, 0.024 µg/L for β-endosulfan and 0.015 µg/L for endosulfan sulfate (ATSDR 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for endosulfan is 0.006 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.57 mg/kg bw/day for decreased bodyweight gain in a 12-month dietary study in dogs, and a NOEL of 0.66 mg/kg bw/day for decreased bodyweight gain and damage to kidney tissue in long-term studies in rodents and a developmental study in rats. The ADI incorporates a safety factor of 100 and was established in 1997.

The previous ADI, set in 1968, was 0.007 mg/kg bw, based on a NOEL of 0.7-0.75 mg/kg bw/day established in a 1-year dog dietary study, a 13-week rat dietary study, and rat reproduction and developmental studies, using a 100-fold safety factor.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Endosulfan is readily absorbed via the gastrointestinal tract and widely distributed in the body. It is extensively metabolised to sulfates and has a low potential for bioaccumulation. Excretion occurs mainly in faeces and is complete within 72 hours.

Acute effects: Endosulfan has a high acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In short-term dermal studies in rats, endosulfan produced inhibition of serum and brain acetylcholinesterase and mild liver changes at 9 mg/kg bw/day. In medium-term dietary studies in mice and rats, there was an increase in granular pigment formation in the proximal tubule cells and increased kidney weight at 3.9 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies were conducted in rodents and dogs. A 2-year rat study reported reduced bodyweight, an increased incidence of enlarged kidneys and blood vessel aneurysms, and marked progressive glomerulonephrosis at 2.9 mg/kg bw/day. A 1-year study in dogs reported decreased bodyweight gain at 0.75 mg/kg bw/day. The NOEL of 0.57 in rats and dogs is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for endosulfan.

Genotoxicity: Endosulfan is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A reproduction study in rats and developmental toxicity studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Endosulfan is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010) depending on concentrations and use patterns. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for endosulfan was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.57 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.57 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variations.

The World Health Organization has not established a guideline value for endosulfan, because it occurs at concentrations well below those at which toxic effects are observed (WHO 2004b).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

ATSDR (Agency for Toxic Substances and Disease Registry) (2000) Toxicological profile for endosulfan. Published by the US Department of Health and Human Services, Public Health Service, ATSDR, Atlanta, GA.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

IPCS (International Programme on Chemical Safety) (1984). Endosulfan. Environmental Health Criteria 40. Published by the World Health Organization, IPCS, Geneva, Switzerland.

Mishra PC, Patel RK (2007). Removal of endosulfan by sal wood charcoal. *Journal of Hazardous Materials* 152(2):730-736.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NRMMC (Natural Resource Management Ministerial Council), EPHC (Environment Protection and Heritage Council), NHMRC (National Health and Medical Research Council) (2008). *Australian Guidelines for Water Recycling: Managing health and environmental risks (Phase 2) - Augmentation of drinking water supplies*. NRMMC, EPHC, NHMRC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

WHO (World Health Organization) (2004a). Endosulfan in drinking water. Background document for development of WHO Guidelines for Drinking-water Quality. World Health Organization, Geneva, Switzerland.

WHO (World Health Organization) (2004b). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

Yazgan MS, Kinaci C (2003). Beta-endosulfan removal from water by ozone oxidation. *Water Science and Technology,* 48(11-12): 511-517.

Yazgan MS, Kinaci C, Toroz I (2003). Ozonation of aqueous solution of alpha endosulfan. *Journal of Environmental Science and Health Part B,* 38(6):843-853.

NOTE: Important general information is contained in PART II, Chapter 6

Endothal

(endorsed 2011)

GUIDELINE

Based on human health concerns, endothal in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Endothal (CAS 145-73-3) belongs to the dicarboxylic-acid class of chemicals. There are currently no other pesticides in this chemical class registered for use (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, endothal would not be a health concern unless the concentration exceeded 0.1 mg/L. While the health-based guideline is based on long-term effects, there is potential for effects on the gastrointestinal tract after short-term exposure at levels 2-3 times higher than the health-based guideline. Therefore, excursions above this level even for a short period are of concern.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Endothal is a selective contact herbicide used for the control of winter grass in turf crops and lawns.

There are currently products registered in Australia that contain endothal, with both containing endothal as its di-potassium salt. Endothal products are intended for professional use on lawns. Products are liquid concentrates that are applied in diluted form using hand spray or boom spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to endothal and its metabolites s residues in foods such as potato and cotton seed oil. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of endothal may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are no published reports on endothal occurrence in Australian drinking water supplies.

TREATMENT OF DRINKING WATER

Endothal can be removed from drinking water using granular activated carbon.

MEASUREMENT

Endothal can be detected in drinking water using liquid-solid extraction followed by gas chromatography and electron capture detection, with a limit of detection (LOD) of 11.5 µg/L (EPA Method 548). Endothal has been analysed using derivatisation ion exchange extraction, acidic methanol methylation, and gas chromatography with mass spectrometry (GC/MS), with a LOD of 1.8 µg/L (EPA Method 548.1). Determination can be done using a flame ionisation detector instead of GC/MS, with a reported LOD of 0.7 µg/L (Hodgeson *et al.* 1992).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for endothal is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 3.75 mg/kg bw/day from a long-term (1-year) study in dogs. This NOEL is based on liver necrosis and stomach hyperplasia and hyperkeratosis. The ADI incorporates a safety factor of 100 and was established in 1990.

The previous ADI for endothal was 0.1 mg/kg bw/day, established in 1977 and based on a NOEL of 10 mg/kg bw/day in a 2-year study in dogs and a 100-fold safety factor. This study was later found to be inadequate for regulatory purposes, and the ADI was amended in 1990.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Endothal is well absorbed via the gastrointestinal tract and widely distributed. Peak levels in blood are reached at 1 hour after dosing. Metabolism is limited and endothal is excreted unchanged in the urine within 48 hours (99% of total dose). A small amount of unidentified metabolites is excreted in the faeces.

Acute effects: The di-potassium salt of endothal has a moderate acute oral toxicity in rats and endothal itself has a high acute dermal toxicity in rabbits. No studies are available on the skin sensitisation potential of endothal.

Short-term effects: The main toxic effect of endothal in oral short-term toxicity studies was gastrointestinal toxicity. In dogs treated for 6 weeks there was significant gastrointestinal tract erosion at the lowest dose tested, 10 mg/kg bw/day. A NOEL was not obtained for this study. A NOEL of 50 mg/kg bw/day was obtained from a short-term (28-day) dietary study in rat. In this study, high mortality rates associated with gastrointestinal necrosis and overt signs of toxicity were seen at doses of 500 mg/kg bw/day.

Following repeated dermal application of endothal to rabbits, mortality with associated histological changes in skin, liver, and kidney resulted at the lowest dose of 50 mg/kg bw/day.

Long-term effects: In long-term toxicity studies, the main toxic effects are on the stomach and liver. Stomach hyperplasia and hyperkeratosis was seen at 7.5 mg/kg bw/day in rats and 25 mg/kg bw/day in dogs. Liver necrosis was observed at 11 mg/kg bw/day in dogs, and liver hyperplasia/adenoma and ovarian cysts at 15 mg/kg bw/day in rats. The lowest NOEL was 3.75 mg/kg bw/day in a 1-year study in dogs, and this is the basis for the current ADI.

Carcinogenicity: Based on long-term toxicity studies in rats, there is no evidence of carcinogenicity for endothal.

Genotoxicity: Endothal is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and mice did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Endothal is in Schedules 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and use pattern. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for endothal was determined as follows:

$$0.1 \text{ mg/L} = \frac{3.75 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 3.75 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dog.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hodgeson JW, Collins J, Bashe WJ (1992). *Method 548.1 Determination of Endothall in Drinking Water by Ion-Exchange Extraction, Acidic methanol Methylation and Gas Chromatography/Mass Spectometry. Revision 1.0*. Environmental Monitoring Systems Laboratory Office of Research and Development, USEPA, Cincinnati, Ohio.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Epichlorohydrin

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of epichlorohydrin in drinking water should not exceed 0.0005 mg/L.

GENERAL DESCRIPTION

Epichlorohydrin is used in the manufacture of glycerine and unmodified epoxy resins, including resins used in water treatment (polyelectrolytes). The United States Environmental Protection Agency has proposed that the maximum residual epichlorohydrin content in flocculating agent shall not exceed 0.01% which, at maximum resin usage rates of 20 mg/L, would lead to an epichlorohydrin concentration in drinking water of less than 0.002 mg/L. No monitoring of epichlorohydrin concentrations in drinking water has been reported. Epichlorohydrin hydrolyses in water and this can cause difficulties in detection.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Epichlorohydrin has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

No published reports are available on water treatment procedures for the removal of epichlorohydrin. Aeration is unlikely to be successful.

MEASUREMENT

Epichlorohydrin can be determined using gas chromatography with electron capture detection (Pesselman and Feit 1988). The limit of determination is approximately 0.05 mg/L.

HEALTH CONSIDERATIONS

In laboratory animals epichlorohydrin is rapidly absorbed after ingestion, inhalation and skin contact, and is distributed to the liver, kidneys and pancreas.

An extensive review and summary of the human and animal toxicity data for epichlorohydrin is available (IPCS 1984).

In humans, skin contact with high concentrations can cause initial redness, itching, or a burning sensation. The initial effects of inhalation are similar, and can be followed by vomiting and severe headache. Long-term exposure can cause kidney and liver damage. Epichlorohydrin has been reported to increase chromosome damage in lymphocytes, and decrease blood cell counts in occupationally exposed workers.

A long-term study where male rats were given epichlorohydrin in their drinking water for 81 weeks reported a decrease in leucocytes and an increase in the incidence of fore-stomach tumours from a dose of 39 mg/kg body weight per day and fore-stomach hyperplasia from 18 mg/kg body weight per day. A 2-year gavage study in rats, using doses of 2 and 10 mg/kg body weight per day, also reported induction of fore-stomach carcinomas. Inhalation studies in rats have reported the appearance of nasal cavity carcinomas.

Epichlorohydrin has been shown to be genotoxic both *in vitro* and *in vivo*. It is an alkylating agent and a direct-acting mutagen.

The International Agency for Research on Cancer has concluded that epichlorohydrin is probably carcinogenic to humans (Group 2A, insufficient evidence in humans, sufficient evidence in animals, and other supportive data) (IARC 1987).

DERIVATION OF GUIDELINE

The assessment of the toxicological data for epichlorohydrin by the World Health Organization (WHO) has been used without review. The guideline value of 0.0005 mg/L was determined as follows:

$$0.0005 \text{ mg/L} = \frac{2 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10{,}000} \times \frac{5}{7}$$

where:

- 2 mg/kg body weight per day is the lowest effect level based on a 2-year gavage study using rats (Wester *et al*. 1985).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 10,000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations, 10 because a lowest effect level was used instead of a no-effect level, and 10 for carcinogenic effects).
- 5/7 is used to convert data based on a 5 day per week feeding study to a 7-day week equivalent.

Although epichlorohydrin is a genotoxic carcinogen, the use of a linear multistage model for estimating cancer risk was considered inappropriate because tumours are seen only at the site of administration where epichlorohydrin is highly irritating.

The limit of determination is approximately 0.05 mg/L; however, concentrations in drinking water can be controlled by product specification.

The WHO guideline value of 0.0004 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1984). Epichlorohydrin. Environmental Health Criteria, 33. World Health Organization, IPCS.

Pesselman RL, Feit MJ (1988). Determination of residual epichlorohydrin and 3-chloropropanediol in water by gas chromatography with electron capture detection. *Journal of Chromatography*, 439:488–542.

Wester PW, Van Der Heijden CA, Bisschop A, Van Esch GJ (1985). Carcinogenicity study with epichlorohydrin (CEP) by gavage in rats. *Toxicology*, 36:325–339.

NOTE: Important general information is contained in PART II, Chapter 6

EPTC

(endorsed 2011)

GUIDELINE

Based on human health concerns, EPTC in drinking water should not exceed 0.3 mg/L.

RELATED CHEMICALS

EPTC (S-ethyl-dipropylthiocarbamate)(CAS 759-94-4) belongs to the thiocarbamate class of chemicals. Other pesticides in this class include pebulate, thiobencarb, and butylate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, EPTC would not be a health concern unless the concentration exceeded 0.3 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: EPTC is a pre-emergent herbicide for the control of certain grasses and broad-leaf weeds in agricultural vegetable crops.

There is at least one registered product containing EPTC in Australia. EPTC products are intended for professional use and are available as a concentrated solution to be diluted and applied directly onto the soil using ground, and hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to EPTC and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of EPTC may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on EPTC occurrence in Australian drinking water supplies were found. In the USA, EPTC was not detected at or above the limit of quantitation (1 µg/L) in any of the 3,873 public water systems sampled (serving a total population of 226 million) (USEPA 2008).

TREATMENT OF DRINKING WATER

There is no evidence that EPTC is substantially removed by conventional treatments, such as coagulation/ flocculation, sedimentation, and inert media filtration (USEPA 2008). Rapid degradation of EPTC has been reported with ultraviolet (UV) light at 254 nm. EPTC-sulfoxide, EPTC-sulfone, propylamine and dipropylamine were detected as photoproducts of EPTC at 254 nm There was negligible degradation of EPTC at 290 nm UV light (Abu-Qare *et al*. 2002). EPTC also shows high reactivity during the ozonation and ozone/hydrogen peroxide advanced oxidation process (Chen *et al*. 2008). Granular activated carbon is also considered a good option for the removal of EPTC from drinking water. Carbamate pesticides can be removed with 85.7% efficiency using a cellulose acetate membrane, 79.6–93% efficiency using a polyamide membrane, and greater than 92.9% efficiency using a thin-film composite membrane. These results indicate that reverse osmosis is effective in removing EPTC from drinking water (USEPA 2008).

MEASUREMENT

A solid-phase microextraction (SPME) method in water samples determined by gas chromatography coupled with flame thermionic detection for the measurement of EPTC can achieve a limit of quantitation (LOQ) of 0.01 µg/L. SPME, gas chromatography and mass spectrometric detection achieved a LOQ of 0.02 µg/L (Lambropoulou *et al*. 2002). EPTC can be detected in drinking water by United States Environmental Protection Agency (USEPA) Methods 507 and 525.2. USEPA Method 507 relies on solvent extraction of EPTC and separation by gas chromatography with a nitrogen-phosphorus detector. The method can achieve a LOQ of 0.08 µg/L. USEPA Method 525.2 uses liquid-solid extraction and capillary column gas chromatography/ mass spectrometry. The method can achieve a LOQ for EPTC in the range of 0.05 to 0.12 µg/L (USEPA 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for EPTC is 0.09 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 9 mg/kg bw/day from a 2-year dietary study in rats. The NOEL is based on hindleg muscle atrophy and sciatic nerve demyelination at at 18 mg/kg bw/day. The ADI incorporates a safety factor of 100, and was established in 1995.

The previous ADI for EPTC of 0.01 mg/kg bw, based on a NOEL of 20 mg/kg bw/day from a long-term dietary study in mice with safety factor of 2000 to allow for the limited database, was set in 1970. The ADI was amended in 1995 after additional studies were submitted, including a long-term dietary study in rats demonstrating a lower overall NOEL.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: EPTC is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, mainly via sulfoxidation and ester hydrolysis. Excretion via the urine was essentially complete by 72 hours.

Acute effects: EPTC has low acute oral and dermal toxicity. It is a weak skin sensitiser in guinea pigs.

Short-term effects: In a 90-day dietary study in rats, there was decreased bodyweight gain and evidence of hepatocyte hypertrophy at the highest dose of 32 mg/kg bw/day. In a 16-week dietary study in dogs, there was hair loss at 22 mg/kg bw/day and decreased brain cholinesterase activity and changes to the gastric mucosa at 44 mg/kg bw/day.

Long-term effects: In a long-term (2-year) dietary study in rats, there was hindleg muscle atrophy, sciatic nerve demyelination, and increased serum AST at 18 mg/kg bw/day. Degenerative cardiomyopathy was

NOTE: Important general information is contained in PART II, Chapter 6

seen at doses of 36 mg/kg bw/day and above. The NOEL was 9 mg/kg bw/day, and this is the basis for the current ADI.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for EPTC at doses up to 20 mg/kg/day.

Genotoxicity: EPTC is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study and a developmental toxicity study in rats did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: Hens dosed twice at a 21-day interval with oral doses of 7200 mg/kg bw EPTC did not exhibit any delayed neurotoxicity.

Poisons Schedule: EPTC is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.3 mg/L for EPTC was determined as follows:

$$0.3 \text{ mg/L} = \frac{9 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 9 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Abu-Qare AW, Duncan HJ (2002). Photodegradation of the herbicide EPTC and the safener dichlormid, alone and in combination. *Chemosphere,* 46(8):1183-9.

Chen WR, Wu C, Elovitz MS, Linden KG, Mel Suffet IH (2008). Reactions of thiocarbamate, triazine and urea herbicides, RDX and benzenes on EPA Contaminant Candidate List with ozone and with hydroxyl radicals. *Water Research,* 42(1-2):137-44.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lambropoulou DA, Sakkas VA, Hela DG, Albanis TA (2002). Application of solid-phase microextraction in the monitoring of priority pesticides in the Kalamas River (N.W. Greece). *Journal of Chromatography A,* 963(1-2):107-16.

NOTE: Important general information is contained in PART II, Chapter 6

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2008). *Regulatory Determinations Support Document for Selected Contaminants from the Second Drinking Water Contaminant Candidate List (CCL 2)*. Office of Ground Water and Drinking Water, USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Esfenvalerate

(endorsed 2011)

GUIDELINE

Based on human health concerns, esfenvalerate in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

Esfenvalerate (CAS 66230-04-4) belongs to the pyrethroid class of chemicals. This is a large chemical group and includes fenvalerate, cyfluthrin, permethrin and flucythrinate.

There are four optical isomers of esfenvalerate and fenvalerate (SS, SR, RS, RR). The SS isomer is responsible for the insecticidal activity of these compounds. Fenvalerate contains around 20% as the SS form while esfenvalerate is highly enriched in this form. Most of the toxicity of fenvalerate is caused by the RS isomer (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, esfenvalerate would not be a health concern unless the concentration exceeded 0.03 mg/L. Minor excursions above this level would need to occur over a relatively long period to be a health concern, as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Esfenvalerate is an insecticide for the control of ants, cockroaches, fleas, spiders and other insect pests in domestic and industrial areas, and in field and pasture crops.

There are registered products that contain esfenvalerate in Australia. These products are intended for professional and domestic use. These products are available for use in domestic and industrial areas as hand and ground sprays; or for use on crops using ground and aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to esfenvalerate are the use of domestic products, and residues in food. Esfenvalerate residues are grouped with those of fenvalerate in the maximum residue limit definition. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of esfenvalerate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Esfenvalerate has been monitored but not detected in Australian drinking water supplies (Muschal 2001). In the Goulburn Murray irrigation area esfenvalerate was detected on one occasion at 65 µg/L in 2005 (Victoria Department of Primary Industries 2006).

TREATMENT OF DRINKING WATER

No specific data on the treatment of esfenvalerate in drinking water have been identified.

MEASUREMENT

Esfenvalerate can be measured by liquid/liquid extraction followed by gas chromatography coupled with an electron capture detector. The method can achieve a limit of detection (LOD) of 0.05 µg/L. High-performance liquid chromatography or gas-liquid chromatography with either electron capture detection or electrolytic conductivity detection has been reported for the quantitation of esfenvalerate (Hengel *et al*. 1997). Solid-phase extraction followed by enzyme linked immunosorbert assay can achieve a LOD of 0.1 µg/L for esfenvalerate in water samples (Shan *et al*. 1999).

Trace levels of esfenvalerate in water can be measured by stir-bar-sorptive extraction followed by liquid desorption and large-volume injection capillary gas chromatography with mass spectrometric detection. This method can achieve a LOD of 2.5 ng/L (Serodio and Nogueira 2005). Gas chromatography/electron capture detector and gas chromatography / nitrogen-phosphorous detector methods can achieve a LOD of esfenvalerate below 2 ng/L (Wang *et al*. 2009).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for esfenvalerate is 0.008 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 7.5 mg/kg bw/day from a medium-term (13-week) dietary study in rats. The NOEL is based on clinical toxicity including abnormal behaviour and parotid salivary gland cell hypertrophy at 35 mg/kg bw/day. The ADI incorporates a safety factor of 1000 and was established in 1993.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Esfenvalerate is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, and is readily excreted in the urine, almost completely within 48 hours.

Acute effects: Esfenvalerate has moderate acute oral and dermal toxicity in rats. It is not a skin sensitiser.

Short-term effects: In a 4-week study in mice, there were signs of neurotoxicity in the form of tremors and excessive salivation at 35 mg/kg bw/day, and convulsions and gait abnormalities at 105 mg/kg bw/day.

In a 90-day feeding study in mice, there was decreased bodyweight gain and haematological changes at 30 mg/kg bw/day, and tremors and convulsions at 100 mg/kg bw/day. In a 13-week feeding study in rats, tremors, unsteady movements and convulsions were observed at 15 mg/kg bw/day. Cellular hypertrophy was observed in the parotid salivary glands and in the pituitary glands at 25 mg/kg bw/day. The lowest NOEL was 7.5 mg/kg bw/day in the rat study. This NOEL is the basis for the current ADI.

Long-term effects: Long-term toxicity studies have not been conducted in rodents with esfenvalerate, and were considered unnecessary given the available data on the closely-related fenvalerate (see the fenvalerate fact sheet).

NOTE: Important general information is contained in PART II, Chapter 6

Carcinogenicity: Esfenvalerate is not considered to be carcinogenic, based on the results of long-term toxicity studies in rodents with fenvalerate.

Genotoxicity: Esfenvalerate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Reproductive and developmental toxicity studies with esfenvalerate have not been conducted. There are no reproductive or developmental concerns based on studies with fenvalerate.

Neurotoxicity: Esfenvalerate did not cause delayed neurotoxicity in special oral dosing studies in rats.

Poisons Schedule: Esfenvalerate is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010) depending on concentration. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for esfenvalerate was determined as follows:

$$0.03 \text{ mg/L} = \frac{7.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 7.5 mg/kg bw/day is the NOEL based on a medium-term (13-week) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates factors of 10 for interspecies extrapolation, 10 for intraspecies variation, and an additional 10 for the lack of long-term studies.

Note: This calculated health-based guideline exceeds the normal aqueous solubility of esfenvalerate.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hengel MJ, Mourer CR, Shibamoto T (1997). New method for analysis of pyrethroid insecticides: esfenvalerate, cis-permethrin, and trans-permethrin, in surface waters using solid-phase extraction and gas chromatography. *Bulletin of Environmental Contamination and Toxicology,* 59(2):171-8.

Muschal M (2001). Central & North West Regions' water quality program. 1999-2000 Report on pesticides monitoring, NSW Department of Land and Water Conservation, Parramatta.

Serodio P, Nogueira JM (2005). Development of a stir-bar-sorptive extraction-liquid desorption-large-volume injection capillary gas chromatographic-mass spectrometric method for pyrethroid pesticides in water samples. *Analytical and Bioanalytical Chemistry,* 382(4):1141-51.

NOTE: Important general information is contained in PART II, Chapter 6

Shan G, Stoutamire DW, Wengatz I, Gee SJ, Hammock BD (1999). Development of an immunoassay for the pyrethroid insecticide esfenvalerate. *Journal of Agricultural and Food Chemistry,* 47(5):2145-55.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Victoria Department of Primary Industries (2006). Pesticide Monitoring in Goulburn-Murray Waters Irrigation Supply Channels Covering the Six Irrigation Areas [2004 -2006 Irrigation Season Study Report]. Website: http://74.125.153.132/search?q=cache:wQOWfp-0NsAJ:www.g-mwater.com.au/downloads/R_D/reportchannelpesticides.pdf+DEPARTMENT+OF+PRIMARY+INDUSTRIES+(2006)+Pesticide+Monitoring+in+Goulburn-Murray+Waters&cd=2&hl=en&ct=clnk&gl=au.

Wang D, Weston DP, Lydy MJ (2009). Method development for the analysis of organophosphate and pyrethroid insecticides at low parts per trillion levels in water. *Talanta,* 78(4-5):1345-51.

NOTE: Important general information is contained in PART II, Chapter 6

Ethion

(endorsed 2011)

GUIDELINE

Based on human health concerns, ethion in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Ethion (CAS 563-12-2) is in the organophosphate class of chemicals. There are many other pesticides in this class, including diazinon, dichlorvos, fenthion, parathion and profenofos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, ethion would not be a health concern unless the concentration exceeded 0.004 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Ethion is an insecticide used for the control of cotton worm in agricultural settings, and for controlling mites and fleas in cattle.

There are registered products containing ethion in Australia. These include an emulsifiable concentrate to be diluted and sprayed on cotton, used as ground and aerial sprays; and topical solutions/suspensions used as cattle dips/sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to ethion is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of sources waters through processes such as run-off, spray-drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of ethion in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of ethion in drinking water have been identified.

MEASUREMENT

Ethion can be measured in natural waters using headspace solid-phase microextraction followed by gas chromatography with flame thermionic or mass spectrometric detection. The practical limit of quantitation for this technique is 0.04 µg/L (Lambropoulou and Albanis 2001, Wang *et al.* 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for ethion is 0.0013 mg per kg of bodyweight (mg/kg bw), based on no-observed-effect levels (NOEL) of 0.13 mg/kg bw/day from a 2-year rat dietary study and 0.1 mg/kg bw/day from a 3-generation rat study. In both cases, the NOEL was based on decreased cholinesterase activity. The current ADI incorporates a safety factor of 100 and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Ethion is rapidly absorbed from the gastrointestinal tract and widely distributed in the body. It is rapidly metabolised to monoxons, dioxons, conjugates and numerous polar compounds, and has low potential for bioaccumulation. It is rapidly eliminated, mainly in the urine and to a minor extent in faeces.

Acute effects: Ethion has high acute oral and dermal toxicity. Symptoms of acute poisoning are indicative of central and peripheral nervous system poisoning and included hyperexcitability, salivation, broncoconstriction, headache, vomiting and other behavioural changes. The effects are dose-related and reversible. Ethion is not a skin sensitiser.

Short-term effects: Short-term and long-term dietary studies in rodents and dogs resulted in symptoms indicative of nervous system toxicity. Decreased cholinesterase activity was observed in serum and brain at dose levels above 0.46 mg/kg bw/day in mice and 1.8 mg/kg bw/day in rats.

Long-term effects: A long-term (2-year) dietary study in rats reported decreased serum cholinesterase. The NOEL was 0.13 mg/kg bw/day, and is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for ethion.

Genotoxicity: Ethion showed no evidence of genotoxicity in *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In reproduction studies in rats and developmental studies in rats and rabbits, there was no evidence of effects on reproductive parameters or on foetal development, but serum cholinesterase was decreased at dose levels above 0.l mg/kg bw/day.

Poisons Schedule: Ethion is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.004 mg/L for ethion was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 0.1 mg/kg bw/day is the NOEL based on decreased serum cholinesterase levels in a 3-generation study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies variation and 10 for intraspecies extrapolation.

REFERENCES

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lambropoulou DA, Albanis TA (2001). Optimization of headspace solid-phase microextraction conditions for the determination of organophosphorus insecticides in natural waters. *Journal of Chromatography A,* 922(1-2):243-255.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Wang S, Zhao P, Min G, Fang GZ (2007). Multi-residue determination of pesticides in water using multi-walled carbon nanotubes solid-phase extraction and gas chromatography-mass spectrometry. *Journal of Chromatography A,* 1165(1-2):166-171.

NOTE: Important general information is contained in PART II, Chapter 6

Ethoprophos

(endorsed 2011)

GUIDELINE

Based on human health concerns, ethoprophos in drinking water should not exceed 0.001 mg/L.

RELATED CHEMICALS

Ethoprophos (CAS 13194-48-1) belongs to the organothiophosphate class of chemicals. Other pesticides in this class include terbufos, chlorpyrifos, dimethoate, and ethion (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, ethoprophos would not be a health concern unless the concentration exceeded 0.001 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Ethoprophos is a nematocide and insecticide for the control of root nematodes in agricultural crops including bananas, cereals and tobacco, and for the control of certain grubs in sugar cane and maize.

There are currently no products registered in Australia that contain ethoprophos, but de-registered compounds may still be detected in water. Previously registered products containing ethoprophos were intended for professional use only. These products were granular formulations applied directly to the soil to control root nematodes or were diluted and applied by hand spray for the control of grubs.

Exposure sources: The main sources of public exposure to ethoprophos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of ethoprophos in the future may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of ethoprophos in Australian drinking waters have been identified. It is rarely detected in drinking or environmental waters in overseas countries. When it has occasionally been reported, levels are extremely low; for example, a time-weighted annual concentration in source waters in several USA states of 0.002 ppb and a maximum concentration of 0.012 ppb.

TREATMENT OF DRINKING WATER

No specific data on the treatment of ethoprophos in drinking water have been identified.

MEASUREMENT

Residue determination of ethoprophos is amenable to multiresidue procedures using solvent extraction with dichloromethane followed by gas chromatography with nitrogen-phosphorous detection. Limits of detection are as low as 0.01 µg/L using this technique.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for ethoprophos is 0.0003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.025 mg/kg bw/day from a long-term study. The NOEL is based on decreased erythrocyte cholinesterase activity in a 1-year dietary toxicity study in dogs. The ADI incorporates a safety factor of 100 and was established in 1988.

The previous ADI for ethoprophos was 0.0001 mg/kg bw/day, based on the same NOEL but with a safety factor of 200, due to reporting deficiencies. The ADI was amended after submission of additional information on the study.

The previous *Australian Drinking Water Guidelines* health value was 0.001 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Ethoprophos is rapidly absorbed via the gastrointestinal tract and and widely distributed in the tissues of rats. Metabolism is extensive and excretion of phosphorothioic acid and related esters is mainly through urine, but is incomplete by 7 days, with 1.7–3.5% of total dose remaining in tissues

Acute effects: Ethoprophos has high acute oral toxicity and moderate to high acute dermal toxicity. It is a skin sensitiser in guinea-pig tests.

Short-term effects: In both oral and dermal short-term studies from 3 to 6 weeks in rats, rabbits and dogs, the major effect reported was a decrease in chlolinesterase activity at 0.05 mg/kg bw/day and above. In dietary studies from 3-5 months in the rat and dog, there was a decrease in brain, plasma and erythrocyte cholinesterase levels and elevation of adrenal weights (rat) at 0.025 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies in mice, rats, and dogs reported decreases in erythrocyte cholinesterase activity in all species; the lowest dose was 1 mg/kg bw/day in the dog. Decreased brain and plasma cholinesterase activity and reduced haemoglobin levels were seen at higher doses in all species. The lowest overall NOEL was 0.025 mg/kg/day, based on a decrease in erythrocyte cholinesterase activity in dogs. This NOEL is the basis for the current ADI.

Carcinogenicity: Based on an 18-month study in mice and a 2-year study in rats, there is no evidence of carcinogenicity for ethoprophos.

Genotoxicity: Ethoprophos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Ethoprophos is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.001 mg/L for ethoprophos was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.025 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.025 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Ethylbenzene [CASRN 100-41-4]

(endorsed 2013)

GUIDELINE

Based on aesthetic considerations (taste and odour), the concentration of ethylbenzene in drinking water should not exceed 0.003 mg/L.

Based on health considerations the concentration of ethylbenzene in drinking water should not exceed 0.3 mg/L.

GENERAL DESCRIPTION

Ethylbenzene is a clear colourless liquid, which occurs naturally as a component of crude oil. It constitutes approximately 1-2% of unleaded gasoline by volume.

Ethylbenzene is produced commercially by the alkylation of benzene with ethylene, and by fractionation of petroleum. It is a major component of commercial xylene and is used commercially in paints, insecticides, blends of petrol, and in the production of styrene. It can also be found as a constituent of asphalt and naphtha.

Ethylbenzene has a taste and odour threshold of 0.003 mg/L.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Ethylbenzene has only rarely been identified in Australian drinking waters. Natural concentrations in most water sources are usually very low. Ethylbenzene can occur naturally in groundwater as a result of proximity to, or contact with, coal seams, petroleum and gas deposits, and shales. It may be mobilised by extraction activities (Lesage *et al.*, 1997; Leusch and Bartkow, 2011; Volk *et al*, 2011). However, contamination can occur, usually via exposure to petrochemicals in surface waters or groundwater. Known sources of groundwater contamination include leakage from sub-surface fuel storage tanks (do Rego & Netto, 2007). Emissions of fuel components from boating use is a known source of contamination of multiple-use lakes and reservoirs (Schmidt *et al.*, 2004). Ethylbenzene is generally not detected in groundwater (<0.001 mg/L), but concentrations in contaminated groundwater in the USA were as high as 2 mg/L (IPCS, 1996). Ethylbenzene has been reported at up to 0.000 2 mg/L in municipal drinking water in Croatia (Karaconji *et al.*, 2006), up to 0.011 mg/L in municipal drinking water in Taiwan (Kuo *et al.*, 1997) and is occasionally detected in drinking waters in the USA (Williams *et al.*, 2004) at concentrations up to 0.002 mg/L (ATSDR, 2010). Concentrations in Canadian drinking water ranged from <0.001 - 0.01 mg/L (IPCS, 1996).

TREATMENT OF DRINKING WATER

Volatile organic chemicals such as ethylbenzene are most commonly treated in drinking water by aeration stripping and/or adsorption to granular activated carbon (GAC). A conventional biologically active sand filter has been shown to be highly effective for the removal of ethylbenzene from contaminated water, under suitable conditions (Arvin *et al.*, 2004). Effective bioremediation of highly contaminated groundwaters has also been demonstrated (Sedran *et al.*, 2004; Zein *et al.*, 2006).

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for the analysis of ethylbenzene (APHA, AWWA & WEF, 2012). An inert gas is bubbled through the sample and ethylbenzene is trapped on an adsorbent. The adsorbent is then heated and ethylbenzene analysed using gas chromatography with

mass spectrometric (GC-MS) detection (Method 6200 B) or photoionisation (PI) detection (Method 6200 C) (APHA, AWWA & WEF, 2012). The method detection limit is 32 ng/L for GC-MS and 28 ng/L for GC-PI (APHA, AWWA & WEF, 2012).

HEALTH CONSIDERATIONS

Ethylbenzene is readily absorbed from the human gastrointestinal tract. It can be stored in fat and is metabolised to mandelic and phenylglyoxalic acids and excreted in the urine. It can cross the placenta.

No data are available on the health effects in humans after oral exposure, and inhalation data are limited to short term studies.

A 6 month gavage study using rats reported enlargement of the liver and kidney at high doses (400 mg/kg body weight per day) (Wolf *et al.*, 1956). Mellert *et al.* (2007) also reported liver and kidney related impacts in addition to hepatocyte hypertrophy from 4 and 13 week gavage studies using rats at doses above 75 mg/kg bw/day. No longer-term studies are available.

Studies on the mutagenic activity of ethylbenzene to bacteria, insects and mammalian cells have reported negative results.

Ethylbenzene is classified as Group 2B (possibly carcinogenic to humans) by the International Agency for Research on Cancer (IARC, 2000).

DERIVATION OF GUIDELINE

The USEPA (2009) has set a drinking water guideline of 0.7 mg/L for ethylbenzene, while the WHO (2011) proposes a guideline of 0.3 mg/L.

The health-based guideline value for ethylbenzene in drinking water was determined as follows:

$$0.3 \text{ mg/L} = \frac{75 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

Where:

- 75 mg/kg body weight per day is the no effect level based on a 4 and 13 week gavage study using rats (Mellert *et al.* 2007).
- 70 kg is the average weight of an adult
- 0.1 is the proportion of total daily intake attributable to the consumption of water
- 2 L/day is the average amount of water consumed by an adult
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for the limited data and short duration of the study)

This health-based value exceeds the taste and odour threshold of 0.003 mg/L for ethylbenzene in water.

The WHO drinking water guideline value is the same (0.3 mg/L) but is based on a no effect level of 136 mg/kg bw/d for hepatoxicity and nephrotoxicity in a limited 6-month study with rats (WHO, 2011).

REFERENCES

American Public Health Association (APHA), American Water Works Association (AWWA) and Water Environment Federation (WEF) (2012). Standard Methods for the Examination of Water and Wastewater, 22nd Edition. Eds. Rice EW, Baird RB, Eaton AD and Clesceri LS.

Arvin, E., Engelsen, P. and Sebber, U. (2004) Biodegradation of gasoline compounds (BTEX) in a water

works sand filter. *Water Science & Technology: Water Supply*, **4**(5-6), 29-33.

ATSDR (2010). Toxicological profile for ethylbenzene.US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, USA.

do Rego, E. C. P. and Netto, A. D. P. (2007) PAHs and BTEX in groundwater of gasoline stations from Rio de Janeiro City, Brazil. *B. Environ. Contam. Tox.*, **79**(6), 660-664.

IARC (International Agency for Research on Cancer) (2000) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, volume 77: Some industrial chemicals. Lyon, France.

IPCS (1996). Environmental Health Criteria 186: Ethylbenzene. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland.

Karaconji, B., Skender, L. and Karacic, V. (2006) Benzene, toluene, ethylbenzene, and isomeric xylenes in various water samples in Croatia. *B. Environ. Contam. Tox.*, **76**(3), 458-462.

Kuo, H. W., Chiang, T. F., Lo, L. I., Lai, J. S., Chan, C. C. and Wang, J. D. (1997) VOC concentration in Taiwan's household drinking water. *Sci. Total Environ.*, **208**(1-2), 41-47.

Lesage, S., Hao, X., and Kent, S. (1997) Distinguishing Natural Hydrocarbons from Anthropogenic Contamination in Ground Water. *Groundwater*, 35(1), 149-160

Leusch, F. and Bartkow, M. (2010) A short primer on benzene, toluene, ethylbenzene and xylenes (BTEX) in the environment and in hydraulic fracturing fluids http://www.ehp.qld.gov.au/management/coal-seam-gas/pdf/btex-report.pdf (accessed 19 March 2013)

Mellert W., Deckhardt K., Kaufmann W and van Ranvenzwaay B. (2007) Ethylbenzene: 4- and 13-week rat oral toxicity. Arch Toxicology 81: 361-370

Schmidt, T. C., Haderlein, S. B., Pfister, R. and Forster, R. (2004) Occurrence and fate modeling of MTBE and BTEX compounds in a Swiss Lake used as drinking water supply. *Water Res.*, **38**(6), 1520-1529.

Sedran, M. A., Pruden, A., Wilson, G. J., Suidan, M. T. and Venosa, A. D. (2004) Biodegradation of methyl tert-butyl ether and BTEX at varying hydraulic retention times. *Water Environ. Res.*, **76**(1), 47-55.

USEPA (2009) National Primary Drinking Water Regulations. United States Environmental Protection Agency, Washington, DC, USA.

Volk, H., Pinetown, C., Johnston,W. (2011) A desktop study of the occurrence of total petroleum hydrocarbon (TPH) and partially water-soluble organic compounds in Permian coals and associated coal seam groundwater. CSIRO Petroleum and Geothermal Research Portfolio Report EP-13-09-11-11 **2011** (Bentley, WA, Australia). Available at http://www.agl.com.au/Downloads/Literature%20Review% (accessed 19 March 2013)Williams, P. R. D., Benton, L. and Sheehan, P. J. (2004) The risk of MTBE relative to other VOCs in public drinking water in California. *Risk Anal.*, **24**(3), 621-634.

WHO (2011) Guidelines for Drinking Water Quality, 4th Edition. World Health Organisation, Geneva, Switzerland.

Wolf, M. A., Rowe, V. K., McCollister, D. D., Hollingsworth, R. L. and Oyen, F. (1956) Toxicology studies of certain alkylated benzenes and benzene: experiments on laboratory animals. *AMA Archives of Industrial Health*, **14**, 387–398.

Zein, M. M., Suidan, M. T. and Venosa, A. D. (2006) Bioremediation of groundwater contaminated with gasoline hydrocarbons and oxygenates using a membrane-based reactor. *Environ. Sci. Technol.*, **40**(6), 1997-2003.

NOTE: Important general information is contained in PART II, Chapter 6

Ethylenediamine tetraacetic acid (EDTA)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of ethylenediamine tetraacetic acid in drinking water (as the free acid) should not exceed 0.25 mg/L.

GENERAL DESCRIPTION

EDTA is a metal-complexing agent and may act to mobilise some heavy metals in the environment. It has occasionally been detected in drinking water supplies overseas at concentrations of up to 0.9 mg/L, but usually less than 0.1 mg/L.

EDTA is used widely in industry and agriculture. It is used in laundry detergents, water softening, electroplating, textile and paper production, as a food additive, and in cosmetics. Most of these uses will result in the release of EDTA to the aquatic environment. It is also used as a drug in chelation therapy, particularly in cases involving lead poisoning.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

EDTA has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

There are no published reports on methods for the removal of EDTA from drinking water, although it may be oxidised by ozone.

MEASUREMENT

EDTA can be analysed by potentiometric stripping analysis (Fayyad *et al.* 1988). The limit of determination is 0.001 mg/L.

HEALTH CONSIDERATIONS

EDTA is poorly absorbed in the gut and does not form any significant metabolites. It does not accumulate in the body.

There is considerable clinical experience in the use of EDTA for the treatment of heavy metal poisoning.

Long-term feeding studies with rats and dogs reported no interference to mineral metabolism. Results from other studies have been affected by the formation of zinc complexes in the gastrointestinal tract, which prevents the zinc from being absorbed.

DERIVATION OF GUIDELINE

The guideline value for EDTA (as the free acid) in drinking water was determined as follows:

$$0.25 \text{ mg/L} = \frac{1.9 \text{ mg/kg body weight per day} \times 13 \text{ kg} \times 0.1}{1 \text{ L/day} \times 10}$$

where:

- 1.9 mg/kg body weight per day is the amount of EDTA that can be consumed from all sources per day without adverse effects (WHO 2003).
- 13 kg is the average weight of a child at 2 years of age (this value was used because of the possibility of complexation of zinc, an essential element for humans, and the need to protect the most sensitive group).
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 1 L/day is the average amount of water consumed by a 2-year-old child.
- 10 is a safety factor to reflect the fact that the data for EDTA are relatively old (the World Health Organization assessment was dated 1974), and concern over zinc complexation.

The World Health Organization guideline value of 0.2 mg/L was based on a child body weight of 10 kg. The difference in guideline values is not significant.

REFERENCES

Fayyad M, Tutunji M, Taha Z (1988). Indirect trace determination of EDTA in waters by potentiometric stripping analysis. *Analytical Letters*, 21:1425–1432.

WHO (World Health Organization) (2003). ***Edetic acid (EDTA) in drinking-water. Background document for preparation of WHO Guidelines for drinking-water quality***. WHO, Geneva (WHO/SDE/WSH/03.04/58).

NOTE: Important general information is contained in PART II, Chapter 6

Etridiazole

(endorsed 2011)

GUIDELINE

Based on human health concerns, etridiazole in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Etridiazole (CAS 2593-15-9) belongs to the thiazole fungicide class of chemicals. Other pesticides in this class include thiabendazole (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, etridiazole would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Etridiazole is a broad-spectrum fungicide used for the control of fungi contamination on seeds, roots and stems in turf crops, non fruit-bearing young trees, cotton and ornamental flower crops.

There are currently products registered in Australia that contain etridiazole. These products are intended for professional use. They are applied by spraying or direct incorporation into soil for cotton, non-food producing trees, turf, or ornamental flower crops. They are available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to etridiazole and its metabolites is through residues on treated non-food crops, such as turf and flowers. Residue levels in crops produced according to good agricultural practice are generally low.

Agricultural use of etridiazole may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on etridiazole occurrence in Australian drinking water supplies were found. Estimated concentrations in groundwater from typical use of etridiazole on golf courses indicate that levels in groundwater are not likely to exceed 0.93 µg/L, and in surface water are not likely to exceed 32.3 µg/L for long-term exposure. Both these estimated concentrations are below guideline values indicating low concern (USEPA 2000).

TREATMENT OF DRINKING WATER

No specific data on the treatment of etridiazole in drinking water have been identified.

MEASUREMENT

Etridiazole can be determined by solid-phase extraction followed by gas chromatography–mass spectrometry (USEPA Method 525.2). Typical limit of quantitation (LOQ) is 0.1 µg/L (Munch 1995). Continuous flow micro-extraction combined with high-performance liquid chromatography and ultraviolet detection can achieve a LOQ of <4 ng/mL (He *et al*. 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for etridiazole is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.9 mg/kg bw/day from a long-term (1-year dietary) study. The NOEL is based on decreased bodyweight gain and increased serum ALP activity in dogs. The ADI incorporates a safety factor of 100 and it was established in 1991. Before this, no ADI for etridiazole had been set in Australia.

The previous *Australian Drinking Water Guidelines* health value was also 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Etridiazole is rapidly absorbed from the gastrointestinal tract, followed by wide tissue distribution, including body fat. It is extensively metabolised, with most of the compound excreted rapidly in urine as metabolites (>90% within 24 hours).

Acute effects: Etridiazole has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pig tests.

Short-term effects: Dietary studies in rats and dogs resulted in reduced bodyweight gain and increased liver weight, without associated change in histology, at dose levels of 35 mg/kg bw/day (rat) and 33 mg/kg bw/day (dog).

Long-term effects: Long-term dietary studies have been conducted in mice, rats and dogs and effects observed included reduced bodyweight gain, changes in organ weights relative to bodyweight, and effects on both thyroid and liver in the form of carcinomas. The lowest NOEL in these studies was 2.9 mg/kg bw/day in dogs. This NOEL forms the basis for the current ADI.

Carcinogenicity: An increased incidence of liver and thyroid tumours was observed in rats as a result of severe tissue irritation at the high dose (192 mg/kg bw/day). There was a clear threshold dose for this effect, which is not considered relevant at the low levels to which humans are exposed.

Genotoxicity: Etridiazole is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Etridiazole is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for etridiazole remains unchanged from the value established in 2004, and was determined as follows:

$$0.1 \text{ mg/L} = \frac{2.9 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.9 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

He Y, Lee HK (2006). Continuous flow microextraction combined with high-performance liquid chromatography for the analysis of pesticides in natural waters. *Journal of Chromatography A*, 1122(1-2):7-12.

Munch JW (1995). *Method 525.2 Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and capillarity Columns Gas Chromatography/Mass Spectrometry Revision 2.0*. Cincinnati, Ohio, Environmental Monitoring Systems Laboratory Office of Research and Development USEPA.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2000). *R.E.D. Facts: Etridiazole (Terrazole)*. USEPA – Prevention, Pesticides And Toxic Substances (7508C).

NOTE: Important general information is contained in PART II, Chapter 6

Fenamiphos

(endorsed 2011)

GUIDELINE

Based on human health concerns, fenamiphos in drinking water should not exceed 0.0005 mg/L.

RELATED CHEMICALS

Fenamiphos (CAS 22224-92-6) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes terbufos, chlorpyrifos, ethion, diazinon, fenitrothion, profenofos, trichlorfon, methidathion and acephate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fenamiphos would not be a health concern unless the concentration exceeded 0.0005 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenamiphos is an insecticide used for the control of nematodes and sucking insects (e.g. aphids and thrips) on food and non-food producing crops, and for the control of nematodes in turf.

There are registered products containing fenamiphos in Australia. These products are intended for professional use and are available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to fenamiphos is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of fenamiphos may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Fenamiphos has been found at varying rates in soil (Kookana *et al.* 1997) and in groundwater (Di *et al.* 1995). Point-source fenamiphos pesticide contamination of groundwater from effluent from pest control operators was reported in Perth (Davis *et al.* 1996). Fenamiphos is routinely sampled by drinking water providers but no detections were reported in the literature searched.

TREATMENT OF DRINKING WATER

Reverse osmosis challenger data reduced by more than 99% the initial fenamiphos concentration of 930 µg/L (USEPA 2005). Granular activated carbon is also very efficient in removing trace organic substance including fenamiphos.

MEASUREMENT

Fenamiphos can be extracted from water by liquid/liquid extraction or solid phase extraction. The extract is concentrated and analysed by gas chromatography (GC) coupled with a nitrogen phosphorus detector (NPD) and flame photometric detector. The method can achieve a limit of quantitation (LOQ) of 0.05 µg/L (López-Blanco *et al.* 2006). Solid-phase extraction (SPE) and GC with NPD or mass spectrometry in the selected-ion monitoring mode detection can achieve a LOQ of 0.08–0.60 µg/L, and 0.03–0.13 µg/L, repectively (Psathaki *et al.* 1994). SPE followed by thermospray liquid chromatography–mass spectrometry can achieve a LOQ of 0.012 µg/L (Lacorte *et al.* 1995).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fenamiphos is 0.0001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.014 mg/kg bw/day from a 2-year dietary study in dogs. The NOEL is based on inhibition of plasma cholinesterase activity. The ADI incorporates a safety factor of 100 and it was established in 2005. This ADI is supported by a NOEL of 0.011 mg/kg bw/day based on plasma cholinesterase inhibition in a 6-month dietary dog study.

The acute reference dose (ARfD) of 0.003 mg/kg bw/day for fenamiphos was established in 2005, based on a NOEL of 0.25 mg/kg bw/day from an acute oral toxicity study in dogs. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.0003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Fenamiphos is extensively and readily absorbed from the gastrointestinal tract, with wide distribution in tissues. Metabolism is extensive, and proceeds via sulfoxidation and phenylation pathways. Excretion is predominantly via the urine and to a lesser extent via the faeces, and is complete within 48 hours.

Acute effects: Fenamiphos has very high acute oral toxicity and high acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 15-22 day dermal toxicity studies in rats and rabbits, plasma cholinesterase activity was decreased at 2.5 mg/kg bw/day in rabbits and at 40 mg/kg bw/day in rats.

In three-month oral toxicity studies in rats and dogs, plasma cholinesterase activity was decreased at 0.8 mg/kg bw/day in rats and at 0.05 mg/kg bw/day in dogs. Red blood cell cholinesterase activity was decreased at 1.6 mg/kg bw/day in rats and at 0.125 mg/kg bw/day in dogs. Clinical symptoms of cholinesterase inhibition were observed at 3.2 mg/kg bw/day in rats and at 0.45 mg/kg bw/day in dogs.

Long-term effects: In long-term dietary studies in mice, rats and dogs, plasma, red blood cell cholinesterase activity was decreased at 0.09 mg/kg bw/day in rats and dogs. In rats, brain cholinesterase activity was decreased and clinical symptoms of cholinesterase inhibition were observed at 2.45 mg/kg bw/day. In dogs, other signs of general toxicity were observed at 0.308 mg/kg bw/day. In mice, bodyweight decrease only was observed at 7.4 mg/kg bw/day. The lowest overall NOEL was 0.014 mg/kg bw/day in dogs. This NOEL is the basis for the current ADI.

NOTE: Important general information is contained in PART II, Chapter 6

Carcinogenicity: Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for fenamiphos.

Genotoxicity: Fenamiphos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and a developmental toxicity study in rabbits found no evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: A 21-day neurotoxicity study in hens found no evidence for delayed neurotoxicity for fenamiphos.

Poisons Schedule: Fenamiphos is included in Schedule 6 or 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.0005 mg/L for fenamiphos was determined as follows:

$$0.0005 \text{ mg/L} = \frac{0.014 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.014 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for fenamiphos and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Davis GB, Appleyard S (1996). *Organic pollutants from urban developments over the unconfined groundwater system in Perth WA, Groundwater and Land-use Planning, Fremantle, Western Australia*. Perth, Centre for Groundwater Studies, pp 56-65.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Di HJ, Kookana R, Aylmore LAG (1995). Application of a simple model to assess the groundwater contamination potential of pesticides. *Australian Journal of Soil Research,* 33:1031–1040.

Kookana RS, Phang C, Aylmore LAG (1997). Transformation and degradation of fenamiphos nematicide and its metabolites in soils. *Australian Journal of Soil Research,* 35(4):753-762.

NOTE: Important general information is contained in PART II, Chapter 6

Lacorte S, Barcelo D (1995). Determination of organophosphorus pesticides and their transformation products in river waters by automated on-line solid-phase extraction followed by thermospray liquid chromatography-mass spectrometry. *Journal of Chromatogr A,* 712(1):103-12

López-Blanco C, Gómez-Álvarez S, Rey-Garrote M, Cancho-Grande B, Simal-Gándara J (2006). Determination of pesticides by solid phase extraction followed by gas chromatography with nitrogen-phosphorous detection in natural water and comparison with solvent drop microextraction. *Analytical and Bioanalytical Chemistry,* 384(4):1002-1006.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Psathaki M, Manoussaridou E, Stephanou EG (1994). Determination of organophosphorus and triazine pesticides in ground- and drinking water by solid-phase extraction and gas chromatography with nitrogen-phosphorus or mass spectrometric detection. *Journal of Chromatography A,* 667(1-2):241-8.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2005). *Point of use drinking water treatment system application: Removal of chemical contaminants in drinking water: Reverse osmosis*. The Environmental Technology Verification Program, USEPA.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Fenarimol

(endorsed 2011)

GUIDELINE

Based on human health concerns, fenarimol in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Fenarimol (CAS 60168-88-9) belongs to the pyrimidine class of fungicides. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fenarimol would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenarimol is a fungicide for the control of black spot and powdery mildew in apple and pear agricultural crops.

There is at least one registered product containing fenarimol in Australia. Fenarimol products are intended for professional use and are available as concentrated solutions to be applied in diluted form to foliage of target plants using ground and hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to fenarimol and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of fenarimol may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on occurrence of fenarimol in Australian waters could be found. In the USA, the estimated environmental concentration in surface water was 0.026 mg/L when not in use for residential applications (USEPA 2002).

TREATMENT OF DRINKING WATER

No specific data on the treatment of fenarimol in drinking water have been identified.

MEASUREMENT

Fenarimol can be measured by gas chromatography with alkali flame detector, with a detection limit of 4 µg/L (USEPA Method 633.1).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fenarimol is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a reproduction study and a long-term (2-year) dietary study in rats. The NOEL is based on liver toxicity in the form of fatty changes in the liver, hepatic nodules, and increased blood glucose in the long-term studies, and increased gestation time in the reproduction studies. The ADI incorporates a safety factor of 100, and was established in 1990.

The previous ADI of 0.025 mg/kg bw was established in 1982, based on a NOEL of 2.5 mg/kg bw/day in a long-term dietary study in rats. The ADI was amended to its present level after submission of reproduction and long-term studies demonstrating lower overall NOELs.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Fenarimol was poorly absorbed from the gastrointestinal tract of rats. The absorbed fraction was extensively metabolised (>30 metabolites) with excretion mostly via the faeces within 7 days.

Acute effects: Fenarimol has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Short-term (2-3 week) dietary studies in rats and mice reported increased P-450 liver enzymes at 20 mg/kg bw/day in mice, and increased relative liver weight and centrilobular hypertrophy at 40 mg/kg bw/day in mice and rats. Similar changes were reported in 3-month dietary studies in mice, rats and dogs.

Long-term effects: Long-term (2-year) dietary studies were conducted in mice and rats. In rats, there were fatty changes in the liver, hepatic nodules, and increased blood glucose at doses of 5 mg/kg bw/day. The lowest NOEL was 1.3 mg/kg bw/day in rats, and this NOEL is partly the basis for the current ADI.

Carcinogenicity: Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for fenarimol.

Genotoxicity: Fenarimol is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats reported an increased gestation period at doses of 5 mg/kg bw/day, and decreased pregnancy rate and litter size at doses of 13 mg/kg bw/day. The NOEL was 1.07 mg/kg bw/day and this NOEL is partly the basis for the current ADI. In developmental toxicity studies in rats and rabbits, there was no evidence of effects on foetal development.

Poisons Schedule: Fenarimol is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for fenarimol was determined as follows:

$$0.04 \text{ mg/L} = \frac{1.07 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.07 mg/kg bw/day is the NOEL based on long-term (2-year) dietary studies, and a 3-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2002). Report on FQPA Tolerance Reassessment Progress and Risk Management Decision. USEPA. [http://www.epa.gov/oppsrrd1/reregistration/REDs/fenarimol_tred.pdf]

USEPA Method 633.1. The Determination of Neutral Nitrogen-Containing Pesticides in Municipal and Industrial Wastewaters. United States Environmental Protection Agency.

NOTE: Important general information is contained in PART II, Chapter 6

Fenchlorphos

(endorsed 2011)

GUIDELINE

The health concerns associated with fenchlorphos have not been fully evaluated and therefore a health value for fenchlorphos in drinking water cannot be set.

RELATED CHEMICALS

Fenchlorphos (CAS 299-84-3) belongs to the organophosphate class of chemicals. Other pesticides in this class include dichlorvos, profenofos and acephate (Tomlin 2006).

HUMAN RISK STATEMENT

There are currently insufficient data on which to base a human risk statement.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenchlorphos is an insecticide formerly used for the control of cockroaches and flies in animal houses, dairies, food storages areas, food containers and packaging, food processing equipment, and for the control of insects in a variety of agricultural crops.

There are no registered products containing fenchlorphos in Australia, but de-registered compounds may still be detected in water. Previously registered products were intended for professional use.

Exposure sources: If used in the future, the main source of public exposure to fenchlorphos would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of fenchlorphos in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data on the occurrence of fenchlorphos in Australian waters could be found.

TREATMENT OF DRINKING WATER

No data on the removal efficacy of drinking water treatment for fenchlorphos could be found.

MEASUREMENT

Fenchlorphos can be measured by routine gas chromatography mass spectrometry analysis with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

No acceptable daily intake (ADI) or acute reference dose (ARfD) values have been established for fenchlorphos.

The previous ADI of 0.01 mg/kg bw was set by the World Health Organization in 1968 (IPCS 1968). The basis of this ADI is unknown and it was removed in 2003.

An *Australian Drinking Water Guidelines* health value of 0.03 mg/L was set in 1996 (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: No studies have been evaluated in Australia.

Short-term/long-term effects: No studies have been evaluated in Australia.

Carcinogenicity: No studies have been evaluated in Australia.

Genotoxicity: No studies have been evaluated in Australia.

Reproductive and developmental effects: No studies have been evaluated in Australia.

Poisons Schedule: Fenchlorphos is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

There are currently insufficient data on which to establish a health-based guideline for fenchlorphos in drinking water in Australia.

REFERENCES

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

IPCS (International Programme on Chemical Safety) (1968). INCHEM: WHO/FOOD ADD./69.35. Evaluations of some pesticide residues in food. The monographs. Fenchlorphos. [www.inchem.org/documents/jmpr/jmpmono/v068pr21.htm].

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Fenitrothion

(endorsed 2011)

GUIDELINE

Based on human health concerns, fenitrothion in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Fenitrothion (CAS 122-14-5) belongs to the organophosphate class of chemicals. There are many other pesticides in this class including fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fenitrothion would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenitrothion is an insecticide for the control of a variety of pests (grasshoppers, locusts, beetles, weevils) on crops, fruits, vegetables and pastures. It is also used to treat flour mills, grain storage facilities (prior to storage) and broiler poultry houses (before restocking).

There are registered products containing fenitrothion in Australia, all of which are intended for professional use. All products are liquid concentrates which are diluted and then sprayed. Ground spraying can be done using air-assisted, misting, electrostatic and boom sprayers. Aerial applications are also used, particularly for large scale locust control, where ultra-low volume (ULV) application is common. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to fenitrothion is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of fenitrothion may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on fenitrothion in Australian drinking waters. In the 1981 spruce budworm spray program in Canada, the concentrations of fenitrothion residues detected in water were low (maximum 1.30 µg/L), and post-spray samples did not contain detectable concentrations (<0.01 µg/L) (Mallett and Cassista 1984, cited in WHO 2004a).

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of fenitrothion in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Fenitrothion can be measured by routine gas chromatography with mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fenitrothion is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a 1-year dietary study in dogs. This NOEL is based on plasma cholinesterase inhibition. The ADI incorporates a safety factor of 100 and was established in 1997. The previous ADI was 0.003 mg/kg bw/day based on a NOEL of 0.3 mg/kg bw/day from a 92-week rat study.

The acute reference dose (ARfD) of 0.03 mg/kg bw/day for fenitrothion was established in 2000, based on a NOEL of 0.33 mg/kg bw/day from a single dose study in humans for plasma and red blood cell cholinesterase inhibition. The ARfD incorporates a safety factor of 10 (for intraspecies variation).

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Following oral administration, fenitrothion is readily absorbed from the gastrointestinal tract in all species. It is initially metabolised in the liver to fenitrooxon, then to other metabolites, which are excreted mainly via the urine. The main metabolites are desmethylfenitrothion, desmethylfenitrooxon and 3-methyl-4-nitrophenol (both free and conjugated with sulfate or βglucuronic acid).

Acute effects: Fenitrothion has low to moderate acute oral and dermal toxicity. It does not cause skin sensitisation.

Short-term effects: Short-term studies in rats reported decreased plasma, brain and red blood cell (RBC) cholinesterase activity and symptoms indicative of nervous system toxicity. A decrease in RBC cholinesterase was observed in a 4-week study at dose levels above 1 mg/kg bw/day.

Long-term effects: Long-term dietary studies in rodents, dogs and monkeys also showed the main effect to be on the nervous system, measured as inhibition of cholinesterase activity. A 1-year dietary study in dogs reported a decrease in plasma cholinesterase inhibition at 0.3 mg/kg bw/day and above. The NOEL of 0.2 mg/kg bw/day in this study is the basis for the current ADI.

Carcinogenicity: Based on long-term dietary studies, there is no evidence of carcinogenicity for fenitrothion.

Genotoxicity: Fenitrothion is not considered to be genotoxic, based on short-term *in vitro* and *in vivo* studies (JMPR 2000).

Reproductive and developmental effects: Reproduction studies in rats and developmental studies in rats, rabbits and mice reported no evidence of effects on reproduction, no developmental delays and no evidence of teratogenicity due to fenitrothion.

Neurotoxicity: There was no evidence of delayed neurotoxicity in hens.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Fenitrothion is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for fenitrothion was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for fenitrothion and it is included in the list of agricultural chemicals for which a guideline value has not been established because it "occurs in drinking water at concentrations well below those at which toxic effects may occur" (WHO 2004b).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) T***he Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

JMPR (Joint Meeting on Pesticide Residues) (2000). Pesticide residues in food: Fenitrothion.

Mallett VN, Cassista A (1984). Fenitrothion residue survey in relation to the 1981 spruce budworm spray program in New Brunswick. *Canada. Bulletin of Environmental Contamination and Toxicology*, 32:65–74.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004a). Fenitrothion in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. The World Health Organization.

WHO (World Health Organization) (2004b). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Fenthion

(endorsed 2011)

GUIDELINE

Based on human health concerns, fenthion in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Fenthion (CAS 55-38-9) is in the organophosphate chemical class. There are many pesticides in this group, including chlorpyrifos, diazinon, dichlorvos, ethion, parathion and profenofos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fenthion would not be a health concern unless the concentration exceeded 0.007 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenthion is an insecticide used for pre- and post-harvest treatment of various fruits and vegetables. It is also used as a commercial and domestic insecticide and as an ectoparasiticide in cattle and dogs.

There are registered products containing fenthion in Australia. These products include a spot-on treatment for cattle, an insecticide dust and spray, as well as paint, paste and gels for control of pest birds. Some products are available for use in the home garden and as household insecticides. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to fenthion and its metabolites are use of household insecticide products, and residues in food. Residue levels in crops grown according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data for fenthion in Australian drinking water are available. The highest predicted surface water concentration in the USA was 1.3 µg/L (USEPA 2001). The highest concentration reported in surface water in Japan was 0.27 µg/L (Tsuda *et al.* 1998). Fenthion concentration in Australian treated sewage was less than 2.4 µg/L (supporting data, NRMMC/EPHC/NHMRC, 2008).

TREATMENT OF DRINKING WATER

Although no empirical data is currently available, it is likely that activated carbon would provide efficient removal for this chemical based on its chemical structure.

MEASUREMENT

Fenthion residues in water can be analaysed by solid-phase extraction–liquid chromatography with electrospray tandem mass spectrometry, with a detection limit of 0.021 µg/L (Hernandez *et al*, 2001); or by solid-phase extraction–gas chromatography–mass spectrometry, with a detection limit of 0.13 µg/L (Psathaki *et al*, 1994).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fenthion is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.02 mg/kg bw/day from a 28-day human study showing cholinesterase inhibition. The ADI incorporates a safety factor of 10 and was established in 2004.

The acute reference dose (ARfD) of 0.007 mg/kg bw/day for fenthion was established in 2000, based on a NOEL of 0.07 mg/kg bw/day from a 28-day oral study in human volunteers. The ARfD incorporates a safety factor of 10. This NOEL is further supported by a NOEL of 0.07 mg/kg bw from an acute neurotoxicity study in rats.

An *Australian Drinking Water Guidelines* health value has not been previously established for fenthion.

HEALTH CONSIDERATIONS

Metabolism: Fenthion is rapidly absorbed from the gastrointestinal tract and broadly distributed in tissues, particularly in lipid stores. Metabolism is extensive and elimination is almost complete after 48 hours (90% in the urine). There are 3 major urinary metabolites – the oxygen analogue of fenthion and its sulfoxide and sulfone derivatives.

Acute effects: Fenthion has moderate acute oral and dermal toxicity. Symptoms of acute poisoning were indicative of central and peripheral nervous system poisoning and included hyperexcitability, salivation, broncoconstriction, headache, vomiting and other behavioural changes. It is not a skin sensitiser.

Short-term and long-term effects: In short-term and long-term studies in rodents, monkeys and dogs, the main effect was the inhibition of cholinesterase (plasma, brain and red blood cell) at dose levels of 0.04 mg/kg bw/day (mouse) 0.07mg/kg bw/day (monkey), 0.32 mg/kg bw/day (dog) and above.

In a 4-week human volunteer study, there was a dose-related inhibition of plasma cholinesterase at 0.07 mg/kg bw/day. There was no inhibition of red blood cell cholinesterase. The NOEL of 0.02 mg/kg bw/day is the basis for the ADI.

Carcinogenicity: Based on long-term studies in rodents, there is no evidence of carcinogenicity for fenthion.

Genotoxicity: Fenthion is not considered to be genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproductive and developmental effects: In a reproduction study in rats, there was decreased fertility, decreased litter sizes and increased neonatal deaths at high doses. Similarly, developmental studies in rats and rabbits showed increased resorptions and delayed skeletal development as a result of high dose maternotoxicity. In both cases, this was the result of central nervous system effects and well above the likely levels of human exposure.

Neurotoxicity: There was no evidence of delayed neurotoxicity in hen, rat and dog studies.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Fenthion is included in Schedule 5, 6 or 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and the use of the product. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.007 mg/L for fenthion was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.02 \text{ mg/kg bodyweight /day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.02 mg/kg bw/day is the NOEL based on a short-term (28-day) study in human volunteers.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is a safety factor applied to the NOEL derived a human study to account for variation within the human population (intraspecies variation).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hernandez F, Sancho JV, Pozo O, Lara A, Pitarch E (2001). Rapid direct determination of pesticides and metabolites in environmental water samples at sub-ug/l level by on-line solid-phase extraction–liquid chromatography–electrospray tandem mass spectrometry. *Journal of Chromatography, A* 939(1-2): 1-11.

NRMMC (Natural Resource Management Ministerial Council), EPHC (Environment Protection and Heritage Council), NHMRC (National Health and Medical Research Council) (2008). *Australian Guidelines for Water Recycling: Managing health and environmental risks (Phase 2) - Augmentation of drinking water supplies*. NRMMC, EPHC, NHMRC.

Psathaki M, Manoussaridou E, Stephanou EG (1994). Determination of organophosphorus and triazine pesticides in ground- and drinking water by solid-phase extraction and gas chromatography with nitrogen-phosphorus or mass spectrometric detection. *Journal of Chromatography A*, 667(1-2): 241-248.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Tsuda T, Kojima M, Harada H, Nakajima A, Aoki S (1998). Pesticides and their oxidation products in water and fish from rivers flowing into Lake Biwa. *Bulletin of Environmental Contamination and Toxicology,* 60: 151-158.

USEPA (United States Environmental Protection Agency) (2001). Interim Reregistration Eligibility Decision for Fenthion. EPA 738-R-00-013. USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Fenvalerate

(endorsed 2011)

GUIDELINE

Based on human health concerns, fenvalerate in drinking water should not exceed 0.06 mg/L.

RELATED CHEMICALS

Fenvalerate (CAS 51630-58-1) belongs to the pyrethroid class of chemicals. This is a large chemical group and includes the closely related esfenvalerate (esterified form), cyfluthrin, permethrin, and flucythrinate.

There are four optical isomers of fenvalerate and esfenvalerate (SS, SR, RS, RR). The SS isomer is responsible for the insecticidal activity of these compounds. Fenvalerate contains around 20% as the SS form, while esfenvalerate is highly enriched in this form. Most of the toxicity of fenvalerate is caused by the RS isomer (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fenvalerate would not be a health concern unless the concentration exceeded 0.06 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fenvalerate is an insecticide used for the control of buffalo fly and *Culicoides* midge on cattle and horses.

There is at least one registered product containing fenvalerate in Australia. Fenvalerate products are intended for professional use, to be diluted and applied by handspray to the backline of cattle and horses.

Exposure sources: The main source of public exposure to fenvalerate and its metabolites is residues in meat. Residue levels in food produced according to good agricultural practice are generally low.

The veterinary use of fenvalerate provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Esfenvalerate, a sterified form of fenvalerate, has been detected in 2004-2006 in passive samples and spot water samples from the Goulburn Murray Water irrigation supply channels in Victoria. In October 2005 the Torrumbarry Irrigation Area Kerang Town Channel reported an esfenvalerate concentration of 65 µg/L (Rose and Kibria 2006).

TREATMENT OF DRINKING WATER

No specific data on the treatment of fenvalerate in drinking water have been identified.

MEASUREMENT

After extraction with n-hexane or dichloromethane, fenvalerate can be analysed by gas chromatography equipped with an electron capture detector (ECD) or with a mass selective detector (MSD). A limit of quantitation (LOQ) of 0.05 µg/L using the ECD and 0.1 µg/L using the MSD have been reported (California Department of Food and Agriculture 2000, Xue *et al*. 2005). Fenvalerate can also be extracted and pre-concentrated by micro liquid–liquid extraction and analysed by gas chromatography–mass spectrometry (GC-MS) with selected ion monitoring, with a reported LOQ in the ng/L concentrations (Fernández-Gutiérrez *et al*. 1998). A modification of the EPA method 8270: GC-MS electron ionisation, using a narrow range selected ion scan to analyse pyrethroids, can achieve a LOQ of 5 ng/L in water (Heines and Halpin 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fenvalerate is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.7 mg/kg bw/day from a 2-year dietary mouse study. The NOEL is based on micro-nodular inflammation in the liver, lymph nodes, and spleen. The ADI incorporates a safety factor of 100 and was established in 1987.

The previous ADI of 0.04 mg/kg bw was set in 1985 based on a NOEL of 3.5 mg/kg bw/day from a 20-month dietary study in mice. The ADI was updated after submission of an additional long-term study demonstrating a lower overall NOEL.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Fenvalerate is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, and is rapidly excreted in the urine, almost completely within 48 hours.

Acute effects: Fenvalerate has moderate acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: A 90-day and a 15-month dietary study in rats reported decreased bodyweight gain and decreased haemoglobin levels at 7.5 mg/kg bw/day and above. A 6-month dietary study in dogs reported behavioural effects including tremors and lack of coordination at 6.25 mg/kg bw/day.

Long-term effects: Long-term (2-year) dietary studies in mice and rats reported micro-nodular inflammatory changes in the liver, lymph nodes, and spleen of mice at doses of 9 mg/kg bw/day, and decreased bodyweight gain and decreased haemoglobin levels in rats at higher doses. The lowest overall NOEL was 1.7 mg/kg bw/day in mice. This NOEL is the basis for the current ADI.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for fenvalerate. The micro-nodular changes noted in mouse tissues were considered evidence of localised tissue inflammation rather than cancer.

Genotoxicity: Fenvalerate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal developmental.

Neurotoxicity: Fenvalerate has some potential for neurotoxicity, but only at levels much greater than likely human exposures. A single oral dose of 500 mg/kg bw in rats caused axonal and myelin lesions, with a NOEL of 200 mg/kg bw/day. In a 15-month dietary study in rats, the NOEL was 75 mg/kg bw/day based on sciatic nerve damage at higher doses. Doses up to 1 g/kg bw did not caused delayed neurotoxicity in hens.

Poisons Schedule: Fenvalerate is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.06 mg/L for fenvalerate was determined as follows:

$$0.06 \text{ mg/L} = \frac{1.7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.7 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

Note: This calculated health-based guideline exceeds the normal aqueous solubility of fenvalerate.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

California Department of Food and Agriculture (2000). Method #: EM 52.5 Determination of Permethrin and Esfenvalerate/Fenvalerate in sediment water. Center for Analytical Chemistry, Environmental Monitoring Section, Sacramento, CA.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra. (http://www.comlaw.gov.au/Details/F2010L02386/Download)

Fernández-Gutiérrez, A, Martínez-Vidal JL, Arrebola-Liébanas J, Gonzalez-Casado J, Vílchez JL (1998). Determination of endosulfan and some pyrethroids in waters by micro liquid-liquid extraction and GC-MS. *Fresenius' Journal of Analytical Chemistry,* 360:568-57.

Heines RL, Halpin PW (2006). Pyrethroid pesticides by modified EPA 8270. Caltest Analytical Laboratory [http://www.caltestlabs.com/PyrethroidPesticides.php].

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NOTE: Important general information is contained in PART II, Chapter 6

Rose G, Kibria G (2006). *Pesticide Monitoring in Goulburn-Murray Waters Irrigation Supply Channels Covering the Six Irrigation Areas.* [2004 -2006 Irrigation Season Study Report]. Werribee, Victoria, Department of Primary Industries & Goulburn Murray Rural Water Authority.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Xue N, X Xu, Jin Z (2005). Screening 31 endocrine-disrupting pesticides in water and surface sediment samples from Beijing Guanting reservoir. *Chemosphere,* 61(11):1594-606.

NOTE: Important general information is contained in PART II, Chapter 6

Fipronil

(endorsed 2011)

GUIDELINE

Based on human health concerns, fipronil in drinking water should not exceed 0.0007 mg/L.

RELATED CHEMICALS

Fipronil (CAS 120068-37-3) belongs to the phenylpyrazole class of chemicals. Another pesticide in this class is ethiprole (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fipronil would not be a health concern unless the concentration exceeded 0.0007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fipronil is a broad-spectrum insecticide that has a range of agricultural uses, including seed dressings, control of pests in bananas, cotton, sorghum, vegetables and turf. Fipronil is also included in insect baits for household and commercial uses, and in home veterinary products for cats and dogs.

There are registered products containing fipronil in Australia. The products are intended for agricultural, professional, home garden, and household pet use. They are available as sprays, baits, dusts, and gels for control of cockroaches, fruit flies, ants and termites; as concentrated granular formulations for control of insect pests in turf; spot on and spray insecticide treatments for pets; and as liquid concentrates and/or wettable granules for application to seeds, and vegetable and other crops, to be applied in diluted form by ground and aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to fipronil and its metabolites are residues in food.

Agricultural and veterinary use of fipronil may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of fipronil in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific literature on the removal of fipronil has been identified. Jar testing to identify the effectiveness of various removal methods in specific waters is recommended if fipronil is detected.

MEASUREMENT

The practical limit of quantification (LOQ) for fipronil in water is 0.001 mg/L using liquid chromatography with tandem mass spectrometry (Alder *et al.* 2006). This LOQ is slightly above the guideline value.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fipronil is 0.0002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.02 mg/kg bw/day from a 2-year rat dietary study for effects on the nervous system, thyroids and kidneys. The ADI includes a safety factor of 100 and it was initially established in 1994 and reconfirmed in 2006.

The current acute reference dose (ARfD) is 0.02 mg/kg bw, set in 2006 and based on a NOEL of 2.5 mg/kg bw for effects on the nervous system in an acute dietary study in rats. This incorporates a safety factor of 100. The previous ARfD was 0.003 mg/kg bw.

The ADI and ARfD values are a group value covering the parent compound and its main metabolites.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Fipronil is completely absorbed via the gastrointestinal tract and extensively metabolised. Excretion is slow (more than 8 days) via the faeces. Dermal absorption is low.

Acute effects: Fipronil has moderate oral toxicity and low to moderate dermal toxicity. It is not a skin sensitiser in guinea pigs.

Short-term effects: In 4-6 week studies in mice, rats and dogs, effects were observed on the nervous system (muscular incoordination, convulsions and hyperactivity in mice at 6.5 mg/kg bw/day and head nodding in dogs at 10 mg/kg bw/day), and on the liver (at 2.4 mg/kg bw/day and above in mice, and 3.4 mg/kg bw/day in rats) and thyroid (at 3.4 mg/kg bw/day in rats).

In 3-month dietary studies in rats and dogs, there was increased liver and thyroid weight, and associated follicular cell hypertrophy and hyperplasia in rats, and decreased body weight gain in dogs at doses of 2 mg/kg bw/day. Symptoms of nervous system toxicity in dogs (convulsions, tremors, in-coordination) were seen at higher doses.

Long-term effects: In long-term studies in mice, rats and dogs, effects on the nervous system (irritability, hyperactivity and vocalisation) were observed in rats at 0.06 mg/kg bw/day and in dogs (convulsions, facial twitching, and disorientation) at 2 mg/kg bw/day. Nephropathy and increased liver and thyroid weight associated with hypertrophy and hyperplasia were observed in all species. The lowest NOEL was 0.02 mg/kg bw/day in the 2-year rat study, and is the basis for the current ADI.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for fipronil.

Genotoxicity: Fipronil is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproduction and developmental effects: Reproduction studies in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

NOTE: Important general information is contained in PART II, Chapter 6

Neurotoxicity: In a single oral dosing study in rats, there was decreased hind leg splay at 5 mg/kg bw/day. In a 6-day oral dosing study in rats, there was decreased levels of serotonin in the brain at 5 mg/kg bw/day. In a 13-week dietary study in rats, there was an increased incidence in "startled" responses and decreased forelimb strength at 7.2 mg/kg bw/day.

Poisons Schedule: Fipronil is either exempt from scheduling or is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard) (DoHA 2010)., depending on concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.0007 mg/L for fipronil was determined as follows:

$$0.0007 \text{ mg/L} = \frac{0.02 \text{ mg/kg bodyweight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.02 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from a study conducted in rats. The safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Alder L, Greulich K, Kempe G, Vieth B (2006). Residue analysis of 500 high priority pesticides: better by GC-MS or LC-MS/MS? *Mass Spectrometry Reviews*, 25(6):838-65.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Flamprop-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, flamprop-methyl in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Flamprop-methyl and its isomer, flamprop-m-methyl (CAS 52756-25-9) belong to the arylaminopropionic acid class of chemicals. There are no other pesticides in this class registered for use in Australia (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, flamprop-methyl would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Flamprop-methyl is a herbicide for the control of wild-oat weeds in wheat and triticale crops.

There are currently products registered in Australia that contain flamprop-methyl, all as the m-methyl isomer. Flamprop-methyl products are intended for professional use. Products are emuslifiable concentrate and liquid formulations intended to be sprayed in diluted form onto crops by aerial, boom spray, and hand-held spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to flamprop-methyl and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of flamprop-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on flamprop-methyl occurrence in Australian drinking water supplies have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of flamprop-methyl in drinking water have been identified.

MEASUREMENT

Flamprop-methyl in drinking water can be determined by gas chromatography (GC) or gas-liquid chromatography after abstraction followed by electron capture detection. Determination by GC or GC with mass spectrometry can achieve limits of detection of 0.1 mg/L (Hirahara *et al*. 2005). Flamprop-methyl can also be analysed by high-performance liquid chromatography with ultraviolet detection.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for flamprop-methyl is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.125 mg/kg bw/day from a long-term study. The NOEL is based on liver hypertrophy and increased liver weight in a 2-year dietary study. The ADI incorporates a safety factor of 100. It was established in 1980 and reaffirmed in 1991.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: In rats, flamprop-methyl is readily absorbed and extensively metabolised, and completely excreted (>95-97%) within 48 hours of dosing. Excretion is mostly via faeces.

Acute effects: Flamprop-methyl and its m-isomer have low oral and dermal acute toxicity. Flamprop-methyl is not a skin sensitiser in guinea-pig tests.

Short-term effects: In a 5-week dietary study in rats, increased liver weight was reported at 2.5 mg/kg bw/day and above, and increased caecum weight at 25 mg/kg bw/day. In a 6-week dietary study in dogs, increased liver weights and associated increases in liver enzymes were seen at 125 mg/kg bw/day. The lowest overall NOEL was 0.25 mg/kg bw/day based on increased liver weights in rats.

In 90-day dietary studies in rats and dogs, liver changes were observed at 50 mg/kg bw/day in rats, and 2.5 mg/kg bw/day in dogs.

Long-term effects: In 2-year dietary studies in rats and dogs, liver hypertrophy and increased liver weight were seen at 12.5 mg/kg bw/day in rats, and at 2.5 mg/kg bw/day in dogs. The lowest overall NOEL in these studies was 0.125 mg/kg bw/day in rats.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for flamprop-methyl.

Genotoxicity: Flamprop-methyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and a developmental study in rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Flamprop-methyl and flamprop-m-methyl are included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 0.004 mg/L for flamprop-methyl was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.125 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.125 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hirahara Y, Kimura M, Inoue T, Uchikawa S, Otani S, Haganuma A, Matsumoto N, Hirata A, Maruyama S, Iizuka T, Ukyo M, Ota M, Hirose H, Suzuki S, Uchida Y (2005). Validation of multiresidue methods for the determination of 186 pesticides in 11 agricultural products using gas chromatography. *Journal of Health Science,* 51(5):617-27.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Fluometuron

(endorsed 2011)

GUIDELINE

Based on human health concerns, fluometuron in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Fluometuron (CAS 2164-17-2) belongs to the phenylurea class of chemicals. Other pesticides in this class include linuron and diuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, fluometuron would not be a health concern unless the concentration exceeded 0.07 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Fluometuron is a herbicide for the control of broad-leaf weeds and grasses in cotton, cereal, citrus and sugar cane crops.

There are currently products registered in Australia that contain fluometuron. Fluometuron products are intended for professional use. They are water-soluble granules, wettable powders, or liquid formulations intended to be diluted and applied by ground spray or aerial spray directly onto soil for pre-emergent treatment, and by hand spray directly at weeds for post-emergent treatment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to fluometuron and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of fluometuron may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on fluometuron occurrence in Australian drinking water supplies were found. In the USA, fluometuron was the most detected pesticide in surface water of the Mississippi river (mean = 2.1 µg/L, median = 0.40 µg/L, maximum = 50 µg/L) (Thurman *et al.* 1998).

TREATMENT OF DRINKING WATER

No specific data on the treatment of fluometuron in drinking water have been identified.

MEASUREMENT

Fluometuron in drinking water can be analysed by solid phase extraction followed by high performance liquid chromatography (HPLC) with diode array detector or post-column photolysis and derivatisation detection. Typical limits of reporting (LOR) with DAD range from ranged from 4 to 40 ng/L (Ruberu *et al*. 2000). HPLC method and measurement with ultraviolet detector can achieve a LOR of 11 µg/L. Photo-induced chemiluminescence has been used for the determination of fluometuron in a quick and continuous procedure (Sa *et al*. 2007). Fluometuron can also be analysed by direct injection onto a liquid chromatography mass spectrometer (LC-MS) instrument in multiple reaction monitoring mode, with a LOR of 10 µg/L.

In addition to the standard approach of gas chromatography with mass spectrometry (GC-MS) or LC-MS for the evaluation of fluometuron in water, the enzyme-linked immunosorbent assay (ELISA) can be. ELISA is portable and there is a good correlation between the test and GC-MS (Thurman *et al*. 1998).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for fluometuron is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2 mg/kg bw/day from a long-term study. The NOEL is based on a 2-year dietary study in mice in which conjunctivitis and decreased bodyweight gain were observed. The ADI incorporates a safety factor of 100 and was established in 1989.

The previous ADI of 0.01 mg/kg bw/day was based on a NOEL of 1 mg/kg bw/day from a reproductive toxicity study in rats for decreased body weight gain and haematological disturbances.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Fluometuron is rapidly absorbed from the gastrointestinal tract (65-80%) and extensively metabolised. Excretion is rapid, being almost complete by 72 hours, and is mainly through urine.

Acute effects: Fluometuron has low acute oral and dermal toxicity. It was not a skin sensitiser in guinea-pig tests.

Short-term effects: In a 21-day dermal study in rabbits, no toxicity was observed at 1000 mg/kg bw/day.

In 90-day dietary studies in rats, mice, and dogs, red blood cell haemolysis and increased spleen weights were reported at 12.5 mg/kg bw/day, and decreased bodyweight gain and congested liver, spleen, and kidney at higher dose levels.

Long-term effects: In long-term dietary studies in rats, mice and dogs, anaemia, increased spleen size, and haemosiderin deposition in spleen and liver were reported at doses of 63 mg/kg bw/day. Decreased food consumption and bodyweight gain were seen at doses of 100 mg/kg bw/day. The lowest overall NOEL was 2 mg/kg bw/day in the mouse study, and this is the basis for the current ADI.

Carcinogenicity: Based on long-term dietary studies in mice and rats, there is no evidence of carcinogenicity for fluometuron.

Genotoxicity: Fluometuron is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Fluometuron is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for fluometuron was determined as follows:

$$0.07 \text{ mg/L} = \frac{2.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.0 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ruberu SR, WM Draper, Perera SK (2000). Multiresidue HPLC methods for phenyl urea herbicides in water. *Journal of Agricultural and Food Chemistry,* 48(9):4109-4115.

Sa F, Lopez Malo D, Martinez Calatayud J (2007). Determination of the herbicide fluometuron by photo-induced chemiluminescence in a continuous-flow multicommutation assembly. *Analytical Letters,* 40(13-15):2872-2885.

Thurman EM, Zimmerman LR, Scribner EA, Coupe RH (1998). Occurrence of cotton pesticides in surface water of the mississippi embayment. US Department of the Interior, US Geological Survey.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Fluoride

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of fluoride in drinking water should not exceed 1.5 mg/L.

GENERAL DESCRIPTION

Fluoride occurs naturally in seawater (1.4 mg/L), soil (up to 300 parts per million) and air (from volcanic gases and industrial pollution). Naturally occurring fluoride concentrations in drinking water depend on the type of soil and rock through which the water drains. Generally, concentrations in surface water are relatively low (<0.1–0.5 mg/L), while water from deeper wells may have quite high concentrations (1–10 mg/L) if the rock formations are fluoride-rich.

Inorganic fluorine compounds are used in aluminium production, as a flux in the steel and glass fibre industries, and in phosphate fertilisers, bricks, tiles and ceramics.

Virtually all foodstuffs contain traces of fluoride. In particular, high amounts can be found in dried tea leaves because of natural concentration by the tea plant. Total daily intake from all sources varies considerably, but has been estimated at 0.46 mg to 5.4 mg, with about 10% coming from unfluoridated drinking water.

Fluoride is used to protect teeth against dental caries. It is present in most brands of toothpaste, and it is often added to drinking water supplies.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In unfluoridated supplies, fluoride concentrations are typically less than 0.1 mg/L, but can range from less than 0.05 mg/L up to 1.5 mg/L, with the higher values reported from groundwater sources.

In fluoridated supplies, the target fluoride concentration is between 0.7 and 1 mg/L, with the lower concentrations applying where the climate is hot, to allow for a higher average consumption of water.

TREATMENT OF DRINKING WATER

Fluoride concentrations in drinking water can be reduced by dilution with other sources, or by using activated alumina or bone char. Conventional coagulation with alum is much less effective.

MEASUREMENT

The fluoride concentration in drinking water can be determined using an ion-specific electrode (APHA Method 4500-F^- Part C 1992). The limit of determination is 0.1 mg/L.

HEALTH CONSIDERATIONS

Because fluoride is widely dispersed in the environment, all living organisms are exposed to it and all tolerate modest amounts. It has been claimed that fluoride is an essential trace element for humans, but this is difficult to establish conclusively, and no data are available on the minimum amount needed. Fluoride is absorbed quickly following ingestion. It is not metabolised, but diffuses passively into all body compartments. About 40% is excreted in urine within 9 hours, and about 50% over 24 hours. Fluoride has an affinity for mineralising tissues of the body: in young people, bone and teeth; in older people, bone. Thus excretion is somewhat greater in adults because they have proportionately less mineralising tissue than children.

Fluoride has been shown to prevent dental caries very effectively, and knowledge of its anti-caries effect came from the observed association of low caries prevalences with naturally occurring fluoride in drinking water (at about 1 mg/L). The NHMRC has extensively reviewed health aspects of fluoride and its prevention of dental disease. Many health authorities around the world recommend fluoridation of public water supplies as an important public health measure.

Concentrations above 1.5 mg/L may disturb tooth mineralisation in children up to about 6 to 8 years, leading to dental fluorosis, a mottling of the teeth which can occasionally occur to an unsightly degree.

Skeletal fluorosis, characterised by hypermineralisation and thus brittle bones, has occurred in association with high fluoride concentrations in drinking water, and also with occupational exposure to fluoride-containing dust. It generally occurs after prolonged exposure (several years) and is reversible: if the exposure is removed, the fluoride levels in bones gradually decline.

Regular consumption of water with fluoride concentrations above about 4 mg/L involves progressively increasing risks of skeletal fluorosis. The United States Environmental Protection Agency has set this level as the maximum acceptable for drinking water: above it, communities are required to lower the fluoride concentration by treatment to remove it, or by dilution.

People with kidney impairment have a lower margin of safety for fluoride intake. Limited data indicate that their fluoride retention may be up to three times normal.

There is no substantiated epidemiological evidence that fluoride or fluoridation causes cancer. One animal study showed an increased incidence of bone tumours in some male rats that were exposed to very high concentrations of fluoride in water, but female rats and mice were not affected.

Tests for mutagenicity with strains of bacteria have been negative. Chromosome aberrations have been reported in tests with mammalian cells but only at extremely high fluoride concentrations.

The International Agency for Research on Cancer has concluded that fluoride is not classifiable as to its carcinogenicity in humans (Group 3, inadequate evidence in humans and in animals) (IARC 1987).

DERIVATION OF GUIDELINE

It was recognised in setting the guideline value of 1.5 mg/L that there is a narrow margin between concentrations producing beneficial effects to teeth and those producing objectionable fluorosis.

The minimum concentration required for a protective effect against dental caries is about 0.5 mg/L, and concentrations around 1 mg/L in temperate climates are optimal for caries prevention. At concentrations between 1.5 and 2 mg/L, mottling of teeth due to dental fluorosis may occur, sometimes to an objectionable degree.

The guideline value of 1.5 mg/L has been set to protect children from the risk of dental fluorosis. If this value is exceeded in circumstances where it is not practical to defluoridate, then parents should be advised to use rainwater or bottled water for children up to about 6 years to limit or prevent dental fluorosis.

The guideline value should not be regarded as a recommended value for fluoridation of water supplies.

REFERENCES

APHA Method 4500-F^- Part C (1992). Fluoride: Ion-selective electrode method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

NHMRC (1991). The effectiveness of water fluoridation. National Health and Medical Research Council, and Department of Health, Housing and Community Services, Canberra.

NOTE: Important general information is contained in PART II, Chapter 6

Flupropanate

(endorsed 2011)

GUIDELINE

Based on human health concerns, flupropanate in drinking water should not exceed 0.009 mg/L.

RELATED CHEMICALS

Flupropanate (CAS 756-09-2) belongs to the halogenated alkanoic acid class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, flupropanate would not be a health concern unless the concentration exceeded 0.009 mg/L. Excursions above this level even for a relatively short period are of concern as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Users: Flupropanate is a herbicide used to control the growth of grass weeds in industrial or pasture land.

There are registered products containing flupropanate in Australia. These are available as concentrated solutions applied as a dilute spray by aerial, ground boom or hand spray techniques. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The potential sources of public exposure to flupropanate and its metabolites are residues in food and drinking water. Flupropanate is not registered for use on food crops and the maximum residue limits are set at the level of detection.

Agricultural use of flupropanate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of flupropanate in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of flupropanate in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

No suitable analytical techniques have been identified, but the use of high performance liquid chromatography with tandem mass spectrometry is expected to be suitable for residue levels of this pesticide in water.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for flupropanate is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day from a 90-day dietary rat study. The NOEL is based on effects on the liver. The ADI incorporates a safety factor of 2000 and it was established in 1987.

An *Australian Drinking Water Guidelines* health guideline has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: No data are available on the absorption, distribution, metabolism or excretion of flupropanate.

Acute effects: Flupropanate has low acute oral and dermal toxicity. Its skin sensitisation potential is unknown.

Short-term effects: A 90-day dietary study in rats reported changes in liver weight at 15 mg/kg bw/day and changes in kidney weight at 50 mg/kg bw/day. The NOEL from the rat study was 5 mg/kg bw/day, and this is the basis of the ADI.

Long-term effects: A 1-year dietary study in mice reported kidney weight changes at 15 mg/kg bw/day and changes in bodyweight gain and liver weight, together with evidence of liver toxicity, at 30 mg/kg bw/day. The study was of poor quality and the NOEL of 7 mg/kg bw/day was not used to establish the ADI.

Carcinogenicity: There are no studies available to assess the potential carcinogenicity of flupropanate.

Genotoxicity: Only short-term bacterial *in vitro* studies were available, and based on these, flupropanate is not considered to be genotoxic; however, no *in vitro* mammalian studies or *in vivo* studies were available.

Reproductive and developmental effects: There were no data available on the reproductive effects of flupropanate. Developmental studies in mice and rats did not show any evidence of effects on foetal development; however, both studies were considered to be of poor quality.

Poisons Schedule: Flupropanate is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.009 mg/L for flupropanate was determined as follows:

$$0.009 \text{ mg/L} = \frac{5 \text{ mg/kg bodyweight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 2000}$$

where:

- 5 mg/kg bw/day is the NOEL based on a medium-term (90-day) dietary study in rats.
- 70 kg is taken as the average weight of an adult.

NOTE: Important general information is contained in PART II, Chapter 6

- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 2000 is a safety factor applied to the NOEL derived from animal studies. The safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variations, and an additional factor of 20 for using a NOEL derived from a medium-term study.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Formaldehyde

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of formaldehyde in drinking water should not exceed 0.5 mg/L.

GENERAL DESCRIPTION

Formaldehyde may be present in drinking water through ozonation of naturally occurring humic material, contamination by accidental spills, or deposition from the atmosphere. Typical concentrations in air are probably in the low parts-per-billion range. Overseas, formaldehyde has been detected in ozonated drinking water at concentrations up to 0.03 mg/L.

Formaldehyde is used industrially in the wood, paper and textile industries. It is also used in the production of a number of chemicals and for the preservation of biological material. It is occasionally used as a disinfectant, sometimes to disinfect water filters. Other sources of exposure include cigarette smoke and food. Formaldehyde is present in almost all common foods, and adult dietary intake is estimated at 11 mg/day. Drinking water would contribute less than 10% of total intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on the concentrations of formaldehyde in Australian drinking waters.

TREATMENT OF DRINKING WATER

There are no published reports on methods for the removal of formaldehyde from drinking water.

MEASUREMENT

Formaldehyde can be determined by formation of the 2,4-dinitro-phenylhydrazone derivative followed by analysis with high performance liquid chromatography with UV detection (Whittle and Rennie 1988). The limit of determination is 0.006 mg/L.

HEALTH CONSIDERATIONS

Formaldehyde is readily absorbed from the gastrointestinal tract and is rapidly metabolised to formic acid and subsequently to carbon dioxide and water.

An extensive review and summary of the human and animal toxicity data for formaldehyde is available (IPCS 1989).

Most human health data are from inhalation studies, where formaldehyde causes irritation of the respiratory tract, and dermal studies, where it causes skin irritation. Formaldehyde has been linked to outbreaks of haemolytic anaemia in patients using improperly serviced dialysis units, where formaldehyde was used to disinfect the units and residual amounts remained in the water filter.

A number of epidemiological studies have looked at the effects of inhalation of formaldehyde. No effects could be directly attributed to long-term occupational exposure, but studies among exposed workers have reported elevated incidences of a number of cancers including nasal, buccal, nasopharyngeal, skin, prostate and colon cancers. The available human evidence indicates that formaldehyde does not have a high carcinogenic potential (IPCS 1989).

In a 2-year drinking water study using rats, severe damage to gastric mucosa was reported only at the highest doses (over 80 mg/kg body weight per day), but no tumours were observed, either in the stomach or at other sites. Other studies have shown similar pathological changes to the stomach, but again only at the highest doses.

There was no evidence of tumour-promoting activity when formaldehyde was applied to mouse skin, but rats inhaling formaldehyde exhibited a markedly increased incidence of cancer of the nasal cavity. Formaldehyde has demonstrated mutagenic activity when applied to cells *in vitro* but not when applied *in vivo*.

The International Agency for Research on Cancer has concluded that formaldehyde is probably carcinogenic to humans (Group 2A, limited human evidence, sufficient animal evidence, based on inhalation studies) (IARC 1987). The weight of evidence indicates that formaldehyde is not carcinogenic by the oral route.

DERIVATION OF GUIDELINE

The guideline value for formaldehyde in drinking water was determined as follows:

$$0.5 \text{ mg/L} = \frac{15 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 15 mg/kg body weight per day is the no-effect level based on a 2-year drinking water study in rats (Til *et al*. 1989).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations). The use of this safety factor was recommended by the NHMRC Standing Committee on Toxicity.

The World Health Organization derived a guideline value of 0.9 mg/L based on a 20% allocation of total daily intake to drinking water. In determining the Australian guideline value, it was felt that sufficient data were available to indicate that 10% was a more realistic figure.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1989). Formaldehyde. Environmental Health Criteria, 89. World Health Organization, IPCS.

Til HP, Woutersen RA, Feron VJ, Hollanders VHM, Falke HE, Clary JJ (1989). Two-year drinking water study of formaldehyde in rats. *Food and Chemical Toxicology*, 27:77–87.

Whittle PJ, Rennie PJ (1988). Determination of formaldehyde in river water by high-performance liquid chromatography. *Analyst*, 113:665–666.

NOTE: Important general information is contained in PART II, Chapter 6

Glyphosate

(endorsed 2011)

GUIDELINE

Based on human health concerns, glyphosate in drinking water should not exceed 1 mg/L.

RELATED CHEMICALS

Glyphosate (CAS 756-09-2) is an aminophosphonic analogue of the natural amino acid glycine. It is available in a variety of salts, but is not chemically related to other pesticides (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population to glyphosate is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, glyphosate would not be a health concern unless the concentration exceeded 1 mg/L. Excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Glyphosate is a non-selective post-emergence herbicide used in the control of weeds in agriculture industry, forestry and public service areas including the aquatic environment.

There are many registered products containing glyphosate salts in Australia. Glyphosate products are intended for professional and home garden use, and are available as concentrated or ready-to-use solutions to be applied using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to glyphosate and its metabolites are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of glyphosate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Glyphosate is generally not reported in analysis of Australian waters. It is strongly sorbed to soils but overseas studies show that there is potential for it to be present in waters as a result of over-spray or run-off from agricultural drainage ditches. For example, Netherlands surface waters were shown to contain up to 0.001 mg/L, and pond water in the USA was shown to contain up to 1.7 mg/L (IPCS 1994), demonstrating the high levels of contamination possible with some applications.

TREATMENT OF DRINKING WATER

Glyphosate has been shown to be completely removed by ozonation (Bozkaya-Schrotter *et al*, 2008). Moderate removal can be achieved using powdered activated carbon adsorption.

MEASUREMENT

Glyphosate in Australian laboratories in commonly analysed by high performance liquid chromatography (HPLC) followed by post-column derivitisation using orthophthaldehyde and subsequent fluorescent detection (Eaglesham, personal communication). Some of the best equipped Australian laboratories are now using HPLC–tandem mass spectrometry for rapid and sensitive analysis without the need for derivitisation. Sensitivities for both procedures allow for low microgram per litre levels to be determined (Eaglesham, personal communication). The National Measurement Institute notes a limit of reporting of 0.01 mg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for glyphosate is 0.3 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 30 mg/kg bw/day from a long-term (3-generation reproduction) study. This NOEL is based on no adverse effects observed at the highest dose in rats. The ADI incorporates a safety factor of 100 and was established in 1985.

The previous *Australian Drinking Water Guidelines* health value was 1.0 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Glyphosate has low absorption from the gastrointestinal tract (30-36%) in rats and rabbits. Dermal absorption is less than 5% in monkeys. It is essentially not metabolised, with 99% excreted in the urine as unchanged glyphosate.

Acute effects: Glyphosate has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Three-month dietary studies in mice and dogs reported decreased bodyweight and food consumption at dose of 7500 mg/kg bw/day (mice) and 500 mg/kg bw/day (dogs).

Long-term effects: Long-term dietary studies in mice reported decreased bodyweight, hepatotoxicity, and changes in the urinary bladder and in the kidney at 814 mg/kg bw/day.

A long-term dietary study in dogs reported no significant toxicological effects up to the dose level of 45 mg/kg bw/day.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for glyphosate.

Genotoxicity: Glyphosate is not considered genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity at the dose levels up to 30 mg/kg bw/day. The ADI was based on the NOEL from this study, which was the highest dose tested (30 mg/kg bw/day).

A single-generation reproduction study in rats at doses up to 10 mg/kg bw/day reported no significant reproductive effects.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Glyphosate is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF HEALTH-BASED GUIDELINE

The health-based guideline of 1 mg/L for glyphosate was determined as follows:

$$1 \text{ mg/L} = \frac{30 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 30 mg/kg bw/day is the NOEL based on a 3-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for glyphosate and it is excluded from the list of agricultural chemicals guideline value derivation because "its health-based value is orders of magnitude higher than concentrations normally found in drinking-water" (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bozkaya-Schrotter B, Daines C, Lescourret A, Bignon A, Breant P, Schrotter J (2008). Treatment of trace organics in membrane concentrates I: pesticide elimination, *Water Science & Technology. Water Supply*, 8(2):223-230.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Eaglesham G (2009). Queensland Health Scientific Services, personal communication.

IPCS (Programme on Chemical Safety) (1994). *Glyphosate.* World Health Organisation Programme on Chemical Safety, Environmental Health Criteria, 159.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Haloacetonitriles
dichloroacetonitrile
trichloroacetonitrile
dibromoacetonitrile
bromochloroacetonitrile

(endorsed 2011)

GUIDELINE

Data are inadequate to set guideline values for haloacetonitriles in drinking water

GENERAL DESCRIPTION

Haloacetonitriles are formed from organic precursors during chlorination or chloramination of drinking water. Concentrations of dihaloacetonitriles reported overseas range up to 0.04 mg/L but are typically less than 0.003 mg/L. Concentrations of trichloroacetonitrile are less than 0.001 mg/L.

Trichloroacetonitrile has been used as an insecticide. No data are available on uses for the other haloacetonitriles.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on concentrations of haloacetonitriles in Australian drinking waters.

LIMITING FORMATION IN DRINKING WATER

The presence of haloacetonitriles in drinking water can be minimised by removing naturally occurring organic matter from the source water, reducing the amount of chlorine added, or using alternative disinfectants

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of haloacetonitriles (USEPA Method 551.1 1995). A salting agent is added to the sample and the haloacetonitriles extracted using methyl tert-butyl ether or pentane. The extracts are then analysed using gas chromatography with an electron capture detector. Limits of determination are less than 0.0001 mg/L.

HEALTH CONSIDERATIONS

Haloacetonitriles are rapidly absorbed from the gastrointestinal tract and metabolised to single carbon compounds, including cyanide. Insufficient data are available to indicate whether haloacetonitriles can accumulate in specific organs.

No data are available on the health effects of haloacetonitriles in humans.

Dichloroacetonitrile and dibromoacetonitrile caused decreased body weights in 90-day feeding studies with rats, but specific target organs were not identified. Dibromoacetonitrile and bromochloroacetonitrile caused an increase in the incidence of squamous cell carcinomas when applied to the skin of mice in the presence of agents that promote tumour growth. No significant increase was observed for dichloroacetonitrile or trichloroacetonitrile.

Dichloroacetonitrile and bromochloroacetonitrile were direct-acting mutagens in tests on bacteria, whereas tests with dibromoacetonitrile and trichloroacetonitrile were negative. All four compounds

induced DNA damage (sister chromatid exchange and DNA strand breaks) in mammalian cells.

Studies with rats indicate that dichloroacetonitrile and trichloroacetonitrile can cause foetal deformities. A short-term reproductive and developmental toxicity study of dibromoacetonitrile in male and female rats found no evidence of reproductive toxicity at dose levels up to 150 mg/L.

A two-year study of dibromoacetonitrile in drinking water conducted recently by the US National Toxicology Program (2008) is reported to have shown clear evidence of carcinogenicity in male rats, and some evidence of carcinogenic activity in female rats, as well as clear evidence of carcinogenic activity in male and female mice, however the final report from this study is not yet available.

The NHMRC Standing Committee on Toxicity reviewed the available data for haloacetonitriles in 1991, and concluded that data were insufficient to set no-effect levels for these compounds. This finding was supported by a review by the NHMRC Water Quality Advisory Committee in 2009, but will be reassessed when the National Toxicology Program report on dibromoacetonitrile becomes available.

DERIVATION OF GUIDELINE

The World Health Organization (WHO 2004) has set a provisional guideline value for dichloroacetonitrile (0.02 mg/L) based on a 90-day study which found increased liver weight in male and female rats. This calculation involved a high uncertainty value. The WHO guideline value for dibromoacetonitrile (0.07 mg/L) is derived from the no-observed-adverse-effects level in a 13-week study of increased body weight in male rats.

These data were not considered to be sufficient to set an Australian guideline value for dichloroacetonitrile. It was also considered not appropriate to set a guideline for dibromoacetonitrile in view of the pending National Toxicology Report report on carcinogenicity for this compound.

REFERENCES

National Toxicology Program (2008) TR-544 Toxicology and carcinogenesis studies of dibromoacetonitrile (CAS No. 3252-43-5) in F344/N Rats and B6C3F1 mice (Drinking water studies) Draft abstract.

USEPA Method 551.1 (1995). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid-liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Haloxyfop

(endorsed 2011)

GUIDELINE

Based on human health concerns, haloxyfop in drinking water should not exceed 0.001 mg/L.

RELATED CHEMICALS

Haloxyfop (CAS 69806-34-4)(Haloxyfop-methyl (CAS 69806-40-2), Haloxyfop-P-methyl (CAS 72619-32-0) belongs to the aryloxyphenoxypropionate class of chemicals. Haloxyfop is a racemic mixture while haloxyfop-R is the resolved R-enantiomer (also known as haloxyfop-P) and has the greater herbicidal activity. It is used as the methyl ester. Other pesticides in this class include diclofop-methyl and diclofop-P-methyl (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, haloxyfop would not be a health concern unless the concentration exceeded 0.001 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Haloxyfop is a post-emergence herbicide for the control of a wide range of annual and perennial grass weeds in a range of agricultural crops, including pasture and fruits as well as in forests.

There are registered products that contain haloxyfop in Australia. The products are intended for professional use and are available as concentrated solutions or wettable granules to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to haloxyfop are residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of haloxyfop may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on the occurrence of haloxyfop in Australian waters were found.

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficiency were found for haloxyfop.

MEASUREMENT

Haloxyfop can be measured by routine liquid chromatography–mass spectrometry analysis, with a limit of reporting of 0.01 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for haloxyfop is 0.0003 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.03 mg/kg bw/day from a long-term (2-year) dietary study in mice. The NOEL is based on the presence of liver tumours at 0.065 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was established in 1987.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Haloxyfop is rapidly absorbed via the gastrointestinal tract. In mice, male rats and dogs, the major excretion route is faeces, while in female rats, monkeys and humans, it is urine. Haloxyfop is not extensively metabolised and its half life varies from 24 hours in monkeys, to 1.8 days in mice, 6.3 days in humans, and 10 days in rats.

Acute effects: Haloxyfop has a low acute oral and dermal toxicity. It is not a skin sensitiser in guinea-pigs.

Short-term effects: Short-term studies have been performed in mice, rats, dogs and monkeys and indicate that the main target organ is the liver.

In a 4-day study in mice, haloxyfop caused liver enlargement from 10 mg/kg bw/day, and in a 14-day study in rats, there were hepatocellular hypertrophy and cytoplasmic changes at 30 mg/kg bw/day. Peroxisome proliferation in the liver was measured in 4-week dietary studies in mice and rats at 0.5 mg/kg bw/day and above. Liver effects were not noted in a 5-week dietary study in dogs, where haematological changes and increase in kidney weight were observed at 45 mg/kg bw/day.

A 13-week study in monkeys resulted in a dose-related increase in liver and kidney weights and decrease in thyroid weight from 10 mg/kg bw/day to 30 mg/kg/day.

Long-term effects: In a 2-year dietary study in mice, liver changes (increased liver weight and cytoplasmic alterations in both sexes, and increased alkaline phosphatase in males) were observed from 0.6 mg/kg bw/day. Liver tumours were noted from 0.065 mg/kg bw/day. The NOEL was 0.03 mg/kg bw/day, and this study is the basis for the ADI. In a 2-year dietary study in rats, changes in the liver (increased liver weight and clinical chemistry) were observed at 0.065 mg/kg bw/day. Kidney discolouration (increased pigment in the proximal convoluted tubules) and a decrease in haematological parameters were observed at 0.1 mg/kg bw/day at 6 months. Haematological parameters and liver weight were normal at the end of the study. A 1-year study in dogs showed decreased haematological values in males from 0.5 mg/kg bw/day.

Carcinogenicity: A 2-year dietary study in mice resulted in an increased incidence of liver adenomas and carcinomas from 0.065 mg/kg bw/day. Tumours were not noted in long-term studies in rats. The carcinogenic risk to humans is considered low, as the mice liver tumours are considered to be species-specific.

Genotoxicity: Only short-term *in vitro* studies are available, and based on these, haloxyfop is not considered to be genotoxic. There are no *in vivo* studies available.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 2- and 3-generation reproduction study and developmental study in rats did not produce any evidence of effects on reproductive parameters or foetal development. In rabbits, there was a marginal increase in abnormalities and delayed development at maternotoxic dose levels that were well in excess of the likely level of human exposure.

Poisons Schedule: Haloxyfop is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.001 mg/L for haloxyfop was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.03 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.03 mg/kg bw/day is the NOEL based on a long-term (2-year) study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Hardness (as calcium carbonate)

(endorsed 1996)

GUIDELINE

To minimise undesirable build-up of scale in hot water systems, total hardness (as calcium carbonate) in drinking water should not exceed 200 mg/L.

GENERAL DESCRIPTION

Hard water requires more soap than soft water to obtain a lather. It can also cause scale to form on hot water pipes and fittings. Hardness is caused primarily by the presence of calcium and magnesium ions, although other cations such as strontium, iron, manganese and barium can also contribute.

Total hardness is the sum of the concentrations of calcium and magnesium ions expressed as a calcium carbonate equivalent. Hardness may also be classified as carbonate (temporary) or noncarbonate (permanent) hardness. Carbonate hardness is the total alkalinity expressed as calcium carbonate, where alkalinity is the sum of the carbonate, bicarbonate and hydroxide content. Noncarbonate hardness is the difference between the total and carbonate hardness.

Degrees of hardness can be described as follows:

<60 mg/L $CaCO_3$	soft but possibly corrosive
60–200 mg/L $CaCO_3$	good quality
200–500 mg/L $CaCO_3$	increasing scaling problems
>500 mg/L $CaCO_3$	severe scaling

Public acceptance of hardness can vary considerably among communities and is generally related to the hardness that the consumer has come to expect, which in turn is due to the source of the water.

Soft water may lead to greater corrosion of pipes, although this will depend on other factors such as pH, alkalinity and dissolved oxygen concentration. Total hardness above 200 mg/L may lead to excessive scaling of pipes and fittings, and cause blockage of safety relief valves in hot water systems.

High total hardness may be a problem for supplies reliant on groundwater. Surface waters can generally be expected to have acceptable values.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Total hardness in major Australian reticulated supplies ranges between about 5 mg/L and about 380 mg/L.

MEASUREMENT

Hardness can be determined by titration of calcium and magnesium with EDTA (APHA Method 2340C 1992).

TREATMENT OF DRINKING WATER

Carbonate (temporary) hardness can be readily reduced by treatment, for example using lime softening; however, this is rarely practised for Australian drinking water. Sodium hexametaphosphate has been used to reduce scale build-up, but does not affect hardness.

HEALTH CONSIDERATIONS

Some epidemiological studies have found that hard water may have a beneficial effect on health, particularly on some types of cardiovascular disease (NAS 1977), but the data are inadequate to conclude that the association is causal.

There is some indication that soft water, with a hardness of less than about 75 mg/L, may adversely affect mineral balance.

DERIVATION OF GUIDELINE

The guideline value is based on two considerations:

- difficulty in obtaining a lather with soap;
- water with a total hardness (as calcium carbonate) above 200 mg/L can cause a rapid build-up of undesirable deposits, or scale, in hot water pipes and fittings. Removal of these deposits can be costly.

GUIDELINES IN OTHER COUNTRIES

The 1984 World Health Organization (WHO) Guideline value for total hardness is 500 mg/L. The 1993 WHO Guidelines do not provide a specific value for hardness.

The Canadian Guidelines rate over 500 mg/L as unacceptable, over 200 mg/L as poor, and 80–100 mg/L as acceptable.

The EEC standards do not include a maximum concentration for hardness, but consider a minimum concentration of at least 60 mg/L to be desirable.

REFERENCES

APHA Method 2340C (1992). Hardness: EDTA titrimetric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NAS (National Academy of Sciences) (1977). Drinking water and health. National Academy of Sciences, Washington DC.

NOTE: Important general information is contained in PART II, Chapter 6

Heptachlor and heptachlor epoxide

(endorsed 1996)

GUIDELINE

Heptachlor should not be detected in drinking water. If present in drinking water, heptachlor would not be a health concern unless the concentration exceeded 0.0003 mg/L.

If it is detected, remedial action should be taken to stop contamination. The limit of determination is 0.00005 mg/L (50 ng/L).

GENERAL DESCRIPTION

Heptachlor is a broad spectrum insecticide used in Australia until September 1994 to protect wooden structures against termites. Its other former uses were withdrawn in the late 1970s and early 1980s. Heptachlor epoxide, an oxidation product of heptachlor, is not commercially available.

Heptachlor is moderately persistent in soil. It is transformed slowly to the epoxide, which is very resistant to further chemical or biological degradation.

Heptachlor has been detected at low nanogram per litre concentrations in water supplies in Europe and the United States. It has been found in a number of foods including human milk. The daily adult intake for heptachlor and the epoxide in the United States has been estimated at about 0.000007 mg/day (7 ng/day) and 0.0002 mg/day respectively. The 1990 Australian Market Basket Survey did not find heptachlor or the epoxide in any of the foods tested (NHMRC and NFA 1991).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Heptachlor has not been detected in major Australian drinking water supplies.

TREATMENT OF DRINKING WATER

No published reports are available on methods for the removal of heptachlor from drinking water supplies. Granular activated carbon would probably be effective.

MEASUREMENT

Heptachlor can be extracted from water using a nonpolar solvent such as pentane, and analysed using gas chromatography with electron capture detection (APHA Method 6630 Part B 1992). The limit of determination is 0.00005 mg/L (50 ng/L).

HEALTH CONSIDERATIONS

Heptachlor is absorbed rapidly from the gastrointestinal tract of rats and distributed throughout the body. It is metabolised to the epoxide and excreted in faeces.

Extensive reviews and summaries of the human and animal toxicology of heptachlor are available (IPCS 1984, JMPR 1991, IARC 1991, NHMRC 1992).

Heptachlor is acutely neurotoxic in animals and humans at high doses, and is hepatotoxic in animals. It caused liver tumours in mice, and in one study, thyroid follicular cell carcinoma in rats. At high exposure levels, heptachlor can affect the viability of the offspring of rodents and dogs.

Heptachlor has not been reported to be genotoxic or teratogenic in animals.

The International Agency for Research on Cancer has concluded that heptachlor is possibly carcinogenic to humans (Group 2B, inadequate evidence in humans, sufficient evidence in experimental animals) (IARC 1991).

DERIVATION OF GUIDELINE

The health-based guideline value of 0.0003 mg/L for heptachlor and heptachlor epoxide was determined as follows:

$$0.0003 \text{ mg/L} = \frac{0.0001 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day}}$$

where:

- 0.0001 mg/kg body weight per day is the maximum acceptable daily intake (ADI) based on a no-effect level of 0.025 mg/kg body weight per day from two studies using dogs (JMPR 1991).
- 70 kg is the average weight of an adult.
- 0.1 gives a guideline value based on 10% of the ADI.
- 2 L/day is the average amount of water consumed by an adult.

The maximum ADI value includes a safety factor of 200 (10 for interspecies variations, 10 for intraspecies variations and 2 for the inadequacy of the data base). No additional safety factors are necessary.

The World Health Organization guideline value of 0.00003 mg/L (30 ng/L) was determined using 1% of the ADI to allow for increased exposure from other sources. Such a low percentage of the ADI was considered inappropriate for Australia.

REFERENCES

APHA Method 6630 Part B (1992). Organochlorine pesticides: Liquid–liquid extraction, gas chromatographic method I. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1991). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Occupational Exposures in Insecticide Application, and Some Pesticides. World Health Organization, IARC, 53.

IPCS (International Programme on Chemical Safety) (1984). Heptachlor. Environmental Health Criteria, 38. World Health Organization, I{CS.

JMPR (1991). Pesticide Residues in Food — 1991. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Expert Group on Pesticide Residues. Food and Agricultural Organization of the United Nations, FAO Plant Production and Protection Paper III, Rome.

NHMRC (National Health and Medical Research Council) (1992). Cyclodiene Insecticide Use in Australia. NHMRC, AGPS, Canberra.

NHMRC and NFA (National Health and Medical Research Council and National Food Authority) (1991). The 1990 Australian Market Basket Survey. NHMRC and NFA, AGPS, Canberra.

NOTE: Important general information is contained in PART II, Chapter 6

Hexachlorobutadiene

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of hexachlorobutadiene in drinking water should not exceed 0.0007 mg/L.

GENERAL DESCRIPTION

Hexachlorobutadiene has occasionally been detected in drinking water supplies in the United States and some European countries at concentrations less than 0.005 mg/L.

Hexachlorobutadiene is used as a solvent in chlorine gas production, an intermediate in the manufacture of rubber compounds, a lubricant, a pesticide and a fumigant.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Hexachlorobutadiene has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Granular activated carbon has proved effective in trials for the removal of hexachlorobutadiene from drinking water.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and hexachlorobutadiene trapped on an adsorbent. The adsorbent is then heated and hexachlorobutadiene analysed using gas chromatography with electron capture detection. The limit of determination is approximately 0.0004 mg/L.

HEALTH CONSIDERATIONS

Experiments in laboratory animals have revealed that approximately 95% of the ingested dose of hexachlorobutadiene is absorbed. It has been found in the blood, liver, brain, spleen, kidney and mesentery. Hexachlorobutadiene is metabolised in the gastrointestinal tract and kidney to a number of water soluble metabolites, and excreted in the urine.

Long-term intermittent human exposure has been reported to cause higher incidences of hypotension, myocardial dystrophy, nervous system and liver disorders, and respiratory tract lesions.

In studies using rats, hexachlorobutadiene caused multiple toxicologic effects, with the kidney being the organ most affected. Kidney tumours have been induced at doses of 20 mg/kg body weight per day.

Tests for mutagenicity with different strains of bacteria have reported both positive and negative results. Some metabolites have given positive results.

The International Agency for Research on Cancer has concluded that hexachlorobutadiene is not classifiable as to its carcinogenicity to humans (Group 3, no adequate evidence in humans and limited evidence in animals) (IARC 1987).

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF GUIDELINE

The assessment of the toxicological data for hexachlorobutadiene by the World Health Organization (WHO) has been used without review. The guideline value of 0.0007 mg/L was determined as follows:

$$0.0007 \text{ mg/L} = \frac{0.2 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 0.2 mg/kg body weight per day is the no-effect level based on a 2-year feeding study using rats (Kociba 1977).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for possible carcinogenic effects and genotoxicity of some metabolites).

The WHO guideline value of 0.0006 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

Kociba RJ, Keyes DG, Jersey GC, Ballard JJ, Dittenber DA, Quast JF, Wade CE, Humiston CG, Schwetz BA (1977). Results of a two-year toxicity study with hexachlorobutadiene in rats. *American Industrial Hygiene Association Journal*, 38:589–602.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Hexazinone

(endorsed 2011)

GUIDELINE

Based on human health concerns, hexazinone in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Hexazinone (CAS 51235-04-2) belongs to the triazinone class of chemicals. Another pesticide in this class is metribuzin. This class of chemicals is structurally similar to the triazine class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, hexazinone would not be a health concern unless the concentration exceeded 0.4 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Hexazinone is a herbicide used for the control of annual and perennial weeds in pine forest plantations, and annual and perennial grasses, broad-leaf weeds, and vines in industrial areas and sugar cane crops.

There are registered products that contain hexazinone in Australia. The products are intended for professional use and all are available as soluble concentrates intended to be diluted and applied by handspray when used in public areas, and by groundspray and aerial spray when used in other areas. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to hexazinone is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of hexazinone may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on the occurrence of hexazinone in Australian waters could be found. Groundwater contamination has been reported in the USA up to 0.034 mg/L, and the concentration of hexazinone in surface waters can be as high as 0.14 mg/L up to 6 months after application (USEPA 1994).

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficiency could be found for hexazinone.

MEASUREMENT

Hexazinone can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.01 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for hexazinone is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 10 mg/kg bw/day from a long-term (2-year) dietary rat study. The NOEL is based on decreased bodyweight gain at 125 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was first established in 1978.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Hexazinone is readily absorbed from the gastrointestinal tract. It is extensively metabolised by hydroxylation of the cyclohexyl ring, and monodemethylation of the diethylamino group. Excretion is almost complete by 24 hours, mostly as metabolites in urine and to a lesser extent in faeces.

Acute effects: Hexazinone has low acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 90-day oral studies in rats and dogs, there was decreased bodyweight gain at doses of 100 mg/kg bw/day in rats and at 300 mg/kg bw/day in dogs.

Long-term effects: In long-term (2-year) dietary studies in mice and rats, effects were confined to decreased bodyweight gain in rats at doses of 30 mg/kg bw/day, and hepatocellular hypertrophy and increased relative liver weight at doses of 125 mg/kg bw/day in mice. The lowest overall NOEL was 10 mg/kg bw/day in rats. This NOEL is the basis for the current ADI.

Carcinogenicity: Based on a 2-year studies in mice and rats, there is no evidence of carcinogenicity for hexazinone.

Genotoxicity: Hexazinone is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: No reproduction studies have been conducted. Developmental toxicity studies in rats and rabbits did not produce any evidence of effects on foetal development.

Poisons Schedule: Hexazinone is in Schedules 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for hexazinone was determined as follows:

$$0.4 \text{ mg/L} = \frac{10 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 10 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1994). Reregistration eligibility decision (RED) for hexazinone. USEPA.

Hydrogen sulfide

Sulfide

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations, the concentration of hydrogen sulfide in drinking water should not exceed 0.05 mg/L.

No health-based guideline value has been set for hydrogen sulfide, or sulfide, as the aesthetic guideline is considerably below the concentration that would cause health problems.

GENERAL DESCRIPTION

Hydrogen sulfide is formed in drinking water by the hydrolysis of soluble sulfides, or through the reduction of sulfate by the action of microorganisms. Both processes require anoxic conditions. In well-oxygenated water, sulfide will be chemically or biologically oxidised to sulfate or elemental sulfur, and concentrations are extremely low. Higher concentrations can occur in anoxic water drawn from deep storages.

In water, hydrogen sulfide will be in equilibrium with the sulfide and hydrosulfide ions. The ratio will depend on pH, temperature and salinity. At pH 7.4, about a third will be present in undissociated form, with the remainder present as hydrosulfide. Above pH 10, the sulfide ion will be the dominant form; below pH 5, undissociated hydrogen sulfide will predominate.

Hydrogen sulfide has an obnoxious 'rotten egg' gas odour, with a taste and odour threshold of 0.05 mg/L. High concentrations in air can have a deceptively sweet smell and cause 'olfactory fatigue' (a deadening of the sense of smell).

Hydrogen sulfide is used industrially in the production of sulfur, sulfuric acid, inorganic sulfides, thiophenes and other organic compounds. It occurs as a by-product in a number of processes including petrol refining, coke ovens, paper mills, iron smelters, food processing and tanneries. It is present in sewers and is a major component of sewage odour.

Data on the concentration of hydrogen sulfide in food are scarce, although a number of foods and drinks are known to contain sulfides.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for hydrogen sulfide, or sulfide.

TREATMENT OF DRINKING WATER

Hydrogen sulfide can be removed from drinking water by keeping the water well oxygenated.

MEASUREMENT

The sulfide concentration of drinking water can be determined using the methylene blue colorimetric method (APHA Method 4500-S^{2-} Part D 1992). The limit of determination is 0.02 mg/L.

HEALTH CONSIDERATIONS

Soluble sulfides are absorbed rapidly from the gastrointestinal tract, although hydrogen sulfide is absorbed principally by the lung. Animal studies have indicated that after absorption, hydrogen sulfide is distributed to the brain, liver, kidneys, pancreas and small intestine.

An extensive review and summary of the human and animal toxicity data for hydrogen sulfide is available (IPCS 1981).

There are no data on the human health effects of ingesting water that contains hydrogen sulfide. Ingestion of sulfides has been known to cause nausea, vomiting and irritation of the mucous membranes. Inhalation of hydrogen sulfide is known to be extremely toxic to humans, with exposure to amounts as low as 5 ppm for 30 minutes or more producing headaches, dizziness, nausea, gastrointestinal disorders and breathing problems. Inhalation of concentrations above 500 ppm can cause cardiac failure and death.

Animal data are mainly from short-term inhalation studies. Effects include neurotoxic activity and distortions in cardiac rhythm.

No long-term carcinogenicity bioassays have been undertaken on hydrogen sulfide. Sodium sulfide did not induce cancers in experimental animals. Hydrogen sulfide was not found to be mutagenic in tests with different strains of bacteria.

DERIVATION OF GUIDELINE

The guideline value of 0.05 mg/L is based on the aesthetic considerations of taste and odour. Insufficient data are available to determine a guideline value based on health considerations. The guideline value is, however, considerably lower than the concentration likely to have a harmful effect and it is therefore unlikely that a person would consume a harmful dose.

REFERENCES

APHA Method 4500-S^{2-} Part D (1992). Sulfide: Methylene blue method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IPCS (International Programme on Chemical Safety) (1981). Hydrogen Sulfide. Environmental Health Criteria, 19. World Health Organization, IPCS.

NOTE: Important general information is contained in PART II, Chapter 6

Imazapyr

(endorsed 2011)

GUIDELINE

Based on human health concerns, imazapyr in drinking water should not exceed 9 mg/L.

RELATED CHEMICALS

Imazapyr (CAS 81334-34-1) belongs to the imidazolinone class of chemicals. Other pesticides in this class include imazamox and imazethapyr (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, imazapyr would not be a health concern unless the concentration exceeded 9 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Imazapyr is a broad spectrum herbicide for the pre- and post-emergence control of certain annual grass and broad-leaf weeds in agricultural crops

There are registered products that contain imazapyr, or its isopropylamine or ammonium salt, in Australia. The products are intended for professional use and are available as soluble concentrates or water-dispersible granule formulations which are diluted and applied using ground boom sprayers. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to imazapyr is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of imazapyr may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for imazapyr in Australian waters were found. In the USA, the modelled estimated surface and groundwater concentration were 0.34–79 µg/L and 39 µg/L, respectively (USEPA 2006).

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficiency were found for imazapyr.

MEASUREMENT

Imazapyr can be measured in water by liquid chromatography–mass spectrometry with an electrospray interface, with a limit of detection of 0.004 µg/L (D'Ascenzo *et al.* 1998).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for imazapyr is 2.5 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 250 mg/kg bw/day from a 1-year dietary study in dogs. The NOEL is based on the absence of signs of toxicity at the highest dose tested (250 mg/kg bw/day). The ADI incorporates a safety factor of 100 and was established in 1998.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Imazapyr is readily and extensively absorbed via the gastrointestinal tract in rats. It was rapidly excreted in the urine and faeces (within 2 days).

Acute effects: Imazapyr and its isopropylamine salt have low acute oral toxicity and low dermal toxicity. Neither imazapyr nor its isopropylamine salt is a skin sensitiser.

Short-term effects: In a 13-week dietary study in rats, no treatment-related effects were observed at the highest dose tested (879 mg/kg bw/day).

Long-term effects: In an 18-month dietary study in mice, there was no evidence of treatment-related toxicity at the highest dose tested (1639 mg/kg bw/day). In 1-year dietary studies in rats and dogs, there was no evidence of treatment-related toxicity at the highest dose tested (639 and 250 mg/kg bw/day in rats and dogs respectively). The NOEL of 250 mg/kg bw/day in dogs is the basis of the current ADI.

Carcinogenicity: There was no evidence of carcinogenicity, based on an 18-month dietary study in mice and a 2-year dietary study in rats.

Genotoxicity*:* Imazapyr is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive or developmental effects.

Poisons Schedule: Imazapyr is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 9 mg/L for imazapyr was determined as follows:

$$9 \text{ mg/L} = \frac{250 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 250 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.

NOTE: Important general information is contained in PART II, Chapter 6

- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

D'Ascenzo G, Gentili A, Marchese S, Perret D (1998). Development of a method based on liquid chromatography-electrospray mass spectrometry for analyzing imidazolinone herbicides in environmental water at part-per-trillion levels. *Journal of Chromatrography A*, 800(1):109-119.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2006). Reregistration Eligibility Decision for Imazapyr. EPA 738-R-06-007, OPP-2005-0495.

NOTE: Important general information is contained in PART II, Chapter 6

Iodine, Iodide

(endorsed 2011)

GUIDELINE

Iodide: Based on health considerations, the concentration of iodide in drinking water should not exceed 0.5 mg/L.

Iodine: No guideline value has been set for molecular iodine.

GENERAL DESCRIPTION

The element iodine is present naturally in seawater, nitrate minerals and seaweed, mostly in the form of iodide salts. It may be present in water due to leaching from salt and mineral deposits. Iodide can be oxidised to molecular iodine with strong disinfectants such as chlorine.

Molecular iodine solutions are used as antiseptics and as sanitising agents in hospitals and laboratories. Iodine is occasionally used for the emergency disinfection of water for field use but is not used for disinfecting larger drinking water supplies. Iodide is used in pharmaceutical and photographic materials.

Iodine has a taste threshold in water of about 0.15 mg/L.

Iodide occurs in cows' milk and seafood. Some countries add iodide to table salt to compensate for iodide-deficient diets.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Concentrations of iodide in Australian source or treated water ranges from 0.005 to 2.9 mg/L (median 0.03 mg/L, mean 0.1 mg/L).

TREATMENT OF DRINKING WATER

It is unlikely that the concentration of iodine or iodide in drinking water would ever be high enough to justify water treatment.

MEASUREMENT

The iodine or iodide concentration in drinking water can be determined using the Leuco crystal violet method (APHA 4500-I-, Part B 1992). The limit of determination is approximately 0.01 mg/L.

A test for iodide alone may not determine all the iodide present at the time of sampling (given the equilibrium between iodine species in water – molecular iodine, iodide, iodate, and hypoiodite). A screening test for total iodine may be carried out by inductively coupled plasma mass spectrometry. If total iodine is found to be elevated, then analysis for iodide may be carried out by ion selective electrode, with further investigation and sampling if necessary.

HEALTH CONSIDERATIONS

Iodine is an essential trace element for humans and is used in the synthesis of thyroid hormones. Tolerable upper intake levels for iodine in children and adults recommended by Food Standards Australia and New Zealand (FSANZ 2008) are 0.2 and 1.1 mg/day respectively.

Iodine is efficiently absorbed by the gastrointestinal tract and deposited in the thyroid gland, the eye, and muscle tissue. More than 70% is found in the thyroid gland.

High oral doses (more than 30 mg/kg bodyweight) of iodine can be lethal. Lower doses (3.3 mg/kg bodyweight) have been used to treat asthmatic patients without adverse effects.

Chronic exposure to high amounts of iodide in the diet (over 2 mg/day) can result in a condition known as iodism. Symptoms resemble those of a sinus cold. Long-term consumption of iodinated drinking water has not been associated with adverse health effects in humans. Prisoners drinking water containing up to 1 mg/L iodine for five years showed no signs of iodism or hypothyroidism, but some changes in uptake of iodine by the thyroid gland were observed.

Animal studies using chickens susceptible to autoimmune thyroiditis reported an increase in the incidence of the disease when they were given high doses of iodide in their drinking water (200 mg/L). Excessive iodide consumption may increase the incidence of this disease in humans.

Iodide has not been shown to increase the incidence of cancer of the thyroid in laboratory animals. No data are available on the mutagenic activity of iodine.

DERIVATION OF GUIDELINE

In healthy adults, sub-clinical hypothyroidism is associated with intakes of 1.7 to 1.8 mg/day, and for children with intakes of 1.15 mg/day (EFSA 2006, FSANZ 2008). Chronic iodine intakes of approximately 1 mg/day, however, appear to be well tolerated by healthy adults. This is consistent with the provisional maxium tolerated daily intake of 1 mg/day established by the Joint FAO/WHO Expert Committee on Food Additives (JECFA 1989), and the nutrient reference value and tolerable upper intake level of 1.1 mg/day respectively recommended by the NHMRC (2006) and Food Standards Australia New Zealand (FSANZ 2008) for iodine intake by adults in Australia and New Zealand.

The majority of iodine is taken in via the diet. Food Standards Australia New Zealand noted that Australian and New Zealand dietary intakes of iodine were the same (FSANZ 2008). The daily dietary intake of New Zealanders 5 years old and over is 0.1 mg/person (FSANZ 2008). It has been proposed that table salt and salt used in bread be mandatorily fortified with iodine; if this occurs, intake would rise to 0.2 mg/person.

Applying the method of International Program on Chemical Safety (IPCS 2002) for essential trace elements, a drinking water guideline of 0.5 mg/L (rounded up) can be calculated:

$$0.45 \text{ mg/L} = \frac{1.1 \text{ mg/person} - 0.2 \text{ mg/person}}{2 \text{ L/day}}$$

Where:

- 1.1 mg/person is the tolerable daily intake for iodine.
- 0.2 mg/person is the background dietary intake assuming mandatory salt fortification.
- 2 L/day is the estimated maximum amount of water consumed by an adult.

Recent data from studies on rats indicate that the effects of molecular iodine in drinking water on thyroid hormone concentrations in the blood differ from those of iodide. The guideline value therefore applies only to iodide. No guideline value can be established for molecular iodine.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 4500-I-Part B (1992). Iodide: Leuco crystal violet method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

EFSA (European Food Safety Authority (2006). Tolerable upper intake levels for vitamins and minerals. EFSA.

FSANZ (Food Standards Australia New Zealand) (2008). Final Assessment Report: Proposal P230. Consideration of mandatory fortification with iodine for New Zealand. FSANZ.

IPCS (International Programme on Chemical Safety) (2002). Principles and methods for the assessment of risk from essential trace elements. Environmental Health Criteria 228. World Health Organization, IPCS, Geneva, Switzerland.

JECFA (Joint FAO/WHO Expert Committee on Food Additives) (1989). Toxicological evaluation of certain food additives and contaminants: Iodine. World Health Organization Food Additive Series, 24, 267-294. The 33rd meeting of the Joint FAO/WHO Expert Committee on Food Additives, Geneva, Switzerland.

NHMRC (National Health and Medical Research Council) (2006). *Nutrient Reference Values for Australia and New Zealand including Recommended Dietary Intakes*. NHMRC, Commonwealth of Australia, Canberra.. http://www.nhmrc.gov.au/publications/synopses/_files/n35.pdf

NOTE: Important general information is contained in PART II, Chapter 6

Iprodione

(endorsed 2011)

GUIDELINE

Based on human health concerns, iprodione in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Iprodione (CAS 36734-19-7) belongs to the dicarboximide class of chemicals. Another pesticide in this class is procymidone (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, iprodione would not be a health concern unless the concentration exceeded 0.1 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Iprodione is a fungicide for the control of disease in a wide range of agricultural crops.

There are registered products that contain iprodione in Australia. The products are intended for professional use and are available as a suspension concentrate, suspoemulsion or wettable granule formulation. They are applied as either a dilute or concentrated spray to crops and turf, or as a liquid seed dressing. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to iprodione is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of iprodione may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of iprodione in Australian drinking waters were found.

TREATMENT OF DRINKING WATER

No reports of the treatment of iprodione in drinking water were found. However, research indicates that ultraviolet photolysis and advanced oxidation are likely to be effective treatment processes (Garbin *et al.* 2007).

MEASUREMENT

Iprodione can be measured in water by solid phase extraction followed by high performance liquid chromatography with diode array detection (D'Archivio *et al*. 2007). The reported limit of quantitation for the technique in groundwater is 0.05 µg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for iprodione is 0.04 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 4 mg/kg bw/day from a 1-year dietary study in dogs. The NOEL is based on changes in organ weights and haematological parameters at 25 mg/kg bw/day. The ADI incorporates a safety factor of 100 and it was established in 1986.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Iprodione is extensively metabolised and excreted in the urine and faeces, almost completely within 96 hours.

Acute effects: Iprodione has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Short-term studies in mice and dogs identified the liver as the major target organ. In short-term dietary studies in mice, reduced bodyweight gain and histopathological changes in the liver, testes, kidney and urinary bladder were seen at 900 mg/kg bw/day. In a 5-month dietary study in rats, no treatment-related adverse effects were reported at the highest dose tested of 50 mg/kg bw/day. In a 3-month dietary study in dogs, there was slight liver hypertrophy and changes in clinical chemistry at 180 mg/kg bw/day.

Long-term effects: In 1- and 2-year dietary studies in mice and rats, there were changes in spleen and kidney histopathology in rats at 12 mg/kg bw/day, testicular interstitial cell hyperplasia in both mice and rats at 115 mg/kg bw/day, changes in liver histopathology in mice at 115 mg/kg bw/day, and increased organ weights in mice at 575 mg/kg bw/day.

In a 1-year dietary study in dogs, changes in liver and prostate weights and histological changes within the adrenal glands, kidneys, liver and urinary bladder were observed at 25 mg/kg/bw/day. At 151 mg/kg bw/day, there were also changes in haematological and clinical chemistry parameters. The NOEL was 4 mg/kg bw/day and this is the basis for the current ADI.

Carcinogenicity: In mice, there was an increased incidence of benign and malignant liver cell tumours and ovarian luteomas at 575 mg/kg bw/day. In rats, there was an increased incidence of benign testicular tumours (interstitial cell tumours and bilateral interstitial cell tumours) at 65 mg/kg bw/day. These effects were noted at dose levels well in excess of the likely level of human exposure in drinking water.

Genotoxicity: Iprodione is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a 3-generation reproduction study in rats, there was no effect on reproductive parameters at the highest dose tested (119 mg/kg bw/day), however, the litter size and pup bodyweights were reduced in the third generation at the high dose in the absence of maternal toxicity. In a separate 2-generation study in rats, there were no effects on reproductive parameters or on pups. In developmental studies in rats and rabbits, there was no evidence of effects on foetal development at doses that did not result in maternotoxicity.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Iprodione is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for iprodione was determined as follows:

$$0.1 \text{ mg/L} = \frac{4 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 4 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

D'Archivio AA, Fanelli M, Mazzeo P, Ruggieri F (2007) Comparison of different sorbents for multiresidue solid-phase extraction of 16 pesticides from groundwater coupled with high-performance liquid chromatography. *Talanta,* 71(1):25-30.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Garbin JR, Milori D, Simoes ML, da Silva WTL, Neto LM (2007) Influence of humic substances on the photolysis of aqueous pesticide residues. *Chemosphere,* 66(9):1692-1698.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Iron

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (precipitation of iron from solution and taste), the concentration of iron in drinking water should not exceed 0.3 mg/L.

No health-based guideline value has been set for iron.

GENERAL DESCRIPTION

Iron occurs commonly in soil and rocks as the oxide, sulfide and carbonate minerals. In water, it is present in oxidised forms as ferric (Fe(III)) or ferrous (Fe(II)) compounds.

Iron has many domestic and industrial applications, ranging from iron and steel products and pigments in paints to food colours and preparations for preventing iron deficiency in humans. Iron sulfate (hydroxylated ferrous sulfate) is used as a flocculant in water treatment.

In aerated surface waters, iron is often complexed with organic matter such as humic material, or adsorbed onto suspended matter. Iron concentrations in uncontaminated surface waters are usually less than 1 mg/L; however, water supplied through rusting iron pipes can have concentrations of 5 mg/L or higher.

In oxygen-depleted ground water, iron concentrations of up to 100 mg/L have been recorded.

Iron has a taste threshold of about 0.3 mg/L in water, and becomes objectionable above 3 mg/L. High iron concentrations give water an undesirable rust-brown appearance and can cause staining of laundry and plumbing fittings, fouling of ion-exchange softeners, and blockages in irrigation systems. Growths of iron bacteria, which concentrate iron, may cause taste and odour problems and lead to pipe restrictions, blockages and corrosion.

Food is the major source of iron intake, and iron is a natural constituent in plants and animals. Fish, green vegetables and tomatoes have high iron content.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, total iron concentrations range up to 4 mg/L, with typical concentrations around 0.1 mg/L.

TREATMENT OF DRINKING WATER

Iron salts can be effectively removed by the standard water treatment processes of coagulation followed by filtration. Groundwater supplies with a high iron content can be treated to form iron precipitates using aeration, oxidation with chlorine, pH adjustment or lime softening.

MEASUREMENT

The iron concentration in drinking water can be determined using inductively coupled plasma emission spectroscopy or graphite furnace atomic absorption spectroscopy (APHA Method 3500-Fe Parts B or C 1992). The limits of determination are 0.01 mg/L and 0.005 mg/L respectively. Alternatively the phenanthroline colorimetric method (APHA 3500-Fe Part D 1992), which has a limit of determination of 0.01 mg/L, can be used. Flame atomic absorption spectroscopy is not sufficiently sensitive.

HEALTH CONSIDERATIONS

Iron is an essential trace element for humans. Minimum daily requirement varies with age and sex. For example, women aged 11–50 years need about 14 mg per day but this requirement doubles for pregnant women, while men require about 7 mg per day. Iron deficiency is common and affects people throughout the world.

The amount of iron absorbed from food by the gastrointestinal tract varies from 1% to 20%, according to individual requirements and the source of iron. It is used in the production of haemoglobin, myoglobin and a number of enzymes, and is stored in the spleen, liver, bone marrow and muscle.

Numerous cases of iron poisoning have been reported, mainly among young children who ingest medicinal iron supplements formulated for adults. Physiological regulation of iron absorption confers a high degree of protection against iron toxicity and there are a number of reports of people, particularly adults, taking high doses of iron with no adverse effects.

Studies with animals over long periods have reported only very mild adverse effects associated with a high iron intake.

There is no evidence that iron induces cancer in laboratory animals. Most iron salts have been inactive in tests for mutagenicity and do not induce chromosome aberrations in human cells.

DERIVATION OF GUIDELINE

Insufficient data are available to determine a health-based guideline value for iron in drinking water. The guideline value is based on the taste threshold of 0.3 mg/L, which is similar to the concentration that would result in iron precipitating out of solution. Sufficient human data exist to indicate that iron in drinking water would not become a health concern unless the concentration was above 3 mg/L, well in excess of the concentration that would cause water to taste objectionable, and it is unlikely that such water would be consumed.

REFERENCES

APHA Method 34500-Fe Part B (1992). Iron: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Fe Part C (1992). Iron: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Fe Part D (1992). Iron: Phenanthroline method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

Lanthanum

(endorsed 2017)

GUIDELINE

Based on human health considerations, the concentration of lanthanum in drinking water should not exceed 0.002 mg/L.

GENERAL DESCRIPTION

Lanthanum is an element in the rare earth group (also known as lanthanides group) that can enter water via run-off from agricultural soil where it has been used as fertiliser, from the weathering of rock, from specific discharges or use as a phosphate binder, and from leaching from the tailings of rare earth mining.

In water, lanthanum's oxidation state is primarily trivalent and it may be present in varying amounts as dissolved lanthanum or as insoluble forms associated with particulates. The concentration of total lanthanum in raw drinking water sources in the Netherlands was reported to range between 0.0005 to 0.013 mg/L, although concentrations in surface waters within rare earth mining areas or downstream of some industrial activities may be much higher (de Boer *et al* 1996, Protano and Riccobono 2002, Kulaksiz and Bau 2011).

An assessment estimating the total daily intake of lanthanum in humans was not available.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for lanthanum. Limited analytical results from a small number of water sources in Australia indicate levels orders of magnitude lower than when lanthanum is applied for phosphate control.

The National Industrial Chemical Notification and Assessment Scheme (NICNAS) recommends regular monitoring of Australian drinking water reservoirs if they have been subject to the addition of a lanthanum-based water treatment product (NICNAS 2014). In this circumstance, as part of a drinking water supply system assessment, consideration should be given to the possibility of accumulation of lanthanum in the water or sediment following multiple applications of a lanthanum-based product.

TREATMENT OF DRINKING WATER

It is expected that lanthanum levels in water will be reduced by the processes used to prepare water for drinking (e.g. coagulation, flocculation, sedimentation, filtration, pH correction, anti-scaling, or a combination of these) (NICNAS 2014).

MEASUREMENT

The concentration of lanthanum in water samples can be determined by inductively coupled plasma mass spectroscopy (ICP-MS) with a limit of reporting of less than 0.001 mg/L. The guideline value is for total lanthanum, so an analytical method should be used which measures both soluble and insoluble lanthanum.

HEALTH CONSIDERATIONS

In Australia, NICNAS reviewed the literature on lanthanum in its Secondary Notification Assessment for Phoslock™ (NICNAS 2014).

The health information for lanthanum is based on the data for soluble and insoluble lanthanum salts.

All lanthanum salts have very low oral bioavailability. The absorption and kinetics of lanthanum from lanthanum carbonate (a relatively insoluble salt) have been reasonably well studied in humans; it has an oral bioavailability of 0.00015–0.02%, but with a terminal half-life of 15–37 hours and only 1.7% of the absorbed dose excreted in urine (NICNAS 2014). The oral bioavailability of soluble forms of lanthanum may be one or two orders of magnitude higher than that of lanthanum carbonate (Pennick *et al* 2006, He *et al* 2007).

Several studies on the effects of human exposure to lanthanum carbonate, approved for medical use in non-pregnant adults with end-stage renal failure to prevent absorption of dietary phosphate, indicate that no adverse systemic effects were seen and the most frequently reported local effect following ingestion of the chemical is gastrointestinal in nature (Health Canada 2007, US FDA 2008, Swedish MPA 2006).

There is very little epidemiological data on lanthanum. The available published studies are poorly documented and inconclusive for determination of effects of lanthanum exposure due to the absence of direct exposure measurements and potential confounding factors, for example co-exposure to other chemicals in the environment (NICNAS 2014).

Lanthanum toxicity is caused by the free cation, with adverse systemic effects being observed in experimental animals from exposure to soluble lanthanum compounds. A number of oral repeat dose studies with lanthanum carbonate in a variety of animal species show no systemic toxicity relevant to humans; the observed local effect is gastric irritation due to high doses precipitating in the rodent stomach (NICNAS 2014). Repeated oral exposures of rodents to lanthanum chloride caused adverse systemic effects in the liver, and local irritation effects in the stomach (Cheng *et al* 2012, Cheng *et al* 2014, NICNAS 2014).

Studies in rodents of up to six months' exposure to lanthanum chloride have reported that it can cause histopathological neurotoxicity, learning deficiency, small but measurable increases of lanthanum in the brain after high doses, and various changes in brain biochemistry (Briner *et al* 2000, Feng *et al* 2006a, Feng *et al* 2006b, He *et al* 2008, NICNAS 2014). The no-observed-adverse-effect-level (NOAEL) for lanthanum chloride established from the critical studies is 0.1 mg/kg bw/day, based on neurotoxicity (decreased numbers of brain cells) and learning decrements (NICNAS 2014). The equivalent amount of lanthanum ion is 0.06 mg La^{3+}/kg bw/day.

There is no firm evidence that lanthanum is carcinogenic. The weight of evidence indicates that lanthanum is not mutagenic in tests with bacteria and that it does not damage DNA (NICNAS 2014).

DERIVATION OF GUIDELINE

The guideline value for lanthanum in drinking water was derived as follows:

$$0.002 \text{ mg/L} = \frac{0.06 \text{ mg/kg bw/day} \times 70 \text{ kg} \times 0.1}{100 \times 2 \text{ L/day}}$$

where:

- 0.06 mg/kg bw/day is the La^{3+} NOAEL for neurotoxic and neurobehavioural effects in rats.
- 70 kg is the average weight of an adult.

NOTE: Important general information is contained in PART II, Chapter 6

- 0.1 is a proportionality factor based on the assumption that 10% of daily intake is attributable to drinking water.
- 100 is the uncertainty factor to account for intra- and inter-species variations.
- 2 L/day is the estimated maximum amount of water consumed by an adult.

Factors for 'less than lifetime' exposure and/or uncertainty in the 'toxicological database' are not recommended because a comprehensive database for lanthanum carbonate, consisting of many repeat oral dose investigations in different species, including lifetime carcinogenicity studies, indicates only local effects at the site of application (stomach) and a NOAEL of 100 mg/kg bw/day. These toxicological studies have been performed using an insoluble lanthanum salt and such insoluble forms may be in drinking water sources to variable extents, and included in the total lanthanum analytical measurement. That is, some of the measured lanthanum may be in a form that has much less toxicity than the soluble lanthanum chloride upon which the drinking water guideline is based.

This guideline value is based on the effects of lanthanum from chronic exposure. As such, occasional detections of lanthanum above the guideline value would not normally be a human health concern.

REFERENCES

Briner W, Rycek RF, Moellenberndt A, Dannull K (2000). Neurodevelopmental effects of lanthanum in mice. *Neurotoxicology and Teratology,* 22(4):573-581.

Cheng J, Cheng Z, Hu R, Cui Y, Cai J, Li N, Gui S, Sang X, Sun Q, Wang L, Hong F (2014). Immune dysfunction and liver damage of mice following exposure to lanthanoids. *Environmental Toxicology*, 29(1):64-73.

Cheng J, Li N, Cai J, Cheng Z, Hu R, Zhang Q, Wan F, Sun Q, Gui S, Sang X, Wang L, Hong F (2012). Organ histopathological changes and its function damage in mice following long-term exposure to lanthanides chloride. *Biological Trace Element Research*, 145(3):361–368.

de Boer JL, Verweij W, van der Velde-Koerts T, Mennes W (1996). Levels of rare earth elements in Dutch drinking water and its sources. Determination by inductively coupled plasma mass spectrometry and toxicological implications. A pilot study. *Water Research*, 30(1):190-198.

Feng LX, Xiao HQ, He X, Li ZJ, Li FL, Liu NQ, Zhao YL, Juang YY, Zhang ZY, Chai ZF (2006a). Long-term effects of lanthanum intake on the neurobehavioral development of the rat. *Neurotoxicology and Teratology*, 28:119-124.

Feng LX, Xiao HQ, He X, Li ZJ, Li FL, Liu NQ, Zhao YL, Huang YY, Zhang ZY, Chai ZF (2006b). Neurotoxicological consequence of long-term exposure to lanthanum. *Toxicology Letters,* 165(2):112–120.

He X, Zhang ZY, Feng LX, Li ZJ, Yang JH, Zhao YL, Chai ZF (2007). Effects of acute lanthanum exposure on calcium absorption in rats. *Journal of Radioanalytical and Nuclear Chemistry,* 272(3):557-559.

He X, Zhang ZY, Zhang H, Zhao YL, Chai ZF (2008). Neurotoxicological evaluation of long-term lanthanum chloride exposure in rats. *Toxicological Sciences,* 103(2):354–361.

Health Canada (2007). Summary basis of decision. Fosrenol Lanthanaum carbonate hydrate Submission Control No 102240.

Kulaksiz S and Bau M (2011). Rare earth elements in the Rhine River, Germany: first case of anthropogenic lanthanum as a dissolved microcontaminant in the hydrosphere. *Environment International* 37(5):973-979.

NOTE: Important general information is contained in PART II, Chapter 6

NICNAS (2014). Phoslock, Existing Chemical Secondary Notification Assessment Report NA/899S. NICNAS, Australian Government Department of Health. Available from https://www.nicnas.gov.au/chemical-information/other-assessment-reports.

Pennick M, Dennis K, Damment SJP (2006). Absolute bioavailability and disposition of lanthanum in healthy human subjects administered lanthanum carbonate. *Journal of Clinical Pharmacology*, 46(7):738-746.

Protano G and Riccobono F (2002). High contents of rare earth elements (RREs) in stream waters of a Cu-Pb-Zn mining area. *Environmental Pollution*, 117(3):499-514.

Swedish MPA (2006). Public Assessment Report, Scientific discussion, Fosrenol (lanthanum) SE/H/481/01-04/E01. Lakemedelsverket – Sweden's Medical Product Agency. http://www.lakemedelsverket.se/SPC_PIL/Pdf/par/Fosrenol%20250%20mg,%20500%20mg,%20750%20mg,%201000%20mg%20chewable%20tablet.pdf .

USFDA (2008). Fosrenol product information sheet. Revision date April 2008. http://www.fda.gov/medwatch/safety/2008/Oct_PI/Fosrenol_PI.pdf.

Lead

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of lead in drinking water should not exceed 0.01 mg/L.

GENERAL DESCRIPTION

Lead can be present in drinking water as a result of dissolution from natural sources, or from household plumbing systems containing lead. These may include lead in pipes, or in solder used to seal joints. The amount of lead dissolved will depend on a number of factors including pH, water hardness and the standing time of the water.

Lead is the most common of the heavy metals and is mined widely throughout the world. It is used in the production of lead acid batteries, solder, alloys, cable sheathing, paint pigments, rust inhibitors, ammunition, glazes and plastic stabilisers. The organo-lead compounds tetramethyl and tetraethyl lead are used extensively as anti-knock and lubricating compounds in gasoline.

Drinking water concentrations of lead reported overseas are usually less than 0.002 mg/L, but concentrations of 0.1 mg/L have been reported in Scotland where lead pipes and soft, acidic water are contributing factors.

Approximately 80% of the daily intake of lead is from the ingestion of food, dirt and dust. Food contains small but significant quantities of lead, which can increase when acidic food is stored in lead-glazed ceramic pottery or lead-soldered cans. The use of lead-free solders is becoming more widespread in the food processing industry. The average Australian adult dietary intake of lead is approximately 0.1 mg per day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, total lead concentrations range up to 0.01 mg/L, with typical concentrations less than 0.005 mg/L.

TREATMENT OF DRINKING WATER

Lead concentrations in drinking water can be reduced by conventional methods of water treatment using coagulants or lime softening.

MEASUREMENT

The concentration of lead in drinking water can be determined by graphite furnace atomic absorption spectroscopy (APHA Method 3500-Pb Part B 1992). The limit of determination is 0.005 mg/L.

HEALTH CONSIDERATIONS

Lead can be absorbed by the body through inhalation, ingestion or placental transfer. In adults, approximately 10% of ingested lead is absorbed but in children this figure can be 4 to 5 times higher. After absorption, the lead is distributed in soft tissue such as the kidney, liver, and bone marrow where it has a biological half-life in adults of less than 40 days, and in skeletal bone where it can persist for 20 to 30 years.

In humans, lead is a cumulative poison that can severely affect the central nervous system. Infants, fetuses and pregnant women are most susceptible. Placental transfer of lead occurs in humans as early as the 12th week of gestation and continues throughout development.

Many epidemiological studies have been carried out on the effects of lead exposure on the intellectual development of children. Although there are some conflicting results, on balance the studies demonstrate that exposure to lead can adversely affect intelligence.

These results are supported by experiments using young primates, where exposure to lead causes significant behavioural and learning difficulties of the same type as those observed in children.

Other adverse effects associated with exposure to high amounts of lead include kidney damage, interference with the production of red blood cells, and interference with the metabolism of calcium needed for bone formation.

Epidemiological studies have found no association between lead and tumour incidence. Kidney tumours, however, have been reported in rats, mice and hamsters fed lead salts in their diet, but only at doses above 27 mg/kg body weight per day. Gliomas (brain tumours) have also been reported in rats. In addition, lead salts given orally to rats have increased the carcinogenic activity of known carcinogens.

Tests for mutagenicity using strains of bacteria have largely been negative. Tests using mammalian cells have been inconclusive, with some studies reporting negative results and some reporting chromosome damage.

The International Agency for Research on Cancer has concluded that lead is possibly carcinogenic to humans (Group 2B, inadequate human data but sufficient evidence in animals for inorganic lead compounds) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value for lead in drinking water is based on a World Health Organization assessment and was determined by the need to protect young children, infants and pregnant women, the groups most at risk. The value was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.0035 \text{ mg/kg body weight per day} \times 13 \text{ kg} \times 0.2}{1 \text{ L/day}}$$

where:

- 0.0035 mg/kg body weight per day is the lead intake which, based on metabolic studies with infants, does not result in an increase in lead retention (Ziegler *et al*. 1978, Ryu *et al*. 1983).
- 13 kg is the average weight of a child at 2 years of age.
- 0.2 is the proportion of total lead intake attributable to water consumption. Sufficient data are available to indicate that 80% of intake is from food, dirt and dust.
- 1 L/day is the average amount of water consumed by a young child.

The NHMRC in 1993 established guidelines for lead in Australians, which provide the basis for establishing acceptable levels of lead in air, food, soil and water. Pending an assessment of the impact of this review on the guideline value for lead, the guideline should be regarded as an interim value.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 3500-Pb Part B (1992). Lead: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

NHMRC (National Health and Medical Research Council) (1993) Revision of Australian Guidelines for Lead in Blood and Lead in Ambient Air, 115th Session, NHMRC, www.nhmrc.gov.au/publications/reports/eh8.pdf

Ryu JE, Ziegler EE, Nelson SE, Fomon SJ (1983). Dietary intake of lead and blood lead concentration in early infancy. *American Journal of Disease of Children*, 137:886–891.

Zeigler EE, Edwards BB, Jensen RL, Mahaffey KR, Fomon SJ (1978). Absorption and retention of lead by infants. *Pediatric Research*, 12:29–34.

NOTE: Important general information is contained in PART II, Chapter 6

Lindane

(endorsed 2011)

GUIDELINE

Based on human health concerns, lindane in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Lindane (CAS 58-89-9) belongs to the cyclodiene organochlorine class of chemicals. Another pesticide in this class is endosulfan. Lindane exists in a number of isomeric forms – lindane technical is the gamma stereoisomer; however it contains 0.5% as the alpha, beta, and delta isomers (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, lindane would not be a health concern unless the concentration exceeded 0.01 mg/L. Excursions above this level even for a relatively short period are of concern, as the health-based guideline is based on effects observed in a 3-month dietary study.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Lindane is an insecticide for the control of grubs and symphylids in pineapple plantations.

There is at least one registered product containing lindane in Australia. Lindane products are intended for professional use and are available as a pre- and post-emergent insecticide on pineapple crops. They are to be diluted and applied to soil using ground or aerial spray, or by soil fumigation. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to lindane and its metabolites is residues in pineapples. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of lindane may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Lindane has been occasionally reported in Australian drinking waters in concentrations of less than 1 µg/L. Lindane has been detected indrinking water at similar concentrations in many other parts of the world (Aydin and Yurdun 1999, Badach *et al.* 2000, Na *et al.* 2006, Badach *et al.* 2007, Thacker *et al.* 2008).

TREATMENT OF DRINKING WATER

No specific data on the treatment of lindane in drinking water have been identified.

MEASUREMENT

Lindane may be measured in drinking waters by gas chromatography–mass spectrometry, with a limit of detection 0.01 µg/L (WHO 2004, Van Hoof *et al.* 2001). Alternatively, extraction from water may be undertaken using solid phase micro-extraction (Arrebola *et al.* 2004).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for lindane is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.31 mg/kg bw/day from a short-term (3-month) dietary study in rats. The NOEL is based on kidney tubule distension, nephritis, increased liver weight and centrilobular hypertrophy. The ADI incorporates a safety factor of 100, and was established in 1986.

The previous ADI of 0.01 mg/kg bw was established in 1975, based on a NOEL of 1.25 mg/kg bw/day from a long-term study in rats. The ADI was amended after submission of a three-month dietary study in rats that demonstrated a lower overall NOEL.

The previous *Australian Drinking Water Guidelines* health value was 0.02 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Lindane is rapidly absorbed from the gastrointestinal tract of rats, with wide distribution to tissues and moderate accumulation in fats. Metabolism is extensive, with hydroxylation and conjugation being the main pathways in humans and other mammals. Excretion is mostly through urine.

Acute effects: Lindane has high acute oral toxicity and moderate dermal toxicity. It is not a skin sensitiser in humans or guinea pigs.

Short-term effects: A 3-month oral study in rats reported kidney tubule distension, nephritis, increased liver weight and centrilobular hypertrophy at 1.6 mg/kg bw/day. Liver lesions were fully recovered and kidney lesions partially recovered after 6 weeks. The overall NOEL was 0.31 mg/kg bw/day and this NOEL is the basis for the current ADI.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. No adverse effects were reported in mice up to 10 mg/kg bw/day. Liver toxicity in the form of hepatocyte hypertrophy, fatty infiltration, and necrosis was reported in rats at doses of 2 mg/kg bw/day, and cerebellar vacuolisation, nephritis and glomerular fibrosis at 6 mg/kg bw/day. Fibrosis in liver and kidney tissue was reported at doses of 3.75 mg/kg bw/day and increased liver weight at 15 mg/kg bw/day in dogs.

Carcinogenicity: The liver tumours noted at high dose levels in mice were not considered relevant to humans at the normal levels of exposure. There was no evidence of carcinogenicity for lindane based on a 2-year dietary study in rats.

Genotoxicity: Lindane is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats, mice and rabbits did not produce any evidence for effects on reproductive parameters or foetal development.

Poisons Schedule: Lindane is included in Schedule 2, 4, 5, and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for lindane was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.31 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.31 mg/kg bw/day is the NOEL based on a medium-term (3-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a health-based guideline value of 0.002 mg/L for lindane (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Arrebola FJ, Aguado SC, Sanchez-Morito N, Frenich AG, Vidal JLM (2004). Pesticide residue analysis in waters by solid-phase microextraction coupled to gas chromatography-tandem mass spectrometry. *Analytical Letters*, 37(1):99-117.

Aydin A, Yurdun T (1999). Residues of organochlorine pesticides in water sources of Istanbul. *Water, Air, and Soil Pollution*, 111(1-4):385-398.

Badach H, Nazimek T, Kaminska IA (2007). Pesticide content in drinking water samples collected from orchard areas in Central Poland. *Annals of Agricultural and Environmental Medicine*, 14(1):109-114.

Badach H, Nazimek T, Kaminski R, TurskiWA (2000). Organochlorine pesticides concentration in the drinking water from regions of extensive agriculture in Poland. *Annals of Agricultural and Environmental Medicine*, 7(1):25-28.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Na T, Fang Z, Zhanqi G, Ming Z, Cheng S (2006). The status of pesticide residues in the drinking water sources in Meiliangwan Bay, Taihu Lake of China. *Environmental Monitoring and Assessment*, 123(1-3), 351-370.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Thacker N, Bassin J, Deshpande V, Devotta S (2008). Trends of organochlorine pesticides in drinking water supplies. *Environmental Monitoring and Assessment*, 137(1-3):295-299.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Van Hoof F, Van Wiele P, Bruchet A, Schmitz I, Bobeldiji I, Sacher F, Ventura F, Marti I, Do Monte MHM, Da Costa MS (2001). Multiresidue determination of pesticides in drinking and related waters by gas chromatography/mass spectrometry after solid-phase extraction: Interlaboratory study. *Journal of AOAC. International*, 84(5):1420-1429.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Maldison (Malathion)

(endorsed 2011)

GUIDELINE

Based on human health concerns, maldison in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Maldison (malathion)(CAS 121-75-5) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, maldison would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Maldison is an insecticide and parasiticide for the control of various insect pests such as fruit fly and locusts on various crops, and in grain storage facilities. It is also used on dogs and cats and in aviaries for treatment of lice, brown dog tick and mange.

There are registered products containing maldison in Australia. The products are intended for both professional and home garden/veterinary use. Products are formulated as liquid concentrates (to be diluted and sprayed), dusts, lures/traps and insecticidal washes. Aerial ultra-low volume (ULV) application is permitted, as well as ground-based ULV application. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to maldison and its metabolites are the use of home garden and home veterinary products, and residues in food. Residue levels in food produced according to good agricultural practice are anticipated to be generally low.

Agricultural use of maldison may potentially lead to contamination of source waters through processes such as run-off, spray drift (especially from aerial and ULV application) or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on maldison in Australian waters. Maldison was not detected in surveys of municipal and private drinking-water supplies conducted in Canada between 1971 and 1986 (Health Canada 1989). It was detected in 4 of 949 stream samples in southern Ontario agricultural watersheds at concentrations of 0.24 to 1.8 µg/L (Health Canada 1989). In the USA, maldison has been reported in surface water at levels up to 0.18 µg/L and in drinking-water at 0.1 µg/L (ATSDR 2000, WHO 2004a).

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of maldison in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective. Maldison has been shown to have relatively high removal rates when water undergoes advanced oxidation with iron-catalysed ultraviolet irradiation and peroxide (Fenton reaction) (Huston and Pignatello 1999).

MEASUREMENT

Maldison can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for maldison is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2 mg/kg bw/day from a 2-year dietary study in rats. This NOEL is based on inhibition of red blood cell cholinesterase. The ADI incorporates a safety factor of 100 and it was established in 2005.

The previous ADI was 0.02 mg/kg bw/day, based on a NOEL of 0.26 mg/kg bw/day for inhibition of red blood cell and plasma cholinesterase in a human study and a safety factor of 10. The NOEL in this study was not maintained due to the absence of information regarding the purity of the material tested.

The acute reference dose (ARfD) of 1.5 mg/kg bw/day for maldison was established in 2005, based on a NOEL of 15 mg/kg bw/day from an acute dietary study in humans. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Maldison is rapidly and extensively absorbed from the gastrointestinal tract. Metabolism is extensive, with oxidation to malaoxon, and further hydroxylation to another six to eight metabolites. Maldison has a low potential for accumulation, with <1% of the dose present in blood and tissues after 72 hours.

Acute effects: Maldison has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pigs, although there is some evidence of skin sensitisation in humans.

Short-term and long-term effects: Short-term and long-term dietary studies in rats report cholinesterase inhibition as the main toxicological effect. In a 3-month study, cholinesterase inhibition occurred at 34 mg/kg bw/day and above. In a 2-year study, inhibition of red blood cell cholinesterase occurred at 29 mg/kg bw/day and above. The NOEL of 2 mg/kg bw/day in this rat study is the basis for the current ADI.

A 56-day human study reported inhibition of red blood cell cholinesterase at a dose of 0.4 mg/kg bw/day.

Carcinogenicity: Adenomas were reported in the liver in mice and rats and in the thyroid in rats at high dose levels, but these were considered rodent-specific. There was no other evidence of carcinogenicity for maldison.

Genotoxicity: Maldison is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits reported no evidence of effect on reproductive parameters or foetal development.

Neurotoxicity: Maldison did not cause delayed neurotoxicity in hens.

Poisons Schedule: Maldison (Malathion) is included in Schedule 3, 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for maldison was determined as follows:

$$0.07 \text{ mg/L} = \frac{2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health value for malathion and it is excluded from the list of agricultural chemicals guideline value derivation because it "occurs in drinking-water at concentrations well below those at which toxic effects may occur" (WHO 2004b).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

ATSDR (Agency for Toxic Substances and Disease Registry) (2000). Toxicological profile information sheet – Malathion. Draft for public comment. Atlanta, GA, US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1989). Guidelines for Canadian Drinking Water Quality – Technical Documents: Malathion. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/malathion/]

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction. *Water Research* 33(5):1238-1246.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

NOTE: Important general information is contained in PART II, Chapter 6

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004a). Malathion in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. WHO.

WHO (World Health Organization) (2004b). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Mancozeb

(endorsed 2011)

GUIDELINE

Mancozeb degrades in the environment to ethylene thiourea (ETU), hence the health-based guideline for mancozeb has been based on the toxicity of ETU. Based on human health concerns, ETU in drinking water should not exceed 0.009 mg/L.

RELATED CHEMICALS

Mancozeb (CAS 8018-01-7; Ethylene thiourea CAS 96-45-7) belongs to the ethylenebis-dithiocarbamate class of chemicals. Other pesticides in this class include metiram and zineb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, the environmental degradant of mancozeb, ethylene thiourea (ETU), would not be a health concern unless the concentration exceeded 0.009 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Mancozeb is a broad spectrum fungicide for the control of fungal diseases in tree fruits, vegetable crops, field crops, and grapes, sod crops (lawns), ornamental plants and harvested seed.

There are registered products that contain mancozeb in Australia. The products are intended for professional use and are available as concentrated solutions to be applied in diluted form using ground and aerial spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: Mancozeb hydrolyses rapidly in the environment to ETU and carbon disulfide (CS_2), both of which have higher toxicity than mancozeb. It is considered highly unlikely that residues of mancozeb or its degradants will be present in food. Mancozeb residues are grouped with other dithiocarbamates (mancozeb, metham, metiram, propineb, thiram, zineb and ziram) in the maximum residue limit definition.

Agricultural use of mancozeb may potentially lead to contamination of source waters by both mancozeb and ETU through adsorption into soil and subsequent entry into ground water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for mancozeb or ETU in Australian waters were found. In the USA, the predicted surface and groundwater concentrations for ETU were 0.1–25.2 µg/L and 0.21 µg/L, respectively (USEPA 2005). The parent compound is short-lived in soil and water and is not expected to reach water used for human consumption (USEPA 2005).

TREATMENT OF DRINKING WATER

Chlorine dioxide and ozonation appear moderately effective at removing mancozeb and ETU from water, however these processes produce as-yet undefined degradation products (Hwang *et al.* 2003).

MEASUREMENT

Mancozeb can be measured in water by liquid chromatography mass spectrometry with an electrospray interface, with a limit of detection of 0.04 µg/L (Hanada *et al.* 2002). ETU can be measured by gas chromatrography with nitrogen-phosphorous detector, with a limit of detection of 2.7 µg/L (USEPA 1992).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for mancozeb is 0.006 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.6 mg/kg bw/day from a long-term (1-year) dietary study in dogs. The NOEL is based on decreased iodine uptake into thyroid tissue at doses of 2.4 mg/kg bw/day and above. The ADI incorporates a safety factor of 100 and was first established in 1993. There is currently no ADI for ETU.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Mancozeb is rapidly and extensively absorbed from the gastrointestinal tract of rats and widely distributed in tissues, particularly the thyroid. A small percentage (3–6%) is metabolised to ETU. Excretion is rapid, mainly as unchanged compound in faeces and urine, and is almost complete by 5 days. ETU is also excreted in the urine.

Acute effects: Mancozeb is of low acute oral and dermal toxicity in mammals. It is a skin sensitiser in humans, based on reports from occupational exposure. ETU has low acute oral toxicity.

Short-term effects: Short-term dietary studies with mancozeb were conducted in mice, rats and dogs. Thyroid hyperplasia was observed in rats at 7.5 mg/kg bw/day. Histopathological changes were also observed in other organs at 30 mg/kg bw/day and above in dogs and mice. In rats, organ weight and histopathological changes were observed in several organs at 60 mg/kg bw/day, as well as decreased serum thyroxine levels. In dogs, decreased bodyweight gain and serum thyroxine levels, as well as clinical chemistry changes were seen at 150 mg/kg bw/day.

In short-term studies with ETU, the thyroid was the target organ. In a dietary study in rats over 14 days, histological changes including thyroid hyperplasia, bone marrow depletion, and lymphatic lesions occurred from 25 mg/kg bw/day. When administered in the drinking water of rats over 28 days, decreased levels of thyroxine and serum triiodothyronine, increased levels of thyroid stimulating hormone in serum, and thyroid follicular necrosis were seen at doses from 10.6 mg/kg bw/day. Other effects seen at 17.6 mg/kg bw/day and above include proximal tubule kidney cell hypertrophy and vacuolisation.

In 3- and 4-month dietary studies in rats and mice with ETU, effects in rats included follicular cell hypertrophy and thyroid hyperplasia from 3 mg/kg bw/day and above. At higher doses there was also increased relative thyroid weight and decreased iodine uptake into the thyroid at 8 mg/kg

NOTE: Important general information is contained in PART II, Chapter 6

bw/day; decreased levels of thyroxine and increased levels of thyroid stimulating hormone in serum, and increased absolute thyroid weights at 10 mg/kg bw/day; and thyroid adenomas at 12.5 mg/kg bw/day. In mice, thyroid adenomas and pituicyte vacuolisation occurred from 12.5 mg/kg bw/day, hepatocellular hypertrophy from 37.5 mg/kg bw/day, and thyroid hyperplasia from 75 mg/kg bw/day. The lowest overall NOEL was 2 mg/kg bw/day (rats) in these studies.

Long-term effects: Long-term dietary studies with mancozeb were conducted in mice, rats and dogs. Decreased iodine uptake into the thyroid was reported at 2.4 mg/kg bw/day in dogs and thyroid hyperplasia at 3 mg/kg bw/day in rats. In mice, effects were confined to decreased bodyweight gain at 150 mg/kg bw/day. The lowest overall NOEL was 0.6 mg/kg bw/day in dogs. This NOEL is the basis for the current ADI.

In a 1-year rat study with ETU, there was increased thyroid vascularisation and thyroid acinar cell papillation at the lowest dose tested of 0.025 mg/kg bw/day. At 1.25 mg/kg bw/day, there was decreased bodyweight gain. Increased relative thyroid weight occurred at 7 mg/kg bw/day and thyroid tumours at 15 mg/kg bw/day.

In a 2-year rat study with ETU, thyroid hyperplasia, elevated TSH and decreased triiodothyronine and thyroxine were seen at the lowest dose of 0.25 mg/kg bw/day. Thyroid carcinomas were observed at 8.7 mg/kg bw/day. In a 2-year mouse study with ETU, decreased bodyweight gain, increased TSH, thyroid cell hypertrophy and hyperplasia and thyroid adenomas and carcinomas were observed at the lowest dose tested of 16 mg/kg bw/day.

Carcinogenicity: Based on long-term studies in rats with both mancozeb and ETU, there was evidence of an increased incidence of tumours (adenomas and carcinomas) in the thyroid at high dose levels only, which are well in excess of the likely level of human exposure. In mice, ETU produced thyroid follicular-cell tumours and tumours of the liver and anterior pituitary gland. However, due to its nongenotoxicity and disturbance of thyroid function, ETU would not be expected to produce thyroid cancer in humans exposed to concentrations that do not alter thyroid hormone homeostasis.

Genotoxicity: Mancozeb is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies. ETU was positive in some *in vitro* short-term assays, but overall, it is not considered to be genotoxic.

Reproductive and developmental effects: Two and three-generation reproduction studies in rats and developmental studies in mice, rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development. Developmental studies in rats and rabbits with ETU caused effects on development only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Mancozeb is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.009 mg/L for the degradant of mancozeb, ETU, was determined as follows:

$$0.009 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 1}{2 \text{ L/day} \times 1000}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 0.25 mg/kg bw/day is the LOEL based on a long-term (2-year) dietary study in rats on ETU.
- 70 kg is taken as the average weight of an adult.
- The proportionality factor is 1 since mancozeb has no residues in food and is degraded to ETU in the environment. It is assumed, therefore, that 100% of the ADI (nominal in this case) for ETU will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the LOEL for ETU derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation, and an additional factor of 10 because a LOEL was used to derive the guideline.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hanada Y, Tanizaki T, Koga M, Shiraishi H, Soma M (2002). LC/MS studies on characterization and determination of N,N'-ethylenebisdithiocarbamate fungicides in environmental water samples. *Analytical Sciences*, 18:441-444.

Hwang ES, Cash JN, Zabik MJ (2003). Determination of degradation products and pathways of mancozeb and ethylenethiourea (ETU) in solutions due to ozone and chlorine dioxide treatments. *Journal of Agricultural and Food Chemistry,* 51(5):1341-1346.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1992). Method 509 - Determination of Ethylene Thiourea (ETU) in Water Using Gas Chromatography with a Nitrogen-Phosphorus Detector. Revision 1.0.

USEPA (United States Environmental Protection Agency) (2005). Reregistration Eligibility Decision for Mancozeb. EPA 738-R-04-012.USEPA.

Manganese

(endorsed 2011)

GUIDELINE

Based on aesthetic considerations, the concentration of manganese in drinking water should not exceed 0.1 mg/L, measured at the customer's tap.

Manganese would not be a health consideration unless the concentration exceeded 0.5 mg/L.

GENERAL DESCRIPTION

Manganese is present in the environment in the divalent (Mn(II)), tetravalent (Mn(IV)), and heptavalent (Mn(VII)) states. Most of the divalent compounds are soluble in water. The most common tetravalent compound, manganese dioxide, is insoluble; however, the heptavalent permanganate is soluble.

Manganese is principally used in the manufacture of iron, steel and alloys.

Uncontaminated rivers and streams generally have low concentrations of manganese, ranging from 0.001 mg/L to 0.6 mg/L. High concentrations may occur in polluted rivers or under anoxic conditions such as at the bottom of deep reservoirs or lakes, or in groundwater.

At concentrations exceeding 0.1 mg/L, manganese imparts an undesirable taste to water and stains plumbing fixtures and laundry. Even at concentrations of 0.02 mg/L, manganese will form a coating on pipes that can slough off as a black ooze. Some nuisance microorganisms can concentrate manganese and give rise to taste, odour and turbidity problems in distribution systems. A discretionary target of 0.01 mg/L is suggested at the treatment plant.

Manganese interferes with the commonly used DPD method for determining chlorine residual, resulting in an overestimation of the residual so that chlorine appears to be present when it may not be.

Concentrations of manganese in food can vary considerably. The highest concentrations have been reported in grains, nuts and vegetables, while tea leaves can have extremely high concentrations.

It has been estimated that the average dietary intake of manganese is 2–4 mg per day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, manganese concentrations can range up to 1.41 mg/L, with typical concentrations less than 0.01 mg/L. For regional NSW, for example, a median value of 0.005 mg/L was found over a nine-year period.

TREATMENT OF DRINKING WATER

Manganese concentrations in drinking water can be lowered by converting soluble forms to insoluble precipitates, followed by filtration. Manganese levels below 0.02 mg/L can be achieved with a well operated and optimised potassium permanganate system. Achieving <0.01 mg/L manganese in treated water is not possible with potassium permanganate alone, so high pH coagulation processes or a two-stage filtration process are employed at several plants; however, this process will not be suitable for all waters. Pre-filtration chlorination can help to achieve a target of 0.01 mg/L.

MEASUREMENT

The manganese concentration in drinking water can be determined using inductively coupled plasma emission spectroscopy or graphite furnace atomic absorption spectroscopy (APHA Method 3500-Mn Parts B or C, 1992). The limits of determination are 0.005 mg/L and 0.001 mg/L respectively.

HEALTH CONSIDERATIONS

Manganese is an essential element and is required by mammals and birds for normal growth. Manganese deficiency affects bone, the brain and reproduction in a number of animal species. Although no specific symptoms have been described in humans, it has been suggested that manganese deficiency may be associated with anaemia and, in children, with bone disorders.

Owing to the low solubility of manganese in gastric juices, only 3-8% of ingested manganese is absorbed by the gastrointestinal tract. After absorption, it is concentrated in the liver and eventually excreted in faeces. In humans it has a relatively short biological half-life of 13 to 37 days.

An extensive review and summary of the human and animal toxicity data for manganese is available (IPCS 1981).

In humans, manganese toxicity has occurred mainly as a result of inhalation of manganese dust over long periods. By the oral route, manganese is regarded as one of the least toxic elements.

In one case involving heavy consumption of highly contaminated well water, resulting symptoms included lethargy, increased muscle tone, tremor and mental disturbances. Concentrations of manganese were over 14 mg/L; however, concentrations of other metals were also high and the reported effects may not have been due solely to manganese.

Experiments with laboratory animals have shown no adverse effects other than a change in appetite and a reduction in the metabolism of iron in haemoglobin synthesis.

There is no firm evidence that manganese is carcinogenic. Some studies indicate that it may, in fact, have an anticarcinogenic effect. Some *in vitro* studies using mammalian and bacterial cells have reported that manganese acts as a mutagen.

DERIVATION OF GUIDELINES

The aesthetic guideline of 0.1 mg/L at the customer's tap is based on practical experience and has been reported by utilities to be acceptable to customers. The discretionary target of 0.01 mg/L at the treatment plant is also based on experience; that although manganese accumulates in distribution systems, a plant producing 0.01 mg/L generally does not generate customer complaints, while a concentration of 0.02 mg/L or more tends to lead to various problems.

The health-based guideline value for manganese in drinking water can be derived as follows:

$$0.5 \text{ mg/L} = \frac{10 \text{ mg/day} \times 0.1}{2 \text{ L/day}}$$

where:

- 10 mg/day is the amount of manganese that can be safely consumed from all sources (WHO 1973).
- 0.1 is a proportionality factor based on the assumption that 10% of daily intake is attributable to the consumption of water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.

The maximum tolerable daily intake value includes adequate safety factors, so no additional safety factors are necessary. This value exceeds the concentration at which manganese can cause taste and odour problems.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 3500-Mn Part B (1992). Manganese: Atomic Absorption Spectrometric method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Mn Part C (1992). Manganese: Inductively Coupled Plasma method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

IPCS (International Programme on Chemical Safety) (1981). Manganese. Environmental Health Criteria, 17. World Health Organization, IPCS.

WHO (World Health Organization) (1973). Trace elements in human nutrition: Manganese. Report of the WHO Expert Committee, Technical Report Service, 532, 34-36, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

MCPA

(endorsed 2011)

GUIDELINE

Based on human health concerns, MCPA in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

2-methyl-4-chlorophenoxyacetic acid (MCPA)(CAS 94-74-6) belongs to the phenoxycarboxylic acid class of chemicals. Other pesticides in this class include 2,4-D, 2,4-DB, dichlorprop, dichlorprop-P, MCPB and mecoprop (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, MCPA would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: MCPA is a herbicide for the control of various broad-leaf weeds in crops, pastures and turf.

There are many registered products that contain MCPA or its salt/variants in Australia. The products are intended for professional and home garden use and are generally available as emulsifiable concentrates, aqueous concentrates and liquids to be applied using ground boom, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to MCPA are the use of home garden products and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of MCPA may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for MCPA in Australian waters could be found, however it has occasionally been measured in some Australian drinking-water supplies at concentrations generally less than 1 µg/L. In the USA, MCPA was detected up to 0.54 µg/L in surface waters and up to 5.5 µg/L in groundwater (WHO 2003).

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficacy could be found for MCPA, although activated carbon is expected to be quite effective, based on its structure.

MEASUREMENT

MCPA can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.01 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for MCPA is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.1 mg/kg bw/day from a 2-year dietary rat study. The NOEL is based on evidence of mild liver effects (changes in clinical chemical parameters). The ADI incorporates a safety factor of 100, and was established in 1994.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: MCPA is readily absorbed via the gastrointestinal tract. It is not extensively metabolised, and is rapidly excreted, mainly unchanged, in the urine.

Acute effects: MCPA has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 13-week dietary studies in rats and dogs, there was evidence of kidney and liver damage. In rats, increased kidney weights were reported at 7.5 mg/kg bw/day and increased creatinine and decreased calcium at 22.5 mg/kg bw/day. In dogs, increased serum glutamic pyruvic transaminase, blood urea nitrogen and creatinine levels were reported at 3 mg/kg bw/day. Decreased bodyweight gain and bile duct proliferation were reported at 12 mg/kg bw/day, and gross pathological changes in the liver and microscopic changes in both the liver and kidney at 48 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice and rats. A 2-year mouse study reported pathological changes in the kidney at 71.4 mg/kg bw/day. A 2-year rat study reported an increase in serum glutamic pyruvic transaminase levels at 4 mg/kg bw/day. At 16 mg/kg bw/day there was a slight decrease in bodyweight gain, haemosiderosis in the spleen, and an increase in absolute kidney weight in males, accompanied by evidence of chronic nephropathy. The NOEL of 1.1 mg/kg bw/day in the rat study is the basis for the current ADI.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for MCPA.

Genotoxicity: MCPA is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats reported reduced pup bodyweight gain at 15 mg/kg bw/day and above. Developmental studies in rats and rabbits reported decreased bodyweight gain and increased post-implantation loss at 75 mg/kg bw/day. There were no effects at lower dose levels on reproductive parameters or foetal development.

Poisons Schedule: MCPA is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for MCPA was determined as follows:

$$0.04 \text{ mg/L} = \frac{1.1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.1 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a health-based guideline value of 0.002 mg/L for MCPA (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2003). MCPA in Drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality. WHO.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Mercury

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of total mercury in drinking water should not exceed 0.001 mg/L.

GENERAL DESCRIPTION

Natural release of mercury into drinking water is extremely low, but contamination can result from industrial emission or spills. Mercury compounds fall into two categories: inorganic mercury salts, many of which are very insoluble in water; and organic mercury compounds, the most notable being methyl mercury. Inorganic mercury can be converted into methyl mercury, possibly by the action of bacteria in sediments, and can then readily enter the food chain.

Mercury is used widely in electrical components including cells, lamps, arc rectifiers and switches. It is also used in dental amalgams, fungicides, antiseptics, preservatives and pharmaceuticals.

Concentrations of total mercury in natural water are generally so low that accurate analysis is difficult. Studies overseas have reported concentrations of less than 0.0005 mg/L, with some sources less than 0.00003 mg/L (30 ng/L). The highest value was 0.0055 mg/L from some wells in Japan.

Food is the main route of exposure, with highest concentrations found in fish and fish products. The average Australian adult dietary intake of mercury is approximately 0.004 mg per day. Drinking water is likely to constitute only a small fraction of total intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, the concentrations of total mercury range up to 0.001 mg/L, with typical concentrations usually less than 0.0001 mg/L.

TREATMENT OF DRINKING WATER

Coagulation is moderately effective in reducing the concentration of inorganic mercury in drinking water. Granular activated carbon is effective in removing both inorganic and organic mercury from water.

MEASUREMENT

The concentration of total mercury in drinking water can be determined by the cold vapour atomic absorption method (APHA 3500-Hg Part B 1992). The limit of determination is 0.0001 mg/L.

HEALTH CONSIDERATIONS

Inorganic mercury

Less than 15% of inorganic mercury in drinking water is absorbed by the gastrointestinal tract. Inorganic mercury compounds accumulate in the kidney and have a long biological half-life, probably many years.

An extensive review and summary of the human and animal toxicity data for inorganic mercury is available (IPCS 1991).

Many studies have looked at groups of workers occupationally exposed to mercury, and have reported health effects including tremors, mental disturbances and gingivitis (inflammation of the mucous membrane surrounding the teeth). The main toxic effects are to the kidney, leading to kidney failure.

In animal studies, the principal target organs of mercury toxicity are the kidney and the central nervous system. Some disruption to ovulation in female rats has also been reported.

Various reports indicate that inorganic mercury binds to, and damages, mammalian DNA. Some evidence of carcinogenicity in rats has been reported.

Organic mercury

Organic mercury compounds are unlikely to be found in uncontaminated drinking water; however, the toxic effects are more severe than those of inorganic mercury.

An extensive review and summary of the human and animal toxicity data for methyl mercury is available (IPCS 1990).

Methyl mercury compounds are almost completely absorbed by the gastrointestinal tract. Methyl mercury has greater lipid solubility than inorganic mercury and can cross biological membranes, especially in the brain, spinal cord, peripheral nerves and placenta.

The main effects of methyl mercury poisoning are severe irreversible neurological disorder and mental disability.

In Japan, two major epidemics of methyl mercury poisoning, known as Minamata disease, were caused by the industrial release into Minamata Bay of methyl mercury and other mercury compounds. The compounds accumulated in fish, which were subsequently eaten by humans. Other countries have reported cases of poisoning caused by mercury contamination of bread and cereal.

Animal studies with rats, cats, monkeys and squirrels have shown similar results, with the main effects of long-term exposure being behavioural changes, neurological disturbances and disturbances to the movement of legs and tails.

Data are insufficient to determine the carcinogenic effects of methyl mercury; however, it is active in inducing chromosomal aberrations *in vivo*.

DERIVATION OF GUIDELINE

The guideline value for mercury in drinking water was derived as follows:

$$0.001 \text{ mg/L} = \frac{0.00047 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day}}$$

where:

- 0.00047 mg/kg body weight per day is the maximum tolerable daily intake to ensure that adverse effects will not occur (WHO 1988).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

The maximum tolerable daily intake value includes adequate safety factors. No additional safety factors are necessary.

The guideline value was set on the basis of the toxicity of methyl mercury, as this is the most toxic form. It is likely that methyl mercury would be less than 10% of the total mercury concentration.

NOTE: Important general information is contained in PART II, Chapter 6

The guideline value should be sufficient to protect pregnant women and nursing mothers, who are at greatest risk from the adverse effects of methyl mercury. Data are insufficient to determine a separate value for this group.

REFERENCES

APHA Method 3500-Hg Part B (1992). Mercury: Cold vapor Atomic Absorption method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IPCS (International Programme on Chemical Safety) (1990). Methyl Mercury. Environmental Health Criteria, 101. World Health Organization, IPCS.

IPCS (International Programme on Chemical Safety) (1991). Inorganic Mercury. Environmental Health Criteria, 118. World Health Organization, IPCS.

WHO (World Health Organization) (1988). Toxicological evaluation of certain food additives and contaminants: Methylmercury. 33rd meeting of the Joint FAO/WHO Expert Committee on Food Additives. WHO Food Additive Series, 24, 296–328.

NOTE: Important general information is contained in PART II, Chapter 6

Metaldehyde

(endorsed 2011)

GUIDELINE

Based on human health concerns, metaldehyde in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Metaldehyde (CAS 108-62-3) belongs to aldehyde class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, metaldehyde would not be a health concern unless the concentration exceeded 0.02 mg/L. Minor excursions above this level would need to occur over a significant period, as the health-based guideline is based on moderate- to long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Users: Metaldehyde is used to control snails and slugs in food crops.

There are registered products containing metaldehyde in Australia. The products are for professional and home garden use and are available in bait or pellet form, as powders, and in granular formations. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to metaldehyde is the use of home garden products.

Agricultural and home garden use of metaldehyde may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of metaldehyde in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of metaldehyde in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

No suitable analytical techniques have been identified for analysis of metaldehyde in drinking water. However, if a need to monitor for metaldehyde is identified, it is expected that gas chromatography–mass spectroscopy should be suitable, since this technique has been used for monitoring metaldehyde in human and animal blood serum.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for metaldehyde is 0.005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day in a rat study and applying a safety factor of 1000. The NOEL was based on neurological effects in rats and was established in 1986.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Metaldehyde is readily absorbed from the gastrointestinal tract. Only a small percentage of the parent compound is excreted in the urine.

Acute effects: Metaldehyde has a moderate acute oral toxicity and low dermal toxicity. Clinical symptoms of toxicity reported in human poisoning cases included salivation, restlessness, muscle cramps and increased heart rate, indicative of mild neurotoxicity. Similar symptoms were observed in animal studies.

Short-term effects: No short-term studies have been evaluated.

Long-term effects: A 2-year dietary study in rats reported liver enzyme changes and increased liver weight at 12 mg/kg bw/day; however, the study was poorly reported.

Carcinogenicity: Based on a long-term study in rats, there is no evidence of carcinogenicity for metaldehyde; however, the study was poorly reported.

Genotoxicity: Only short-term *in vitro* studies are available; these report no evidence that metaldehyde is mutagenic.

Reproductive and developmental effects: A 3-generation reproduction study in rats produced effects on reproductive parameters at extremely high dose levels only. No developmental toxicity studies have been evaluated.

Poisons Schedule: Metaldehyde is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for metaldehyde was determined as follows:

$$0.02 \text{ mg/L} = \frac{5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 5 mg/kg bw/day is the NOEL based on a a long-term (1-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.

NOTE: Important general information is contained in PART II, Chapter 6

- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation and an additional factor of 10 because of the poor quality of the data.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Metham

(endorsed 2011)

GUIDELINE

Metham rapidly degrades to methylisothiocyanate (MITC) in the environment, hence the health-based guideline for metham has been based on the toxicity of MITC. Based on human health concerns, MITC in drinking water should not exceed 0.001 mg/L.

RELATED CHEMICALS

Metham (CAS 144-5-54-7) belongs to the n-methyl-dithiocarbamate class of chemicals. There are no other pesticides in this class. A closely related class of chemicals is ethylenebis-dithiocarbamates, which includes mancozeb, metiram, and zineb. The insecticidal and fungicidal activity of metham sodium is due to degradation to methylisothiocyanate (MITC) in the environment. MITC is also a degradant of the pesticide dazomet (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, the degradation products of metham would not be a health concern unless the concentration exceeded 0.001 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Metham is a herbicide and insecticide for the control of tree roots, and soil-borne pests in ornamental, food and tobacco crops.

There are currently registered fumigant products that contain metham as its sodium salt. The products are for professional use only and are available as various liquids, suspensions or aqueous concentrates. Most products are for use as pre-plant soil fumigants and are applied by soil injection, rotary tiller, spot treatment, and trickle irrigation systems. Some products are for direct addition into pipes in sewage and wastewater collection systems. Methylisothiocyanate was formerly used as a soil fumigant in Australia. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: Metham is highly unstable in the environment and spontaneously hydrolyses to MITC. It is considered highly unlikely that residues of metham itself will be present in food, although it is possible there may be residues of MITC in food.

Agricultural use of metham may potentially lead to contamination of source waters by MITC through absorption into the soil moisture and subsequent leaching into groundwater. Some metham products are also added directly to sewer or wastewater pipes.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on metham occurrence in Australian drinking water supplies were found. Based on the registered use patterns for metham, exposure from drinking water is not expected and no dietary risk mitigation is warranted (USEPA 2009).

TREATMENT OF DRINKING WATER

Powdered activated carbon filtration, granulated activated carbon filtration, and reverse osmosis have been demonstrated to be highly effective processes at removing certain pesticides including dithiocarbamates (USEPA 2001a). Metham has a hydrolysis half-live of less than 1 day in alkaline (pH 9) water and can be removed during lime-soda softening (pH 10~11) by alkaline hydrolysis (USEPA 2001b).

MEASUREMENT

Metham sodium and methyl isothiocyanate (MITC) can be analysed in water by high-performance liquid chromatography (Mullins and Kirkbright 1987, Dhoot *et al*. 1993). These methods can achieve a limit of quantitation (LOQ) of 1 µg/L for MITC and 70 µg/L for metham. Direct immersion solid phase microextraction followed by gas chromatography electron capture detector or gas chromatography nitrogen phosphorus detector analysis can achieve a LOQ of 0.5 µg/L for methyl isothiocyanate in water (Fuster *et al*. 2005).

HISTORY OF THE HEALTH VALUES

An acceptable daily intake (ADI) has not been set for metham since ADIs are required only for pesticides with residues in food.

The current ADI for the degradant MITC is 0.0004 mg per kg body weight (mg/kg bw) based on a no-observed-effect level (NOEL) of 0.04 mg/kg bw/day from a 3-month study in dogs. In this study there were decreased testis weights, increased pancreas weights, and abnormal liver histology at the highest dose tested, 2 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was established in 2004.

An acute reference dose (ARfD) has not been set for metham since ARfDs are required only for pesticides with residues in food.

The current ARfD for the degradant MITC is 0.0005 mg/kg bw/day based on a NOEL of 0.1 mg/kg bw/day from an acute oral dosing study in dogs. At the next highest dose tested of 0.5 mg/kg bw/day and above, haemorrhagic lesions in liver and kidneys were seen at necropsy. The ARfD incorporates a safety factor of 200 and was established in 2004.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Metham sodium is readily and extensively absorbed via the gastrointestinal tract in rats. It is spontaneously hydrolysed to MITC and other alkyl isothiocyanates and isothiocarbamates including 1,3,-dimethylthiourea, ethylenethiourea (ETU), and carbon disulfide by passive chemical processes, and is rapidly excreted in the urine as mercapturic acid derivatives, and in the breath as carbon disulfide, almost completely within 48 hours.

Acute effects: Metham sodium has high acute oral toxicity in mice and moderate acute oral and dermal toxicity in rats. Metham sodium and its breakdown product MITC are skin sensitisers in humans. The breakdown product, MITC, has a similar acute oral and dermal toxicity to metham.

NOTE: Important general information is contained in PART II, Chapter 6

Short-term effects: In a 3-month oral study in dogs with the degradant MITC, an increased incidence and severity of periportal hepatocyte vacuolation and lipid deposition was seen at doses of 0.4 mg/kg bw/day and above, and decreased testis weights and increased pancreas weight at doses of 2 mg/kg bw/day. The NOEL in this study was 0.04 mg/kg bw/day and this is the basis for the current ADI for MITC.

Long-term effects: No long-term studies are available for metham (including metham sodium). In studies where the degradant MITC was added to the drinking water of rats at doses up to 2.7 mg/kg bw/day and in mice up to 27 mg/kg bw/day, the major toxicological findings were decreased food consumption and decreased bodyweight gain at doses of 2.5 mg/kg bw/day in rats, and at 14 mg/kg bw/day in mice.

Carcinogenicity: There are no carcinogenicity studies available for metham. Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for the degradant MITC.

Genotoxicity: Metham sodium and the degradant MITC were positive in some *in vitro* short-term assays, but based on the weight of evidence, neither is considered to be genotoxic.

Reproductive and developmental effects: There are no studies available on reproductive toxicity of metham sodium. A reproduction study in rats with the degradant MITC did not provide any evidence of reproductive toxicity at doses up to 10 mg/kg bw/day. In developmental toxicity studies with metham sodium, there were effects on reproduction and development, but only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Metham and MITC are both included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.001 mg/L for the degradant of metham, MITC, was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.04 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.04 mg/kg bw/day is the NOEL for MITC based on a short-term (3-month) oral study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is the proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NOTE: Important general information is contained in PART II, Chapter 6

Dhoot JS, Del Rosario AR, Tamplin BR (1993). Simultaneous determination and confirmation of sodium N-methyldithiocarbamate (Metham SodiumR) and methyl isothiocyanate in water by high performance liquid chromatography with diode array detection. *International Journal of Environmental Analytical Chemistry,* 53(3):165-171.

Fuster S, Beltran J, Lopez FJ, Hernandez F (2005). Application of solid phase microextraction for the determination of soil fumigants in water and soil samples. *Journal of Separation Science,* 28(1):98-103.

Mullins FG, Kirkbright GF (1987). Determination of sodium N-methyldithiocarbamate (metham sodium) and methyl isothiocyanate in aqueous samples by high-performance liquid chromatography using a micellar mobile phase. *Analyst,* 112(5):701-3.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2001a) The Incorporation of Water Treatment Effects on Pesticide Removal and Transformations in Food Quality Protection Act (FQPA) Drinking Water Assessments, Office of Pesticide Programs Science Policy, USEPA, Washington, D.C.

USEPA (United States Environmental Protection Agency) (2001b) Pesticide Science Policies: Water Treatment Effects on Pesticide Removal and Transformation; Notice of Availability OPP-00731; FRL-6792-9, OPP-00731; FRL-6792-9, USEPA.

USEPA (United States Environmental Protection Agency) (2009) Amended Reregistration Eligibility Decision (RED) for the Methyldithiocarbamate Salts (Metamsodium, Metam-potassium) and Methyl Isothiocyanate (MITC), EPA 738-R-09-310, Prevention, Pesticides and Toxic Substances.

NOTE: Important general information is contained in PART II, Chapter 6

Methidathion

(endorsed 2011)

GUIDELINE

Based on human health concerns, methidathion in drinking water should not exceed 0.006 mg/L.

RELATED CHEMICALS

Methidathion (CAS 950-37-8) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes terbufos, ethion, fenamiphos, and acephate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, methidathion would not be a health concern unless the concentration exceeded 0.006 mg/L. Excursions above this level even for a relatively short period are of concern as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Methidathion is an insecticide for the control of insect pests in orchards, sub-tropical, vegetable, cereal, pasture, cotton, and sunflower agricultural crops.

There are registered products containing methidathion in Australia. The products are intended for professional use, and are available in concentrated solutions, which are diluted and applied to infested areas by ground or aerial spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to methidathion is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of methidathion may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Methidathion was not detected in the Mt Lofty Ranges, the main catchment area for Adelaide's drinking water supply (Oliver *et al.* 2005). No other published reports on methidation occurrence in Australian drinking water supplies were found. Methidathion exposures from water are expected to be non-significant, based on pesticide tolerances (USEPA 2002) and drinking water risk assessment reports (Lewis 2001).

TREATMENT OF DRINKING WATER

Water treatment processes such as granular activated carbon and membranes are capable of removing methidation (USEPA 2000). During chlorination in water treatment plants, methidathion can be oxidised forming oxons and/or other oxidation analogues (Kamel *et al.* 2009).

MEASUREMENT

Methidathion can be extracted from water by liquid/liquid and analysed by gas chromatography coupled with a nitrogen phosphorus detector and flame photometric detector. The method can achieve a limit quantitation (LOQ) of 0.05 µg/L. Methidathion can also be extracted by polypropylene hollow fiber liquid phase microextraction and analysed by gas chromatography with flame thermionic detection. The method can achieve a LOQ of 3 ng/L (Lambropoulou and Albanis 2005). Methidathion can also be accurately quantified by solid-phase extraction (SPE) and liquid chromatography (LOQ 0.01 µg/L) (Wang *et al.* 2007). SPE with multiwalled carbon nanotubes, without the need for chromatographic separation, can achieve a LOQ of 3 µg/L for methidathion (Al-Degs *et al.* 2009). SPE with liquid chromatography and tandem mass spectrometry reported a LOQ of 2.3 µg/L for methidathion (Rodrigues *et al.* 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for methidathion is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.16 mg/kg bw/day from a 90-day dietary study in dogs. The NOEL is based on increased liver cholesterol and decreased red blood cell cholinesterase activity at doses of 1.96 mg/kg bw/day and above. The ADI incorporates a safety factor of 100 and was established in 2004.

The acute reference dose (ARfD) of 0.01 mg/kg bw/day for methidathion was established in 2004, based on a NOEL of 1 mg/kg bw/day from an acute neurotoxicity study based on inhibition of red blood cell and brain cholinesterase activity in rats. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Methidathion is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised via hydrolysis and oxidation to sulfoxides, sulfones, and oxons. Excretion is via the urine and is almost complete within 24 hours.

Acute effects: Methidathion has a moderate to high acute oral toxicity in dogs, rats and mice, and a moderate acute dermal toxicity in rats. It is not a skin sensitiser.

Short-term effects: In a 21-day dermal toxicity study in rabbits, there was decreased red blood cell and brain cholinesterase activity at 10 mg/kg bw/day. In a 4-week oral toxicity study in rats, there was decreased red blood cell cholinesterase activity at 0.83 mg/kg bw/day. In 90-day oral toxicity studies in rats and dogs, there was decreased red blood cell cholinesterase activity and increased levels of cholesterol in the liver at 2 mg/kg bw/day. The lowest overall NOEL was 0.16 mg/kg bw/day in dogs. This is the basis for the current ADI.

In a 6-week oral toxicity study in humans, there was no effect on cholinesterase activity and no clinical signs of toxicity at 0.11 mg/kg bw/day.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: In a 2-year oral toxicity study in mice, there was no evidence of toxicity apart from clinical signs of cholinesterase inhibition at 7 mg/kg bw/day. In 2-year oral toxicity studies in rats and monkeys, there was decreased plasma and red blood cell cholinesterase activity at 1.7 mg/kg bw/day in rats and at 1.0 mg/kg bw/day in monkeys. In a 1-year oral toxicity study in dogs, there were decreases in red blood cell cholinesterase activity and evidence of liver toxicity at 1.3 mg/kg bw/day.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for methidathion.

Genotoxicity: Methidathion is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two- and 3-generation reproduction studies in rats, and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: In a 21-day neurotoxicity study in hens, there was no evidence of delayed neurotoxicity.

Poisons Schedule: Methidathion is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.006 mg/L for methidathion was determined as follows:

$$0.006 \text{ mg/L} = \frac{0.16 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.16 mg/kg bw/day is the NOEL based on a medium-term (90-day) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Al-Degs YS, Al-Ghouti MA, El-Sheikh AH (2009). Simultaneous determination of pesticides at trace levels in water using multiwalled carbon nanotubes as solid-phase extractant and multivariate calibration. *Journal of Hazardous Materials,* 169(1-3):128-35.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Kamel A, Byrne C, Vigo C, Ferrario J, Stafford C, Verdin G, Siegelman F, Knizner S, Hetrick J (2009). Oxidation of selected organophosphate pesticides during chlorination of simulated drinking water. *Water Research,* 43(2):522-34.

NOTE: Important general information is contained in PART II, Chapter 6

Lambropoulou DA, Albanis TA (2005). Application of hollow fiber liquid phase microextraction for the determination of insecticides in water. *Journal of Chromatography A,* 1072(1):55-61.

Lewis CM (2001). *Methidathion: Dietary and drinking water risk assessment*. Medical Toxicology Branch, Department of Pesticide Regulation, Sacramento, CA,

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Oliver D, Kookana R (2005). *Pesticide use in the 6th Creek Sub-catchment, Mt. Lofty Ranges, S.A. and Assessment of Risk of Off-site Movement using Pesticide Impact Rating Index (PIRI)*. CSIRO Land and Water Technical Report, Adelaide.

Rodrigues, A. M., V. Ferreira, V. V. Cardoso, E. Ferreira and M. J. Benoliel. 2007. Determination of several pesticides in water by solid-phase extraction, liquid chromatography and electrospray tandem mass spectrometry. *J Chromatogr A* 1150(1-2), 267-78

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2000). Components and Methodologies of the Calendex™, Lifeline™, and REx Models as Tools for Dietary and Residential Pesticide Exposure and Risk Assessments; and Assessment of Pesticide Concentrations in Drinking Water and Water Treatment Effects on Pesticide Removal and Transformation. Session VI – Progress Report on Estimating Pesticide Concentrations in Drinking Water and Assessing Water Treatment Effects on Pesticide Removal and Transformation. [http://www.epa.gov/scipoly/SAP/tools/atozindex/organophosphate.htm]

USEPA (United States Environmental Protection Agency) (2002). Interim Reregistration Eligibility Decision for Methidathion. List A, Case 0034. Washington, DC, Office of Prevention, Pesticides and Toxic Substances, USEPA. [http://www.epa.gov/oppsrrd1/REDs/methidathion_ired.pdf]

Wang, S., P. Zhao, G. Min and G. Fang. 2007. Multi-residue determination of pesticides in water using multi-walled carbon nanotubes solid-phase extraction and gas chromatography-mass spectrometry. *J Chromatogr A* 1165(1-2), 166-71.

NOTE: Important general information is contained in PART II, Chapter 6

Methiocarb

(endorsed 2011)

GUIDELINE

Based on human health concerns, methiocarb in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Methiocarb (CAS 2032-65-7) is in the carbamate class of chemicals. Other pesticides in this class include aldicarb, carbaryl, methomyl and pirimicarb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, methiocarb would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Methiocarb is an insecticide, acaricide and molluscicide used for the control of slugs and snails, mites, thrips, aphids, leaf-hoppers, fruit flies and soil insects.

There are registered products containing methiocarb in Australia. The products are for both professional and home garden use and include granular and soluble powder formulations for use in sprays. Use patterns include spreading pellets onto soil by hand or by sod-seeding machines in the home garden, and spraying wettable powder formulations onto soil by boom spray, airblast, or hand sprays in agriculture. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to methiocarb are home garden use and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of sources waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Data on levels of methiocarb in Australian drinking waters are not available. Given the environmental properties of methiocarb, which is highly water insoluble, it is likely that levels in drinking water would be very low and associated with particulate matter and sediments.

TREATMENT OF DRINKING WATER

Advanced treatment methods such as ozonation, ozone/biologically activated carbon and advanced oxidation are effective against this type of compound.

MEASUREMENT

Measurement at residue levels in water are by high performance liquid chromatography (HPLC) with post-column derivitisation and HPLC with tandem mass spectrometry.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for methiocarb is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a 2-year dietary study in dogs. The NOEL is based on reduced plasma cholinesterase activity. The ADI incorporates a safety factor of 100 and was established in 1986.

The ADI for methiocarb was first set in 1981 at 0.06 mg/kg bw/day, based on a NOEL of 6.25 mg/kg bw/day from a long-term dietary study in dogs, before being revised in 1983 to 0.001 based on a reassessment of data on decreased plasma acetylcholinesterase activity. In 1986, the ADI was revised to its current value on the basis that estimates of food consumption were replaced with actual data.

The acute reference dose (ARfD) of 0.03 mg/kg bw/day for methiocarb was established in 2001, based on NOELs of 3 mg/kg bw/day from developmental toxicity studies in rats and rabbits. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Methiocarb is rapidly absorbed from the gastrointestinal tract, and distributed broadly. It is extensively metabolised and eliminated within 72 hours, mainly in the urine. Methiocarb has a low potential for bioaccumulation. The primary metabolites are methiocarb phenol, methiocarb sulfoxide phenol and methiocarb phenol sulfone.

Acute effects: Methiocarb has a high acute oral and low acute dermal toxicity. Symptoms of acute poisoning include salivation, lacrimation, vomiting, diarrhoea, muscular tremors, restlessness, convulsions, and paralysis. Methiocarb is not a skin sensitiser.

Short-term effects: In short-term dietary studies in rats, there are clinical symptoms indicative of central nervous system toxicity. Plasma, red blood cell and brain acetylcholinesterase activity was decreased at a dose of 10 mg/kg bw/day.

Long-term effects: Long-term dietary studies in rat, mouse and dog also showed effects on the central nervous system with decreased acetylcholinesterase activity in brain and other tissues, together with decreases in physical activity and grooming behaviours at dose levels above 0.2 mg/kg bw/day in dogs. This NOEL is the basis for the ADI.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for methiocarb.

Genotoxicity: Methiocarb is not considered to be genotoxic, based on short-term *in vitro* and *in vivo* studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits showed no evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Methiocarb is included in Schedule 5, 6 or 7 in the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for methiocarb was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a long-term (2-year) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Methomyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, methomyl in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Methomyl (CAS 16752-77-5) belongs to the carbamate class of insecticides. There are many other pesticides in this class, including carbaryl, methiocarb, and oxamyl (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, methomyl would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level even for a relatively short period are of concern as the health-based guideline is based on short--term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Methomyl is an insecticide for the control of fruit flies in agricultural settings, and domestic flies in home garden, public and industrial settings.

There are registered products that contain methomyl in Australia. The products are intended for professional and home garden use. They are applied by aerial and ground boom spray on agricultural crops and as baits in domestic and commercial buildings. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to methomyl are the use of home garden products and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of methomyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for methomyl in Australian waters could be found. In the USA, the maximum predicted concentrations for methomyl are 20 and 30 µg/L in groundwater and surface water, respectively (USEPA 1998).

TREATMENT OF DRINKING WATER

Oxidation has been reported to be an effective method of removing methomyl. Both ozone and chlorination (using chlorine gas) were successful treatment methods for methomyl removal; however, harmful by-products are formed during oxidation processes (Mason *et al.* 1990).

Activated carbon technologies are limited in their success in the removal of methomyl, and very large doses of powdered activated carbon are required to achieve any significant removal (Hu *et al.* 1998).

MEASUREMENT

The practical limit of quantification for methomyl in water is 1 µg/L by liquid chromatography with tandem mass spectrometry (Alder *et al.* 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for methomyl is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.25 mg/kg bw/day from a medium-term (3-month) dietary study in rats. The NOEL is based on decreased cholinesterase activity. The ADI incorporates a safety factor of 100, and was first established in 1991.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Methomyl is readily absorbed via the gastrointestinal tract in rats, and is extensively metabolised. The major metabolic pathway is via isomerisation to the E-isomer, then hydrolysis followed by rearrangement to form acetonitrile. The metabolites are eliminated in the urine almost completely within 24 hours as sulfate conjugates.

Acute effects: Methomyl has high acute oral toxicity and moderate acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 28-day dietary studies in mice and rats, there was decreased cholinesterase activity at 10 mg/kg bw/day and above. No other effects were observed. Medium-term (3-month) dietary studies reported decreased cholinesterase activity at 12 mg/kg bw/day in mice and decreased bodyweight gain, decreased kidney weight and adverse effects in the spleen in rats. A medium-term study in dogs was not considered acceptable for regulatory purposes. The lowest overall NOEL was 1.25 mg/kg bw/day in the medium-term study in rats. This NOEL is the basis for the current ADI.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, decreased cholinesterase activity was reported at 13 mg/kg bw/day. In rats, decreased bodyweight gain and adverse effects on the spleen were reported at 10 mg/kg bw/day. In dogs, splenic haematopoiesis, and kidney swelling and pigmentation were reported at 10 mg/kg bw/day.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for methomyl.

Genotoxicity: Methomyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Reproduction studies in rodents reported decreased litter size and increased still births in first generation pups at the lowest dose, 3.75 mg/kg bw/day. Developmental studies in rodents reported increased embryo deaths at 1 mg/kg bw/day. The potential effects of methomyl on reproduction and development are the subject of a current review.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Methomyl is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for methomyl was determined as follows:

$$0.02 \text{ mg/L} = \frac{1.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 1.25 mg/kg bw/day is the NOEL based on a medium-term (3-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation., with an additional safety factor of 2 to account for uncertainty in the ADI (which may change as a result of the current review).

The World Health Organization has not established a health-based guideline value for methomyl and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Alder L, Greulich K, Kempe G, Vieth B (2006). Residue analysis of 500 high priority pesticides: better by GC-MS or LC-MS/MS? *Mass Spectrometry Reviews*, 25(6):838-65.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hu J-Y, Aizawa T, Asami M, Magara Y (1998). Analysis of thermolabile and polar pesticides in environmental water with capillary chromatography-frit-fast atom bombardment mass spectrometry. *Water Science and Technology*, 38(7):169-177.

Mason Y, Choshen E, Rav-Acha C (1990). Carbamate insecticides: Removal from water by chlorination and ozonation. *Water Research*, 24(1):11-21.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

USEPA (1998) *Reregistration Eligibility decision (RED) Methomyl* Prevention, Pescticides and Toxic Substances (7508C), United States Environment Protection Agency, EPA 738-R-98-021, December 1998

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Methyl bromide

(endorsed 2011)

GUIDELINE

Based on human health concerns, methyl bromide in drinking water should not exceed 0.001 mg/L.

RELATED CHEMICALS

Methyl bromide (CAS 74-83-9) belongs to the fumigant class of chemicals. Other pesticides in this class include chloropicrin, dimethyl disulfide, ethylene dibromide and sulfuryl fluoride (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, methyl bromide would not be a health concern unless the concentration exceeded 0.001 mg/L. Excursions above this level would need to occur over a reasonably significant period to be a health concern, as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Methyl bromide is a broad-spectrum fumigant effective against a variety of pests including insects, nematodes, rodents, bacteria, viruses, fungi, mites and weeds. It is used in grain silos, buildings, ships' holds and cargos, and in soil.

There are registered products that contain methyl bromide in Australia. The products are intended for professional use only, with restrictions on availability to authorised or licensed persons. Methyl bromide is available as liquefied gas (under pressure) to be applied either by hand equipment or machinery. Methyl bromide is currently in the phase-out stage due to environmental concerns, with very restricted uses allowed only under Critical Use Exemptions (CUEs) and for quarantine uses. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to methyl bromide is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Use of methyl bromide on soil may potentially lead to contamination of source waters through absorption into soil and subsequent entry into groundwater.

A major route of exposure to methyl-bromide is likely to be inhalation from gaseous sources such as leaking and venting from fumigation chambers, and gas escape from treated products.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of methyl bromide in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No reports of the treatment of methyl bromide in drinking water were found. Methyl bromide is a volatile halogenated methane and would be expected to respond to most treatment processes in a similar manner to other halogenated methanes such as dichloromethane and chloroform.

MEASUREMENT

Methyl bromide can be measured in drinking waters using the same techniques as those routinely applied to dihalomethanes and trihalomethanes. Most commonly these substances are analysed by liquid-liquid extraction followed by gas chromatography with electron caption detection. The typical limit of quantitation for this approach is less than 1 µg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for methyl bromide is 0.0004 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.4 mg/kg bw/day from a short-term (90-day) dietary rat study. The NOEL is based on injury to the forestomach. The ADI incorporates a safety factor of 1000 and was established in 2001.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Methyl bromide is readily and extensively absorbed from the gastrointestinal tract in rats. It is widely distributed to tissues, extensively metabolised to bromide, excreted in urine and bile, and exhaled as CO_2. Bromide accumulates in tissues in rats followed by slow elimination. The half-life of bromide has been reported to be 12 days in humans.

Acute effects: Methyl bromide has moderate acute oral toxicity and high dermal toxicity in rats. There are no quantitative data available on skin sensitisation. However, dermal exposure may result in redness, dermatitis, itching, swelling and blistering.

Short-term effects: Short-term dietary studies in rats reported focal hyperaemia and dose-related hyperplasia of the forestomach at doses of 2 mg/kg bw/day and above. The NOEL for these effects was 0.4 mg/kg bw/day and this is the basis for the current ADI. Inflammation, fibrosis and hyperkeratosis were reported at doses of 25 mg/kg bw/day and above. Methyl bromide is much more toxic via inhalation than via the oral route.

Long-term effects: A long-term dietary study in rats reported decreased bodyweight gain at a dose of 16.75 mg/kg bw/day. Relative brain and kidney weights were increased in males at higher doses.

Carcinogenicity: From oral administration studies in rats, there is some evidence of induction of forestomach squamous cell carcinoma. From inhalational studies in mice and rats, there is no evidence of carcinogenicity for methyl bromide.

Genotoxicity: Methyl bromide produced positive results in some *in vitro* assays in bacteria and cultured mammalian cells, as well as in mouse and rat bone marrow cells *in vivo* and in *Drosophila*. It was negative in the mouse dominant lethal assay. Overall, it has some genotoxic potential; however, there is no evidence that this leads to cancer formation.

Reproductive and developmental effects: Two-generation reproduction studies in rats and rabbits did not produce any evidence of reproductive effects. A developmental toxicity study in rabbits by inhalation exposure produced developmental effects at maternotoxic dose levels only. No oral exposure study was available.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Methyl bromide is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), with an Appendix J (Conditions for availability and use of Schedule 7 poisons) rider restricting its availability to authorised or licensed persons. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.001 mg/L for methyl bromide was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.4 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 0.4 mg/kg bw/day is the NOEL based on a short-term (90-day) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation, and an additional 10 because the NOEL is based on a short-term study.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Metiram

(endorsed 2011)

GUIDELINE

Metiram degrades in the environment to ethylene thiourea (ETU), hence the health-based guideline for metiram is based on the toxicity of ETU. Based on human health concerns, the environmental degradant of metiram, ETU, in drinking water should not exceed 0.009 mg/L.

RELATED CHEMICALS

Metiram (CAS 9006-42-2) belongs to the ethylenebis-dithiocarbamate class of chemicals. Other pesticides in this class include mancozeb and zineb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, the environmental degradant of metiram, ethylene thiourea (ETU), would not be a health concern unless the concentration exceeded 0.009 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Metiram is a fungicide for the control of early and late blight in potatoes, and fungal diseases in apples, pears, grapevines, stone fruit, turf and certain vegetable crops.

There are registered products that contain metiram in Australia. The products are intended for professional use and are available as concentrated solutions to be applied in diluted form using ground or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: Metiram hydrolyses rapidly in the environment to ETU and carbon disulfide (CS_2), both of which have higher toxicity than metiram. It is considered highly unlikely that residues of metiram or its degradants will be present in food. Metiram residues are grouped with other dithiocarbamates (mancozeb, metham, metiram, propineb, thiram, zineb and ziram) in the maximum residue limit definition.

Agricultural use of metiram may potentially lead to contamination of source waters by both metiram and ETU through adsorption into soil and subsequent entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Metiram and ETU have been monitored in some drinking water supplies in Australia, with values below the limit of quantitation (Barwon Water 2007). The highest measured value in a public drinking water well in the USA was reported to be 0.21 µg/L (USEPA 2005).

TREATMENT OF DRINKING WATER

Powdered activated carbon filtration, granulated activated carbon filtration, and reverse osmosis have been demonstrated to be highly effective processes at removing certain pesticides including dithiocarbamates (USEPA 2001). Dithiocarbamates are also degraded by hydrogen peroxide, ultraviolet irradiation and Fenton-type advanced oxidation processes (Ikehata and El-Din 2006).

ETU is degraded by ozone at 3 mg/L and chlorine dioxide at 20 mg/L, producing several degradation compounds (Hwang *et al*. 2003).

MEASUREMENT

The analytical methods for metiram rely on acid hydrolysis to release CS_2, which is then measured colorimetrically or by gas chromatography. Metiram degrades in the environment to ETU, hence the analytical methods reported are for the determination of ETU in water. United States Environmental Protection Agency (USEPA) Method 509 for the determination of ETU in water using gas chromatography with nitrogen-phosphorus detector can achieve a limit of quantitation (LOQ) of 2.7 µg/L (Munch and Graves 1992). After extraction with dichloromethane in the presence of thiourea and sodium L-ascorbate, ETU can be analysed by gas chromatography with alkali flame ionization detection and mass spectrometric confirmation. The LOQ is less than 0.1 µg/L in water (Van Der Poll *et al*. 1993). ETU in water can also be analysed by fluorimetric determination based on the inhibitory effect of ETU on the oxidation of thiamine to thiochrome by mercury(II) (Pérez-Ruiz *et al*. 1998). ETU has been determined in water by a cathodic stripping voltammetry method, with a LOQ of 1.4 µg/L (Carvalho *et al*. 2004).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for metiram is 0.02 mg per kg body weight (mg/kg bw), based on a lowest-observed-effect level (LOEL) at the lowest dose tested of 5 mg/kg bw/day from a short-term (26 week) gavage study in monkeys and 250-fold safety factor. The LOEL is based on decreased thyroxine levels at the lowest dose tested of 5 mg/kg bw/day and above. Decreased serum triiodothyronine levels, and partially reversible thyroid enlargement and hyperplasia were seen at doses of 15 and 75 mg/kg bw/day, following a 15-week recovery period. A no-observed-effect level (NOEL) was not demonstrated in this study. The ADI incorporates a safety factor of 200, and was first established in 1988. There is currently no ADI for ETU.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Metiram is readily absorbed via the gastrointestinal tract and is widely distributed in tissues and blood. It is moderately metabolised to ETU, and hydrolysed to carbon disulfide (CS_2). It is slowly excreted as ETU in the urine and faeces, and as CS_2 in the breath within 7 days.

Acute effects: Metiram has low acute oral and dermal toxicity. It is a skin sensitiser in guinea pigs. ETU has low acute oral toxicity.

NOTE: Important general information is contained in PART II, Chapter 6

Short-term effects: In a 3-month dietary study with metiram in rats, atrophy of skeletal muscle fibres in females was seen at 15 mg/kg bw/day, and hind-limb paralysis in females was seen at the highest dose tested, 45 mg/kg bw/day. Reversible decreases in iodine uptake into the thyroid were seen at all doses tested, and reversible decreases in thyroxine levels were seen at 45 mg/kg bw/day.

In a 26-week oral gavage study with metiram in monkeys, levels of thyroxine and triiodothyronine in the thyroid gland were decreased at the lowest dose tested of 5 mg/kg bw/day and above. Decreases in serum thyroxine and partially reversible increases in thyroid weight and thyroid hyperplasia were seen at the next highest doses of 15 mg/kg bw/day and 75 mg/kg bw/day (highest dose tested). A NOEL was not obtained in this study. The LOEL of 5 mg/kg bw/day in this study, with 250-fold safety factor, is the basis for the current ADI for metiram.

In short-term studies with ETU, the thyroid was the target organ. In a dietary study in rats over 14 days, histological changes including thyroid hyperplasia, bone marrow depletion, and lymphatic lesions occurred from 25 mg/kg bw/day. When administered in the drinking water of rats over 28 days, decreased levels of thyroxine and triiodothyronine, increased levels of thyroid stimulating hormone in serum, and thyroid follicular necrosis were seen at doses from 10.6 mg/kg bw/day. Other effects seen at 17.6 mg/kg bw/day and above include proximal tubule kidney cell hypertrophy and vacuolisation.

In 3- and 4-month dietary studies in rats and mice with ETU, effects in rats included follicular cell hypertrophy and thyroid hyperplasia from 3 mg/kg bw/day and above. At higher doses there was also increased relative thyroid weight and decreased iodine uptake into the thyroid (8 mg/kg bw/day), decreased levels of thyroxine and increased levels of thyroid stimulating hormone in serum, and increased absolute thyroid weights (10 mg/kg bw/day), thyroid adenomas (12.5 mg/kg bw/day). In mice, thyroid adenomas and pituicyte vacuolisation occurred from 12.5 mg/kg bw/day, hepatocellular hypertrophy from 37.5 mg/kg bw/day, and thyroid hyperplasia from 75 mg/kg bw/day. The lowest overall NOEL was 2 mg/kg bw/day (rats) in these studies.

Long-term effects: In 96-week studies with metiram in mice and 2-year studies in rats by dietary administration, there was decreased food consumption and bodyweight gain at the highest dose tested, 150 mg/kg bw/day, in mice, and an increased incidence of skeletal muscle atrophy at the highest dose tested, 16 mg/kg bw/day, in rats. No other effects were seen in these studies.

In a 1-year rat study with ETU, there was increased thyroid vascularisation and thyroid acinar cell papillation at the lowest dose tested, 0.025 mg/kg bw/day. At 1.25 mg/kg bw/day, there was decreased bodyweight gain. Increased relative thyroid weight occurred at 7 mg/kg bw/day and thyroid tumours occurred at 15 mg/kg bw/day.

In a 2-year rat study with ETU, thyroid hyperplasia, elevated TSH and decreased triiodothyronine and thyroxine were seen at the lowest dose, 0.25 mg/kg bw/day. Thyroid carcinomas were observed at 8.7 mg/kg bw/day. In a 2-year mouse study with ETU, decreased bodyweight gain, increased thyroid stimulating hormone (TSH), thyroid cell hypertrophy and hyperplasia and thyroid adenomas and carcinomas were observed at the lowest dose tested, 16 mg/kg bw/day.

Carcinogenicity: Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for metiram. In mice, ETU produced thyroid follicular-cell tumours and tumours of the liver and anterior pituitary gland. However, due to its nongenotoxicity and disturbance of thyroid function, ETU would not be expected to produce thyroid cancer in humans exposed to concentrations that do not alter thyroid hormone homeostasis.

Genotoxicity: Metiram is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies. ETU was positive in some *in vitro* short-term assays, but overall, it is not considered to be genotoxic.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development. Developmental studies on rats and rabbits with ETU showed effects on development only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Metiram is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.009 mg/L for the degradant of metiram, ETU, was determined as follows:

$$0.009 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 1}{2 \text{ L/day} \times 1000}$$

where:

- 0.25 mg/kg bw/day is the LOEL based on a long-term (2-year) dietary study in rats on ETU.
- 70 kg is taken as the average weight of an adult.
- The proportionality factor is 1 since metiram has no residues in food and is degraded to ETU in the environment. It is assumed, therefore, that 100% of the ADI (nominal in this case) for ETU will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the LOEL for ETU derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation, and an additional factor of 10 because a LOEL was used to derive the guideline.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Barwon Water (2007) Annual Drinking Water Quality Report, Victoria.

Carvalho LMd., Nascimento PCd, Bohrer D, Del-Fabro L (2004). Determination of ethylenethiourea (ETU) at trace levels in water samples by cathodic stripping voltammetry. *Electroanalysis,* 16(18):1508-1513.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hwang ES, Cash JN, Zabik MJ (2003). Determination of degradation products and pathways of mancozeb and ethylenethiourea (ETU) in solutions due to ozone and chlorine dioxide treatments. *Journal of Agricultural and Food Chemistry,* 51(5):1341-6.

Ikehata K, El-Din MG (2006). Aqueous pesticide degradation by hydrogen peroxide/ultraviolet irradiation and Fenton-type advanced oxidation processes: a review. *Journal of Environmental Engineering and Science*, 5(2):81-135.

Munch DJ, Graves RL (1992). Method 509 Determination of ethylene thiourea (ETU) in water using gas chromatography with nitrogen-phosphorus detector, Revision 1.0, U.S. Environmantal Protection Agency, Cincinnati, Ohio.

Pérez-Ruiz T, Martínez-Lozano C, Tomás V, Sanz A, Martín J (1998) Flow-injection spectrofluorimetric determination of ethylenethiourea. *Fresenius' Journal of Analytical Chemistry,* 362(4):1432-1130.

NOTE: Important general information is contained in PART II, Chapter 6

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2001) The Incorporation of Water Treatment Effects on Pesticide Removal and Transformations in Food Quality Protection Act (FQPA) Drinking Water Assessments, Office of Pesticide Programs Science Policy, USEPA, Washington, D.C.

USEPA (United States Environmental Protection Agency) (2005) Reregistration Elegibility Desicion for Metiram, EPA 73R-05-017, USEPA.

Van Der Poll JM, Versluis-De Haan GG, De Wilde O (1993). Determination of ethylenethiourea in water samples by gas chromatography with alkali flame ionization detection and mass spectrometric confirmation *Journal of Chromatography A,* 643(1-2): 163-168.

NOTE: Important general information is contained in PART II, Chapter 6

Metolachlor/s-Metolachlor

(endorsed 2011)

GUIDELINE

Based on human health concerns, metolachlor in drinking water should not exceed 0.3 mg/L.

RELATED CHEMICALS

Metolachlor (CAS 51218-45-2) belongs to the chloroacetamide class of chemicals. Other pesticides in this class include dimethenamid and propachlor. Metolachlor is a racemic mixture of the R- and S-isomers; S-metolachlor is the purified S-isomer (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, metolachlor would not be a health concern unless the concentration exceeded 0.3 mg/L. Minor excursions above this level would need to occur over a relatively long period to be a health concern, as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Metolachlor is a pre-emergent herbicide for the control of grasses and broad-leaf weeds in agricultural crops.

There are registered products that contain metolachlor or its isomer s-metolachlor in Australia. These products are intended for professional use and are available as concentrated solutions to be applied in diluted form using boom and hand-held ground sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to metolachlor and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of metolachlor may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of metolachlor in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Metolachlor can be a relatively difficult pesticide to treat in drinking water. Oxidation by chlorine or ozone are only partially effective at typical doses, and adsorption to activated carbon can be incomplete (Ormad *et al.* 2008). However, a combination of ozone, activated carbon and coagulation-flocculation can be effective (Ormad *et al.* 2008).

MEASUREMENT

Metolachlor can be measured in drinking waters by gas chromatography with nitrogen–phosphorus detection. The limit of detetion for this method is 0.75–0.01 µg/L (WHO 2004).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for metolachlor is 0.08 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 7.5 mg/kg bw/day from a medium-term (6-month) dietary study in dogs. The NOEL is based on decreased bodyweight gain. The ADI incorporates a safety factor of 100, and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metolachlor and its isomer S-metolachlor demonstrate similar toxicological profiles. Therefore, the toxicological endpoints that occur at the lowest dose level for either chemical (both referred to as metolachlor) have been reported below.

Metabolism: Metolachlor is readily and extensively absorbed in the gastrointestinal tract of rats. It is extensively metabolised and excreted in urine and faeces. Metabolites have not been identified.

Acute effects: Metolachlor has low acute oral and dermal toxicity. It is a skin sensitiser in guinea-pigs.

Short-term effects: Medium-term dietary studies conducted in rats and dogs reported biochemical changes and changes in bodyweight and organ weight as the most sensitive toxicological effects. In a 13-week study in rats, there were effects indicative of mild liver toxicity, reduced bodyweight gain and increases in relative and absolute organ weights at 200 mg/kg bw/day. A 6-month study in dogs reported decreased bodyweight gain, food consumption and absolute liver weights at 25 mg/kg bw/day. The NOEL of 7.5 mg/kg bw/day from this study is the basis for the current ADI.

Long-term effects: Long-term dietary studies have been conducted in mice, rats and dogs. In a 2-year dietary study in mice there were changes in spleen weight at 150 mg/kg bw/day. In a 2-year rat study there was decreased bodyweight gain and food consumption, and an increase in pituitary carcinomas and liver neoplastic nodules at 150 mg/kg bw/day. In a 1-year dietary study in dogs, there was decreased bodyweight gain at 32 mg/kg bw/day.

Carcinogenicity: There was some evidence of carcinogenicity in rats at high dose levels, but the threshold noted for this effect is well in excess of the likely level of human exposure.

Genotoxicity: Metolachlor is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Metolachlor is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.3 mg/L for metolachlor was determined as follows:

$$0.3 \text{ mg/L} = \frac{7.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 7.5 mg/kg bw/day is the NOEL based on a medium-term (6-month) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a health-based guideline value of 0.01 mg/L for metolachlor (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71: 97-106.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Metribuzin

(endorsed 2011)

GUIDELINE

Based on human health concerns, metribuzin in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Metribuzin (CAS 21087-64-9) belongs to the triazinone class of chemicals. Other pesticides in this class include hexazinone, metamitron and toltrazuril (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, metribuzin would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

GENERAL DESCRIPTION

Uses: Metribuzin is a pre- and post-emergent herbicide for the control of broad-leaf weeds and grasses in agricultural crops.

There are registered products that contain metribuzin in Australia. The products are intended for professional use and are available as concentrated solutions, granular formulations or wettable powders to be applied in diluted form using ground boom and aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to metribuzin and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of metribuzin may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of metribuzin in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of metribuzin in drinking water have been identified.

MEASUREMENT

Metribuzin may be measured in drinking waters by gas chromatography with mass spectrometry, with a limit of reporting of 0.1 µg/L (QFSS 2009).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for metribuzin is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2 mg/kg bw/day from a long-term rat study. The NOEL is based on decreased absolute and relative heart weights. The ADI incorporates a safety factor of 100, and was established in 1982.

The acute reference dose (ARfD) of 0.25 mg/kg bw for metribuzin was established in 2007, based on a NOEL of 25 mg/kg bw/day from a developmental study in rats. The ARfD incorporates a safety factor of 100.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Metribuzin is rapidly absorbed via the gastrointestinal tract in rats. It is readily metabolised and rapidly excreted in the urine and faeces with little tissue retention after 96 hours. The major metabolite recovered from rats is N-acetylcysteine.

Acute effects: Metribuzin has low to moderate acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: A short-term dermal study conducted in rabbits reported elevated serum levels of thyroid hormone (thyroxine) in female rabbits at doses of 200 mg/kg bw/day and above. Short-term dietary studies performed in rats reported increased absolute liver weights at the high dose of 4.5 mg/kg bw/day. Associated histopathological effects were not observed.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs showed the main effect to be changes in absolute and relative organ weights. In a 2-year study in mice, increased liver, spleen, and kidney weights and evidence of anaemia were observed at 500 mg/kg bw/day. In a 2-year study in rats, decreased relative and absolute heart weights were observed at 5.7 mg/kg bw/day, and effects on the thyroid hyperplasia and fluctuating levels of triiodothyronine and thyroxine at 13 mg/kg bw/day and above. In a 2-year study in dogs, anaemia, increased mortality, increased relative and absolute organ weights and lesions in heart, liver, kidney and adrenal glands were observed at 52.5 mg/kg bw/day. The overall NOEL was 2 mg/kg bw/day in rats, and this is the basis for the current ADI.

Carcinogenicity: Based on a 2-year study in rats, metribuzin is not considered to be carcinogenic.

Genotoxicity: Metribuzin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and a developmental study in rats did not produce any evidence of effects on reproduction or foetal development. In the developmental study, effects on the dams included increased thyroid weights and fluctuations in thyroid hormones at doses of 75 mg/kg bw/day and above. The NOEL of 25 mg/kg bw/day from this study was used to set the ARfD.

Poisons Schedule: Metribuzin is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for metribuzin was determined as follows:

$$0.07 \text{ mg/L} = \frac{2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

QFSS (Queensland Forensic and Scientific Services) (2009) QFSS, Personal communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Metsulfuron-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, metsulfuron-methyl in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Metsulfuron-methyl (CAS 74223-64-6) belongs to the sulfonylurea class of chemicals. There are many other pesticides in this class including azimsulfuron, chlorsulfuron, ethoxysulfuron, halosulfuron-methyl, iodosulfuron methyl-sodium salt, sulfometuron-methyl, sulfosulfuron, triasulfuron, tribenuron-methyl and trifloxysulfuron (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, metsulfuron-methyl would not be a health concern unless the concentration exceeded 0.04 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Metsulfuron-methyl is used as a post-emergent herbicide for the control of weeds in native pastures, rights of way, commercial and industrial areas, domestic and public service areas and agricultural crops.

There are registered products that contain metsulfuron-methyl in Australia. The products are intended for professional use and are available in wettable powder and granular formulations. Product labels indicate products are to be diluted and applied by boom, hand-held and aerial spray methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to metsulfuron-methyl and its metabolites are residues in food and contact with treated weeds. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of metsulfuron-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on metsulfuron-methyl occurrence in Australian drinking water supplies were found.

TREATMENT OF DRINKING WATER

No specific data on the treatment of metsulfuron-methyl in drinking water have been identified.

MEASUREMENT

Metsulfuron-methyl can be measured in water after filtration by direct injection on a triple quadrapole liquid chromatography–mass spectrometry instrument in multiple reaction monitoring mode, with a limit of reporting of 10 µg/L. Metsulfuron can also be analysed by solid phase extraction (SPE) followed by high performance liquid chromatography with diode array detection, achieving a limit of detection of 10 µg/L (Ruberu *et al.* 2000). SPE followed by liquid chromatography with electrospray mass spectrometric detection can achieve method detection limits of 3 ng/L for drinking water samples (Corcia *et al.* 1999).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for metsulfuron is 0.01 mg per kg of bodyweight (0.01 mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a long-term (2-year) dietary rat study. The NOEL is based on decreased bodyweight gain. The ADI incorporates a safety factor of 100 and was established in 1985.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Metsulfuron-methyl is readily absorbed via the gastrointestinal tract in rats. It is not extensively metabolised and is rapidly excreted mostly unchanged in the urine (90%) and faeces. Less than 2% was retained in the tissue in rats.

Acute effects: Metsulfuron-methyl has low acute oral and dermal toxicity. Metsulfuron-methyl is not a skin sensitiser in guinea pigs.

Short-term effects: Medium-term dietary studies in rats and dogs reported decreased bodyweight gain and clinical evidence of mild toxicity in both species at doses of 12 mg/kg bw/day and above. Medium-term studies in mice did not report any toxic effects at doses up to 750 mg/kg bw/day.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs reported decreased bodyweight gain and decreased food consumption in all species at doses of 12 mg/kg bw/day and above. The most sensitive NOEL was 0.93 mg/kg bw/day based on decreased bodyweight gain in a 2-year dietary study in rats. This NOEL was used to set the current ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for metsulfuron-methyl.

Genotoxicity: Metsulfuron-methyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A two-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any effects on reproductive parameters or on foetal development.

Poisons Schedule: Metsulfuron-methyl is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for metsulfuron-methyl was determined as follows:

$$0.04 \text{ mg/L} = \frac{1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Corcia AD, Costantino A, Crescenzi C, Samperi R (1999). Quantification of phenylurea herbicides and their free and humic acid-associated metabolites in natural waters. *Journal of Chromatography A,* 852(2):465-474.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ruberu SR, WM Draper, Perera SK (2000). Multiresidue HPLC methods for phenyl urea herbicides in water. *Journal of Agricultural and Food Chemistry,* 48(9):4109-4115.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Mevinphos

(endorsed 2011)

GUIDELINE

Based on human health concerns, mevinphos in drinking water should not exceed 0.005 mg/L.

RELATED CHEMICALS

Mevinphos (CAS 7786-34-7) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes chlorfenvinphos, chlorpyrifos, dichlorvos and diazinon (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, mevinphos would not be a health concern unless the concentration exceeded 0.005 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Mevinphos is an anticholinesterase acaricide (miticide) and insecticide for the control of moths in *Brassica* crops (cabbages, cauliflowers, broccoli and brussel sprouts).

There is at least one registered product that contains mevinphos in Australia. Mevinphos products are intended for professional use and are available as concentrated solutions to be applied in diluted form, by boom spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to mevinphos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of mevinphos may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on mevinphos occurrence in Australian drinking water supplies were found.

TREATMENT OF DRINKING WATER

A reverse osmosis (RO) membrane challenge test was conducted by injecting 1200 µg/L of mevinphos before treatment. RO removed 96% of the initial concentration of mevinphos (1200 µg/L), and the post-membrane carbon filter further removed 95% of the remaining mevinphos, from 40 µg/L to 2.1 µg/L (USEPA 2005). In another challenge test, the concentration of mevinphos remained the same after ultraviolet (UV) light and ozone treatment; however, the activated carbon filter located after the UV and ozone treatment removed 99% of the pesticide (USEPA 2007).

MEASUREMENT

Mevinphos is one of the organophosphorus pesticides analysed by the United States Environmental Protection Agency (USEPA) Method 622 (Pressley *et al.* 2002). The sample is solvent-extracted and the extract is dried with sodium sulfate concentrated and analyzed by gas chromatography using a flame photometric or phosphorus/nitrogen detector. The concentrated can also be analysed by gas chromatography–mass spectrometry in selected ion monitoring mode. The method can achieve a limit of quantitation (LOQ) of 0.05 mg/L. Hollow fiber liquid phase micro-extraction with gas chromatography by flame thermionic detection can achieve a LOQ of 40 ng/L in drinking water (Lambropoulou and Albanis 2005). A solid-phase microextraction method coupled with a flame photometric can achieved a LOQ for mevinphos of 420 µg/L (Su and Huang 1999).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for mevinphos is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.016 mg/kg bw/day (1 mg/person/day using a mean bodyweight of 62.5 kg) from a 30-day human volunteer study. The NOEL is based on inhibition of erythrocyte cholinesterase. The ADI incorporates a safety factor of 10 and was established in 1998.

The previous ADI established in 1996 was 0.0008 mg/kg bw based on a NOEL of 0.016 mg/kg bw/day (1 mg/person/day) in a human volunteer study and using a safety factor of 20. An additional factor of 2 was included because of the decrease in slow motor fibre nerve conduction velocity observed in a different human volunteer study at 0.025 mg/kg bw/day.

The acute reference dose (ARfD) of 0.003 mg/kg bw for mevinphos was based on a LOEL of 0.025 mg/kg bw/day from a 28-day human volunteer study. The ARfD incorporates a safety factor of 10 and was established in 2000.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Mevinphos is rapidly and extensively absorbed via the gastrointestinal tract and rapidly metabolised and excreted, predominantly as carbon dioxide in expired air (78%), with only 14% in the urine over 24 hours. There were four major metabolites in the urine.

Acute effects: Mevinphos has high acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 3-, 12- and 13-week dietary studies in rats at dose levels up to 20 mg/kg bw/day, the principal sign of toxicity at the higher dose levels was death. Four dogs also died in a dietary study at a dose of 15 mg/kg bw/day. In a 30-day five-person human volunteer study, erythrocyte cholinesterase activity was decreased at 1.5 mg/person/day. The NOEL was 0.015 mg/kg bw/day (1 mg/person/day) and this NOEL is the basis for the ADI. In a 28-day eight person volunteer study, plasma and erythrocyte cholinesterase activity was decreased at 0.025 mg/kg bw/day. There was also a decrease in slow fibre

NOTE: Important general information is contained in PART II, Chapter 6

motor conduction at this dose level. This LOEL, the only dose tested, is the basis for the ARfD.

Long-term effects: In a 2-year dietary study in rats at dose levels up to 0.6 mg/kg bw/day, there were no treatment-related effects on general health or bodyweight gain. No histopathological changes were observed. In a 2-year dietary study in dogs at doses up to 0.75 mg/kg bw/day, plasma and erythrocyte cholinesterase activity were decreased at 0.075 mg/kg bw/day and above and increased with time. At the two-year period, brain cholinesterase activity was significantly decreased at 0.25 mg/kg/day in females and at 0.75 mg/kg in males and females. All but two high dose male animals survived in good health.

Carcinogenicity: Based on a 2-year study in rats and dogs, there is no evidence of carcinogenicity for mevinphos.

Genotoxicity: Mevinphos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Mevinphos is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.005 mg/L for mevinphos was determined as follows:

$$0.005 \text{ mg/L} = \frac{0.015 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.015 mg/kg bw/day is the NOEL established in a short-term (30-day) study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from the human study to allow for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lambropoulou DA, Albanis TA (2005). Application of hollow fiber liquid phase microextraction for the determination of insecticides in water. *Journal of Chromatography A,* 1072(1): 55-61

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Pressley T, Longbottom J (2002). *Method 622 The Determination of Organophosphorus Pesticides in Municipal and Industrial Wastewater.* http://www.accustandard.com/asi/pdfs/epa_methods/622.pdf

NOTE: Important general information is contained in PART II, Chapter 6

Su PG, Huang SD (1999). Determination of organophosphorus pesticides in water by solid-phase microextraction. *Talanta,* 49(2):393-402.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2005). *Point of use drinking water treatment system application: Removal of chemical contaminants in drinking water: Reverse osmosis*. The Environmental Technology Verification Program, USEPA.

USEPA. (United States Environmental Protection Agency) (2007). *Point of use drinking water treatment system application: Removal of chemical contaminants in drinking water: Advanced simultaneous oxidation process*. The Environmental Technology Verification Program, USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Molinate

(endorsed 2011)

GUIDELINE

Based on human health concerns, molinate in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Molinate (CAS 2212-67-1) belongs to the thiocarbamate class of pesticides. Other herbicides in this class include EPTC, methiobencarb, pebulate, thiobencarb and vernolate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, molinate would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short--term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Molinate is a post-emergence herbicide for the control of grass weeds in rice only.

There are registered products that contain molinate in Australia. The products are intended for professional use. The chemical is available as concentrated solutions to be applied in diluted or undiluted form to rice crops soon after sowing. It may be applied by aerial application, or directly to flooded rice bays by drip applicators mounted on a tractor or a four-wheel drive spray bike. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The possible sources of public exposure to molinate and its metabolites are residues in rice and drinking water. Residue levels in rice produced according to good agricultural practice are generally low and maximum residue limits (MRLs) are at the level of detection.

The agricultural use of molinate involves direct application into water bays of rice crops, which may then enter source waters for drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Molinate was the most commonly applied herbicide to rice crops in southern New South Wales in 1994-1995. It was detected in irrigation drains after application to rice fields at levels up to 0.7 mg/L (Bowmer *et al.* 1998). Molinate was detected on two occasions in the Mulwala supply offtake on the Murray river, at 0.0072 and 0.0005 mg/L (7.2 and 0.5 µg/L). Over a 55-day period of monitoring supply water in the irrigated areas, molinate was found in 90% of the analysed samples, with a maximum concentration of 0.0036 mg/L (3.6 µg/L) (Bowmer *et al.* 1998). The high frequency of molinate detection was due to samples being taken in early summer, when the herbicide is used in rice crops.

Rice-growing areas are within the Murray Darling Basin on the Murrumbidgee and Murray rivers in south-western New South Wales and Victoria. Molinate has been detected in the Coleambally irrigation area, Murrumbidgee region, New South Wales and Murray irrigation area (Ball 2001).

TREATMENT OF DRINKING WATER

Ozonation, granular activated carbon, preoxidation by chlorine and preoxidation by chlorine combined with activated carbon adsorption removes 100% of molinate during drinking water treatment (Ormad *et al.* 2008).

MEASUREMENT

Capillary gas chromatography with a selective nitrogen–phosphorus detector for the determination of molinate can achieve a limit of quantitation (LOQ) of 0.03 µg/L (Worthing and Hance 1991). United States Environmental Protection Agency (USEPA) method 525.2 for the determination of organic compounds in drinking water by liquid-solid extraction and capillary column gas chromatography–mass spectrometry (GC/MS) can achieve a LOQ of 0.05 µg/L to 0.087 µg/L for molinate (Munch 1995a). USEPA method 507 can achieve a LOQ of 0.15 µg/L (Munch 1995b). Molinate can be extracted from water by liquid/liquid extraction with dichloromethane and analysed by gas chromatograpy–mass spectometry in selected ion monitoring mode, with a LOQ of 0.5 µg/L. Liquid chromatography–mass spectrometry with direct injection can achieve a LOQ of 2.2 µg/L (Yu *et al.* 2003). Solid phase extraction and high performance liquid chromatography with ultraviolet detection can achieve a LOQ of 0.1 µg/L. Solid phase micro extraction followed by gas liquid chromatography employing either a nitrogen-phosphorus detector or mass spectrometry can achieve LOQ of 0.11 µg/L and 0.02 µg/L respectively (Choudhury *et al.* 1996).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for molinate is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a 3-generation rat reproduction study. The NOEL is based on reduced litter numbers, litter size and pup survival at the next highest dose of 0.63 mg/kg bw/day. The ADI incorporates a safety factor of 100, and was established in 1986.

The previous ADI of 0.0001 mg/kg bw established in 1984 was based on the same NOEL of 0.2 mg/kg bw/day from the 3-generation rat reproduction study, but included a safety factor of 2000 due to the absence of long-term studies. Following the review of long-term studies in 1986, the NOEL of 0.2 mg. kg bw/day remained the lowest available, but the safety factor was reduced to 100 and the current ADI was established.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Molinate is readily and extensively absorbed via the gastrointestinal tract in rats. It is metabolised to more polar products such as molinate sulfoxide (35%) and hydroxymolinate (26%). The majority of an administered dose is excreted within 48 hours, with 82% in urine, 11% in faeces and less than 1% expired as carbon dioxide.

Acute effects: Molinate is of moderate acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

NOTE: Important general information is contained in PART II, Chapter 6

Short-term effects: A 3-week dietary study in rats reported weakness in the hind limbs, and depressed bodyweight and food consumption at 80 mg/kg bw/day. Interference in blood clotting occurred at higher dose levels. Three-month dietary studies in rats reported pathological changes in the liver, kidney, testis, ovary and adrenal glands at 70 mg/kg bw/day. In 3-month dietary study in dogs, there were increases in thyroid gland weight at 60 mg/kg bw/day.

Long-term effects: Long-term dietary studies in rats reported increased testes weight at 2 mg/kg bw/day and above, and decreased bodyweight gain and increased kidney weight at 6 mg/kg bw/day.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for molinate.

Genotoxicity: Molinate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats noted a reduction in the number of litters, with an associated reduction in litter size and pup survival, in all generations at the highest dose, 0.63 mg/kg bw/day. No effects were noted at the next lowest dose, 0.2 mg/kg bw/day. This NOEL is the basis for the ADI. Further detailed studies have shown that molinate causes infertility in rats through its effects on sperm membranes, interfering with sperm maturation and causing some degeneration of seminiferous tubules.

In a developmental toxicity study in mice, there were no effects on foetal development. In rabbits, there were maternotoxic and foetotoxic effects at dose levels well in excess of the likely level of human exposure. There were no teratogenic effects in the rabbit. Developmental and reproduction studies in rats evaluated by the USEPA (not evaluated in Australia) have reported effects on brain weight at 0.4 mg/kg bw/day and on reproductive parameters at 0.2 mg/kg bw/day (USEPA 2002).

Neurotoxicity: Degeneration and demyelination of the sciatic nerve (combined with muscle atrophy) in a long-term study in rats have been reported at the lowest dose of 0.3 mg/kg bw/day by the USEPA (not yet evaluated in Australia).

Poisons Schedule: Molinate is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), with an Appendix J rider limiting availability to authorised or licensed persons. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.004 mg/L for molinate was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a 3-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation, with an additional factor of 2 to take into account the uncertainty resulting from the new data on neurotoxicity and developmental effects, which have yet to be evaluated in Australia.

The World Health Organization has a health-based guideline value of 0.006 mg/L for molinate (WHO 2004).

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Ball J (2001). *Australia State of the Environment Report 2001 Inland Waters Theme Report*. CSIRO on behalf of the Department of the Environment and Heritage.

Bowmer KH, Korth W, Scott A, McCorkelle G, Thomas M (1998). *Pesticide Monitoring in the Irrigation Areas of South-Western NSW 1990-1995*. CSIRO Land and Water, Canberra, Australia.

Choudhury TK, Gerhardt KO, TP Mawhinney (1996). Solid-phase microextraction of nitrogen- and phosphorus-containing pesticides from water and gas chromatographic analysis. *Environmental Science and Technology,* 30(11):3259–3265.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Munch JW (1995a). *Method 525.2 Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and capillarity Columns Gas Chromatography/Mass Spectrometry Revision 2.0*. Environmental Monitoring Systems Laboratory, Office of Research and Development, United States Environmental Protection Agency, Cincinnati, Ohio.

Munch JW (1995b). *Method 507 Determination of Nitrogen and Phosphorus Containing Pesticides in Water by Gas Chromatography with a Nitrogen Phosphorus Detector*. National Exposure Research Laboratory, Office of Research and Development, United States Environmental Protection Agency, Cincinnati, Ohio.NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71(1):97-106.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2002) Molinate: Reregistration Eligibility Decision (RED). USEPA, Washington DC.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

Worthing CR, Hance RJ (eds) (1991). *The Pesticide Manual,* 9th Edition. British Crop Protection Council, Farnham, Surrey.

Yu K, Krol J, Balogh M, Monks I (2003). A fully automated LC/MS method development and quantification protocol targeting 52 carbamates, thiocarbamates, and phenylureas. *Analytical Chemistry,* 75(16):4103-12.

NOTE: Important general information is contained in PART II, Chapter 6

Molybdenum

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of molybdenum in drinking water should not exceed 0.05 mg/L.

GENERAL DESCRIPTION

Molybdenum is present in ground and surface waters at very low concentrations, generally below 0.01 mg/L. Higher concentrations have been reported in the vicinity of molybdenum mining operations. Fly ash deposited onto soils from coal-fired power stations can be a significant source of molybdenum. Application of fertilisers may also increase the concentration of molybdenum in ground and surface water.

Molybdenum is used in the production of steel, electrical components such as spark plugs, and nonferrous metal alloys. Molybdenum compounds are used as lubricants in oils and greases, and in fertilisers to overcome molybdenum deficiency in soils.

Many foods contain significant amounts of molybdenum. Legumes, grains and liver have the highest concentrations and food is a significant source of intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for molybdenum.

TREATMENT OF DRINKING WATER

There are no published methods for the removal molybdenum from drinking water.

MEASUREMENT

The concentration of molybdenum in drinking water can be determined by inductively coupled plasma emission spectroscopy or graphite furnace atomic absorption spectroscopy (APHA Method 3500-Mo Parts B or C 1992). Limits of determination are 0.04 mg/L and 0.005 mg/L respectively.

HEALTH CONSIDERATIONS

Molybdenum is an essential trace element for humans and other animals. The estimated requirement is between 0.15 mg/day and 0.5 mg/day for adults.

Approximately 30–70% of dietary molybdenum is absorbed in the gastrointestinal tract. Highest concentrations of molybdenum are found in the liver, kidney and bones. There does not appear to be any significant bioaccumulation of molybdenum in the body. Approximately 90% of ingested molybdenum is excreted in the urine.

Data are scarce on the long- and short-term toxicity of molybdenum in humans. One study of people consuming up to 0.2 mg/L of molybdenum in drinking water for 2 years reported no adverse effects. Another study has linked high intake of molybdenum in food with gout-like symptoms, joint pains of the legs and hands, and enlargement of the liver.

A number of long- and short-term animal studies have been undertaken, with considerable variability in the results depending on the chemical nature of the compound and the animal species. Effects included changes in skin and fur pigment, enlargement of joints, weight loss, diarrhoea and emaciation. Not all these effects were observed in each study and effects usually occurred only at high doses.

No relevant data are available on the carcinogenicity of molybdenum. Tests for mutagenicity with bacteria have been inconclusive.

DERIVATION OF GUIDELINE

The guideline value for molybdenum in drinking water was determined as follows:

$$0.05 \text{ mg/L} = \frac{0.5 \text{ mg/day} \times 0.2}{2 \text{ L/day}}$$

where:

- 0.5 mg/day is the upper range of the estimated adult requirement for molybdenum.
- 0.2 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

Studies with animals and humans, while unable to establish a no-effect level, reported no adverse effects due to molybdenum in drinking water at concentrations of 0.05 mg/L (Chappell *et al*. 1979).

Adverse human health effects have been reported with molybdenum intakes of 10 mg/day (Chappell *et al* 1979). This is a hundred times higher than the guideline value, assuming that water consumption is 2 litres per day.

The World Health Organization guideline value of 0.07 mg/L was determined using a different approach which, upon review, was considered to be questionable. The difference between the two values is not significant.

REFERENCES

APHA Method 3500-Mo Part B (1992). Molybdenum: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Mo Part C (1992). Molybdenum: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Chappell WR, Meglen RR, Moure-Eraso R, Solomons CC, Tsangas TS, Walravens PA, Wilson PW (1979). Human health effects of molybdenum in drinking water. United States Environmental Protection Agency, EPA-600/1-79-006, Cincinnati, Ohio, 100 pages, NTIS PB-292755.

NOTE: Important general information is contained in PART II, Chapter 6

Monochloramine

CAS NO 10599-90-3

(endorsed 2014)

GUIDELINE

Based on health considerations, the concentration of monochloramine in drinking water should not exceed 3 mg/L (equivalent to 5 mg Cl as Cl_2/L in chloraminated systems).

GENERAL DESCRIPTION

Monochloramine is used as a disinfectant for drinking water supplies. It is increasingly being used in conjunction with chlorine, or in its own right, to provide primary disinfection of drinking water entering the distribution system and/or maintain a disinfectant residual through the distribution network. Although it is not as strong an oxidant as chlorine, monochloramine can be quite useful and effective in distribution systems with long water ages as it persists for longer. Where monochloramine is used overseas, concentrations typically range from 1.5 to 2.5 mg/L (as Cl_2).

Use of monochloramine for primary disinfection at the treatment facility needs to be considered carefully in terms of the range of C.*t* (disinfectant concentration × contact time) values achievable prior to the first customer.

Use of monochloramine can significantly reduce the level of disinfection by-products compared to that produced by similar levels of chlorine. If not managed proactively, however, use of chloramine can lead to nitrification in the distribution system resulting in a reduction of its effectiveness.

Monochloramine is formed by the addition of ammonia and chlorine in drinking water. This reaction can also result in the formation of dichloramine and trichloramine, both of which have lower taste and odour thresholds than monochloramine, and which should be minimised. The preferential formation of monochloramine is affected by the pH and the physical arrangements of adding the two chemicals.

Monochloramine has an odour threshold of 0.5 mg/L.

For additional information refer to the Disinfection Information Sheet for Chloramines.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Monochloramine is used as a disinfectant in some Australian reticulated supplies, and concentrations up to 4-5 mg/L (as total chlorine) have been applied at the start of long distribution systems to achieve concentrations ranging from 0.5-1.5 mg/L at the ends of distribution systems.

TREATMENT OF DRINKING WATER

Monochloramine can be removed from drinking water by the use of granular activated carbon, or by reducing agents such as sodium sulphite or sodium bisulphite.

MEASUREMENT

The concentration of monochloramine in drinking water can be determined by the DPD ferrous titrimetric method (APHA Method 4500-Cl Part F 2012) or by amperometric titration (APHA Method 4500-Cl Part D 2012). The limit of determination is typically 0.1 mg/L for the DPD method and can be lower for amperometric titration. Water utilities should refer to Standard Methods when selecting a method (APHA 2012).

HEALTH CONSIDERATIONS

In studies with rats it has been shown that monochloramine is readily absorbed and does not accumulate in tissues. It is metabolised rapidly to the chloride ion and excreted in urine in mammals. No specific toxic effects have been reported for monochloramine from either short-term or long-term studies. However, monochloramine is toxic to fish.

In humans, short-term exposure to concentrations of up to 24 mg/L of monochloramine in drinking water did not produce adverse effects. Similarly, volunteers given water containing up to 5 mg/L of monochloramine for 12 weeks did not exhibit adverse effects.

Acute haemolytic anaemia has been reported in haemodialysis patients when tap water containing chloramines was used for dialysis (Eaton ***et al***. 1973; Kjellstrand ***et al***. 1974; Tipple ***et al***. 1988). Chloramines present in water are harmful to people on kidney dialysis and to animal species in aquaria; therefore, it is important for water utilities using chloramination to inform consumers at risk. Water suppliers that disinfect with chloramines need to contact coordinators of home dialysis and renal dialysis clinics to advise on the presence and concentrations of chloramines in drinking water.

Carcinogenicity studies have reported a slight increase in the incidence of mononuclear cell leukaemia in female rats exposed to monochloramine for 2 years, at doses of approximately 10 mg/kg bodyweight per day. There was no evidence of carcinogenic activity in male rats, or male and female mice.

Monochloramine exhibited weak mutagenic activity in one test using bacteria but was negative in another test (Shih and Lederberg 1976; Thomas ***et al***. 1987). It did not induce chromosome aberrations in mammalian cells.

Based on inadequate evidence in humans and experimental animals, the International Agency for Research on Cancer concluded that chloramines are not classifiable as to their carcinogenicity to humans (Group 3) (IARC 2004).

No data are available on the health effects of dichloramine or trichloramine in drinking water.

DERIVATION OF GUIDELINE

The guideline value for monochloramine in drinking water was derived as follows:

$$3 \text{ mg/L} = \frac{9.4 \text{ mg/kg bodyweight per day} \times 70 \text{ kg}}{2 \text{ L/day} \times 100}$$

where:

- 9.4 mg/kg bodyweight per day is the no-observed-adverse-effect level (NOAEL) based on 2-year drinking water study using rats (NTP 1992). A similar value was obtained from a human study but this was of a limited duration.
- 70 kg is taken as the average weight of an adult.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOAEL derived from an animal study (10 for interspecies variations, and 10 for intraspecies variations).

Where monochloramine is measured by determining mg Cl as Cl_2 by standard DPD ferrous titrimetric methods the equivalent guideline value is:

$$5 \text{ mg/L} = \frac{9.4 \text{ mg/kg bodyweight per day} \times 70 \text{ kg} \times 71 \text{ (molecular weight } Cl_2)}{2 \text{ L/day} \times 100 \times 51.5 \text{ (molecular weight } NH_2Cl)}$$

It is assumed that all monochloramine intake is from drinking-water.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

APHA Method 4500-Cl Part D (2012). Chlorine (residual): Amperometric titration method. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

APHA Method 4500-Cl Part F (2012). Chlorine (residual): DPD Ferrous Titrimetric method. ***Standard Methods for the Examination of Water and Wastewater***, 22nd edition. APHA (American Public Health Association), AWWA (American Water Works Association) and WEF (Water Environment Federation), Washington, DC.

Eaton JW, Kolpin CF, Swofford H, Kjellstrand CM and Jacob HS (1973). Chlorinated urban water: a cause of dialysis-induced hemolytic anaemia. ***Science***, 181:463–464.

IARC (International Agency for Research on Cancer) (2004). Chloramine. IARC Monograph Vol 84, IARC Publications, Lyon.

Kjellstrand CM, Eaton JW, Yawata Y, Swofford H, Kolpin CF, Buselmeier TJ, von Hartitzsch B and Jacob HS (1974). Hemolysis in dialyzed patients caused by chloramines. ***Nephron***, 13:427–433.

NTP (National Toxicology Program) (1992). Toxicology and carcinogenesis studies of chlorinated water and chloraminated water in F344/N rats and B6C3F1 mice (drinking water studies). NTP, Technical Report No. 392, Publication No. 92-2847. United States Department of Health and Human Services, National Institute of Health.

Shih KL and Lederberg J (1976). Chloramine mutagenesis in Bacillus subtilis. ***Science*** 192: 1141-1143.

Tipple MA, Bland LA, Favero MS and Jarvis WR. (1988) Investigation of hemolytic anaemia after chloramine exposure in a dialysis center. ***American Society for Artificial Internal Organs (ASAIO) Transactions***, 34(4):1060.

Thomas EL, Jefferson MM, Bennett JJ and Learn DB (1987). Mutagenic activity of chloramines. ***Mutation Research*** 188:35-43.

NOTE: Important general information is contained in PART II, Chapter 6

Naphthalophos

(endorsed 2011)

GUIDELINE

The health concerns associated with naphthalophos have not been fully evaluated and therefore a health value for naphthalophos in drinking water cannot be set.

RELATED CHEMICALS

Naphthalophos (CAS 1491-41-4) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes acephate, chlorfenvinphos, diazinon and profenofos (Tomlin 2006).

HUMAN RISK STATEMENT

There are currently insufficient data on which to base a human risk statement.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Naphthalophos is a parasiticide for the control of organophosphate-susceptible gastrointestinal roundworms in sheep and lambs.

There are registered products that contain naphthalophos in Australia. Products containing naphthalophos are intended for professional use and are administered orally to sheep and lambs using drench guns. Products are typically diluted in water in original packaging before being transferred into the backpack reservoir of a drenching gun. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to naphthalophos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The veterinary use of naphthalophos provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on occurrence of naphthalophos in water could be found.

TREATMENT OF DRINKING WATER

No information on efficiency of drinking water treatment to remove naphthalophos could be found.

MEASUREMENT

No method to measure naphthalophos in water could be found.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for naphthalophos is 0.0001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day. The ADI incorporates a safety factor of 2500 and was established in 1971. The toxicological endpoint used to establish this ADI is unknown. The reason for such a large safety factor is also unknown, although it is likely to be due to the limited toxicological database for naphthalophos.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: There are no metabolic or kinetic studies available for naphthalophos.

Acute effects: Naphthalophos has moderate acute oral toxicity in rats and mice and moderate dermal toxicity in rats. No other acute toxicity data are available for naphthalophos.

A quantitative evaluation of the dermal absorption of naphthalophos is not available. However the moderate acute dermal toxicity in rats suggests that it is well absorbed dermally.

Short-term and long-term effects, carcinogenicity, genotoxicity, reproductive and developmental effects: No repeat-dose studies, or developmental or reproductive studies have been evaluated in Australia. Therefore the potential toxicity from exposure to naphthalophos cannot be adequately determined. There are no short-term studies available for naphthalophos. However, as an organophosphate compound, there is potential for cumulative anti-cholinesterase toxicity through repeated minor exposure.

Poisons Schedule: Naphthalophos is included in Schedule 6 or 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

Due to its current status as a chemical under review in Australia, a health-based guideline value for naphthalophos in drinking water is not recommended at this stage.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Napropamide

(endorsed 2011)

GUIDELINE

Based on human health concerns, napropamide in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Napropamide (CAS 15299-99-7) belongs to the alkanamide class of chemicals. Another pesticide in this class is diphenamid (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, napropamide would not be a health concern unless the concentration exceeded 0.4 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Napropamide is a pre-emergent herbicide for the control of grass weeds in tomato, almond, grape, and stone fruit crops and commercial farms.

There is at least one registered product that contains napropamide in Australia. Napropamide products are intended for agricultural use and are available as a concentrated water-soluble granule formulation to be diluted and sprayed onto soil and then incorporated mechanically or by irrigation. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to napropamide and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of napropamide may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on the occurrence of napropamide in Australian waters. Napropamide is broken down very quickly in water, with a half-life as rapid as seven minutes (EXTOXNET 1993). In water, the breakdown is predominantly mediated by the action of sunlight (photolysis). Napropamide was not detected in groundwater in the USA in a national survey in 1988 (Williams *et al*. 1988).

TREATMENT OF DRINKING WATER

No specific data on the treatment of napropamide in drinking water have been identified.

MEASUREMENT

Napropamide can be analysed by high-performance liquid chromatography with ultraviolet detection, with a limit of quantitation of 0.3 µg/L (USEPA Method 632.1).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for napropamide is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 11 mg/kg bw/day from a long-term (2-year) dietary study in rats. The NOEL is based on liver spongiosis and decreased bodyweight gains. The ADI incorporates a safety factor of 100 and was established in 1994.

The previous ADI was 0.4 mg/kg bw based on a NOEL of 40 mg/kg bw/day in a long-term dietary study in rats and a medium-term dietary study in dogs. It was set in 1987.

The previous *Australian Drinking Water Guidelines* health value was 1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Napropamide is readily absorbed from the gastrointestinal tract in rats. Over 80% of the dose is excreted in urine and faeces within 48 hours, and excretion is complete by 96 hours. There is no evidence of accumulation in blood or tissues.

Acute effects: Napropamide has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea-pigs.

Short-term effects: Short-term (13-week) dietary studies were conducted in rats and dogs. In dogs, effects included increased absolute liver weights, and increased levels of glutamic transaminase and alkaline phosphatase enzymes in serum at 100 mg/kg bw/day. In rats, no toxic effects were seen up to the highest dose tested, 50 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice and rats and long-term dietary studies in dogs. The study in rats reported hepatic spongiosis and increased absolute liver and kidney weights at doses of 45 mg/kg bw/day and above. Effects in both mice and dogs were confined to decreased bodyweight gains at the highest doses tested (427 mg/kg bw/day for mice, 500 mg/kg bw/day for dogs). The lowest overall NOEL was 11 mg/kg bw/day (rat), based on liver spongiosis and decreased bodyweight gain.

Carcinogenicity Based on a 2-year dietary study in rats and an 18-month dietary study in mice, there is no evidence of carcinogenicity for napropamide.

Genotoxicity: Napropamide is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of reproductive effects, delayed development or teratogenicity.

Poisons Schedule: Napropamide is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for napropamide was determined as follows:

$$0.4 \text{ mg/L} = \frac{11 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 11 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES.

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

EXTOXNET (1993). Pesticide Information Profile – Napropamide. http://pmep.cce.cornell.edu/profiles/extoxnet/metiram-propoxur/napropamide-ext.html.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA Method 632.1. The Determination of Carbamate and Amide Pesticides in Municipal and Industrial Wastewater. United States Environmental Protection Agency.

Williams WM, Holden PW, Parsons DW, Lorber MN (1988). Pesticides in Groundwater Data Base. 1988 Interim report. US Environmental Protection Agency. Office of Pesticide Programs. Environmental Fate and Effects Division. Environmental Fate and Ground Water Branch.

NOTE: Important general information is contained in PART II, Chapter 6

Nicarbazin

(endorsed 2011)

GUIDELINE

Based on human health concerns, nicarbazin in drinking water should not exceed 1 mg/L.

RELATED CHEMICALS

Nicarbazin (CAS 330-95-0) does not belong to a recognised chemical class. It is a synthetic complex composed of an equimolar amount of 4,4'-dinitrocarbanilide (DNC) and 2-hydroxy-4,6-dimethylpyrimidine (HDP). There are no related pesticides (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, nicarbazin would not be a health concern unless the concentration exceeded 1 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Users: Nicarbazin is a non-ionophoric parasiticide that is used as a coccidiostat feed additive for the prevention of faecal and intestinal coccidiosis in broiler chickens.

There are registered products containing nicarbazin in Australia. The products are intended for professional use and are available as concentrated granular and powder formulations for addition to feed. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to nicarbazin and its metabolites is residues in food. Residue levels in chicken products produced according to good veterinary practice are generally low.

The veterinary use of nicarbazin provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of nicarbazin in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of nicarbazin in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

No suitable analytical techniques have been identified, but the use of high performance liquid chromatography–tandem mass spectrometry is expected to be suitable for residue levels of this pesticide in water. This technique has previously been used for the analysis of nicarbazin in poultry products.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for nicarbazin is 0.4 mg per kg of bodyweight (mg/kg bw) based a no-observed-effect level (NOEL) of 200 mg/kg bw/day in a rat developmental study, with a safety factor of 500.

The previous ADI of 2 mg/kg bw established in 1982 was based on a NOEL of 200 mg/kg bw/day from a 2-year rat dietary study. The ADI incorporated a safety factor of 100.

The current acute reference dose (ARfD) is 0.4 mg/kg bw, based on a NOEL of 200 mg/kg bw/day in a rat developmental study with a safety factor of 500.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Nicarbazin consists of equimolar amounts of 4,4'-dinitrocarbanilide (DNC) and 2-hydroxy-4,6-dimethylpyrimidine (HDP). HDP is readily absorbed via the gastrointestinal tract while DNC is not. There is no evidence of bioaccumulation. Elimination is mainly via the urine for HDP and via the faeces for DNC.

Acute effects: Nicarbazin has a low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: Information on short-term effects is very limited. The available data indicate that in rats there are effects on the kidney at 500 mg/kg bw/day, while in dogs there is bile duct proliferation at 1600 mg/kg bw/day.

Long-term effects: A 2-year dietary study in rats reported no treatment-related effects up to 300 mg/kg bw/day of DNC plus 100 mg/kg bw/day of HDP. A 2-year dietary study in dogs reported transitory liver enzyme changes and slight effects on bile duct at highest dose only, namely, 600 mg/kg bw/day of DNC plus 200 mg/kg bw/day of HDP. The NOEL was 180 mg/kg bw/day DNC plus 60 mg/kg bw/day HDP.

Carcinogenicity: Based on long-term studies in rats, there is no evidence of carcinogenicity for nicarbazin.

Genotoxicity: Nicarbazin was weakly positive in *in vitro* bacterial studies, but *in vivo* studies were negative. There is insufficient information to determine its genotoxic potential.

Reproductive and developmental effects: There are no studies available to examine the potential effects of nicarbazin on reproductive parameters. A developmental study in rats reported effects on the dams and foetuses at 600 mg/kg bw/day, but the study was of poor quality. The NOEL was 200 mg/kg bw/day and is the basis of the current ADI.

Poisons Schedule: Nicarbazin is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 1 mg/L for nicarbazin was determined as follows:

$$1 \text{ mg/L} = \frac{200 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 500}$$

where:

- 200 mg/kg bw/day is the NOEL based on a developmental study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 500 is a safety factor applied to the NOEL from a developmental study conducted in rats. The safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variations, and an additional safety factor of 5 for limitations in the current database.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Nickel

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of nickel in drinking water should not exceed 0.02 mg/L.

GENERAL DESCRIPTION

Drinking water generally contains very low concentrations of nickel. Concentrations reported overseas are usually less than 0.01 mg/L. Higher concentrations, up to 0.5 mg/L, have been reported where water has been in prolonged contact with nickel-plated tap and plumbing fittings; however, these higher concentrations are unusual.

Nickel is used in the electroplating industry and in alloys used in the chemical, marine, nuclear and aerospace industries. It is used as a catalyst in industrial processes, and in oil refining. Main releases to the environment are from the burning of fossil fuels and in waste discharges from electroplating industries.

Nickel is present in many foods. Highest concentrations occur in cocoa, soy beans and some cereals. It has been estimated that the average daily dietary intake is between 0.1 mg/day and 0.3 mg/day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, concentrations of nickel range up to 0.03 mg/L, with typical concentrations less than 0.01 mg/L.

TREATMENT OF DRINKING WATER

Nickel can be co-precipitated with iron and manganese oxides.

MEASUREMENT

The nickel concentration in drinking water can be determined using inductively coupled plasma emission spectroscopy or graphite furnace atomic absorption spectroscopy (APHA Method 3500-Ni Parts B and C 1992). The limits of determination are approximately 0.02 mg/L and 0.005 mg/L respectively. Lower concentrations can be determined with pre-concentration using chelation or solvent extraction techniques.

HEALTH CONSIDERATIONS

Intestinal absorption of soluble nickel in drinking water can be as high as 27%, compared with only 0.7% from food. After absorption, nickel appears to be distributed to most organs, with higher amounts in the kidneys, lung and liver. It can cross the human placenta.

An extensive review and summary of the human and animal toxicity data for nickel is available (IPCS 1991).

In humans, long-term exposure may result in toxic effects to the kidney. Increased beta-microglobulin concentrations were reported among electroplating workers exposed to high amounts of nickel.

Nickel is known to be a common skin allergen and can cause dermatitis, particularly in younger women. While skin is sensitised, oral intake of low doses (0.0083 mg/kg body weight per day) may provoke contact dermatitis in sensitised individuals.

Several epidemiological studies have demonstrated that inhalation of nickel can cause lung, sinus and nasal cancer. There is no evidence that other organs are affected, or that nickel is carcinogenic when ingested.

Animal studies have reported altered body weights, some evidence of liver toxicity and mild kidney toxicity with high nickel doses (over 100 mg/kg body weight per day). Nickel has also affected the immune system in laboratory mice.

Some nickel compounds are carcinogenic when injected into laboratory animals but not when administered orally. Tests for mutagenicity with strains of bacteria have mostly been negative but gene mutations and chromosome aberrations have been reported in mammalian cells.

The International Agency for Research on Cancer has concluded that nickel compounds are carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans) (IARC 1990).

DERIVATION OF GUIDELINE

The guideline value for nickel in drinking water was derived as follows:

$$0.02 \text{ mg/L} = \frac{5 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 5 mg per kg body weight per day is the no-effect level for altered organ-to-body-weight ratios in a 2-year study with rats (Ambrose 1976).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in applying the results of animal studies to humans (10 for interspecies variations, 10 for intraspecies variations and 10 to compensate for the lack of adequate studies on chronic effects and for increased intestinal absorption when taken on an empty stomach). An additional factor for carcinogenicity was not included as effects only occurred on inhalation (no effects were observed on ingestion) and were localised to the lung and nasal passages.

REFERENCES

Ambrose AM, Larson PS, Borzelleca JG, Hannigar GR Jr (1976). Long term toxicologic assessment of nickel in rats and dogs. *Journal of Food Science and Technology*, 13:181–187.

APHA Method 3500-Ni Part B (1992). Nickel: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Ni Part C (1992). Nickel: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1990). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: chromium, nickel and welding. World Health Organization, IARC, 49.

IPCS (International Programme on Chemical Safety) (1991). Nickel. Environmental Health Criteria, 108. World Health Organization, IPCS.

NOTE: Important general information is contained in PART II, Chapter 6

Nitrate and nitrite

(endorsed 2011)

GUIDELINE

Nitrate: Based on health considerations, the guideline value of 50 mg-NO_3/L (as nitrate) has been set to protect bottle-fed infants under 3 months of age. Up to 100 mg-NO_3/L can be safely consumed by adults and children over 3 months of age.

Where a water supply has between 50 and 100 mg-NO_3/L nitrate, active measures are required to ensure that those caring for infants are aware of the need to use alternative water sources in making up bottle feeds for babies under 3 months of age. Water may be used for bottle-fed infants if the nitrate concentration is between 50 and 100 mg/L, but medical authorities need to be increasingly vigilant and the water must also be known to be microbiologically safe.

Nitrite: Based on health considerations, the concentration of nitrite in drinking water should not exceed 3 mg-NO_2/L (as nitrite).

GENERAL DESCRIPTION

Nitrate and nitrite ions are naturally occurring oxides of nitrogen that make up part of the nitrogen cycle.

Nitrate is formed from the oxidation of organic wastes such as manure, by the action of nitrogen-fixing bacteria in soils, or from lightning strikes through air. Nitrates are also manufactured for use in explosives and inorganic fertilisers.

Intensification of farming practices and sewage effluent disposal to streams have led to increasing amounts of nitrate in some waters, particularly groundwater.

The nitrite ion is relatively unstable and can be formed by the reduction of nitrate in poorly oxygenated waters. It is rapidly oxidised to nitrate and is seldom present in well oxygenated or chlorinated supplies. Chemical and biological processes can result in further reduction to various compounds, including ammonia, or oxidation back to nitrate.

Food, particularly vegetables and cured meat, is the major source of nitrate intake for humans.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, nitrate concentrations range up to 51 mg NO_3/L, with typical concentrations usually less than 0.15 mg NO_3/L. Nitrite is generally not present in significant concentrations, although operational difficulties in chloramination can lead to nitrite formation due to the action of nitrifying bacteria.

Very high nitrate concentrations (up to 1300 mg NO_3/L) have been recorded in some groundwater supplies in rural areas.

TREATMENT OF DRINKING WATER

Conventional water treatment is not effective for nitrate removal. Nitrate reduction facilities are expensive to operate and involve the use of anion exchange resins.

MEASUREMENT

The nitrate concentration in drinking water can be determined by a colorimetric procedure following reduction of nitrate to nitrite using a cadmium column (APHA Method 4500-NO_3 Part E 1992). The limit of determination is 0.01 mg/L. Nitrite can be determined separately using the same procedure but without the reduction column (APHA Method 4500-NO_2 Part B 1992). Alternatively, nitrate and nitrite can be determined using ion chromatography (APHA Method 4110 Part B 1992).

HEALTH CONSIDERATIONS

The toxicity of nitrate to humans is thought to be solely due to its reduction to nitrite. The major biological effect of nitrite in humans is its involvement in the oxidation of normal haemoglobin to methaemoglobin, which is unable to transport oxygen to the tissues. This condition is called methaemoglobinaemia. Young infants are more susceptible to methaemoglobin formation than older children and adults. Other susceptible groups include pregnant women and people with a deficiency of glucose-6-phosphate dehydrogenase or methaemoglobin reductase.

Recently the World Health Organization (WHO 2007) reviewed the toxicity and health information for nitrate and nitrite and retained the drinking water guideline of 50 mg-NO_3/L based on epidemiological evidence for methaemoglobinaemia in infants because it is protective for bottle-fed infants and consequently for other parts of the population. The WHO recommended that water should not be used for bottle-fed infants when nitrate levels are above 100 mg/L, but that it may be used if medical authorities are increasingly vigilant when the nitrate concentration is between 50 and 100 mg/L. However, *the water must also be known to be microbiologically safe*. This caveat was included because the dose response data for methaemoglobin formation is complicated by the more-often-than-not concomitant presence of bacterial contamination and the fact that nitrite formation from nitrate in infants is markedly enhanced by gastrointestinal infections. Indeed some cases of infant methaemoglobinaemia have been described in which increased endogenous nitrite synthesis as a result of gastrointestinal infection appeared to be the only causative factor (FAO/WHO 1996, WHO 2007).

The central role that gastrointestinal infection plays in the aetiology of the disease has lead to an opinion that exogenous nitrate in drinking water is not a significant contributor to the prevalence of methaemoglobinaemia, and that current limits of nitrate in drinking water based solely on the health threat of infantile methaemoglobinaemia may be unnecessarily strict (Avery 1999). Indeed, a recent exposure assessment based on drinking water nitrate levels greater than 50 mg NO_3/L could not identify an exposure-response relationship that related drinking water nitrate level to methaemoglobinaemia (Fewtrell 2004). Nevertheless the collective data indicate that concentrations less than the current 50 mg NO_3/L are not commonly associated with methaemoglobinaemia (Fan and Steinberg 1996, FAO/WHO 1996, WHO 2007).

In animals, laboratory experiments suggest that neither nitrite nor nitrate acts directly as a carcinogen. There is concern that nitrite may react with foods rich with secondary amines to form N-nitroso compounds in the stomach: many of these compounds are known to be carcinogenic in animals. Some epidemiological evidence suggests a relationship between nitrate and gastric cancer in humans, but this has not been confirmed in more definitive analytical studies.

Nitrate is not mutagenic in tests with bacteria and mammalian cells *in vitro*. Chromosome aberrations have been observed in the bone marrow of rats but may be due to the formation of N-nitroso compounds. Nitrite is mutagenic in both *in vivo* and *in vitro* experiments using mammalian cells.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF GUIDELINE

The guideline value of 50 mg NO_3/L for nitrate is set to protect young infants, the most sensitive group (USEPA 1990, WHO 2007). Up to 100 mg NO_3/L can be used by adults and children over 3 months of age without significant health effects.

If the value of 50 mg NO_3/L is exceeded, the local health authority should be informed so that parents can be advised to use rainwater or bottled water in making up feeds for babies under 3 months of age. Water with concentrations up to 100 mg NO_3/L may be used if medical authorities are vigilant and the water is free of microbial contamination (WHO 2007).

The guideline level for nitrite of 3 mg NO_2/L is based on a relative potency for nitrite and nitrate with respect to methaemoglobin formation. WHO (2007) developed the same value for nitrite based on human data that show methaemoglobinaemia caused in infants by doses of nitrite ranging from 0.4 to more than 200 mg/kg of bodyweight. The guideline (rounded value) was derived by using the lowest level of the range (0.4 mg/kg of bodyweight), a bodyweight of 5 kg for an infant and a drinking-water consumption of 0.75 litre.

Because it is possible that nitrate and nitrite may occur simultaneously in drinking-water, and the two have a common toxic effect (methaemoglobinaemia), these compounds should be considered together when judging compliance with the guidelines. The sum of the ratios of the concentration (C) of each to its guideline value (GV) should not exceed unity (WHO 2007). This is a standard screening risk assessment approach based on the assumption of dose additivity. Thus for infants:

$$(C_{Nitrate}/GV_{Nitrate}) + (C_{Nitrite}/GV_{Nitrite}) \leq 1 = (C_{Nitrate}/50\ mg/L) + (C_{Nitrite}/3\ mg/L) \leq 1$$

REFERENCES

APHA Method 4110 Part B (1992). Determination of ions by ion chromatography: ion chromatography with chemical suppression of eluant conductivity. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 4500-NO_3 Part E (1992). Nitrogen (nitrate): Cadmium reduction method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 4500-NO_2 Part B (1992). Nitrogen (nitrite): Colorimetric method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

Avery AA (1999). Infantile methemoglobinemia: reexamining the role of drinking water nitrates. *Environmental Health Perspectives,* 107(7):583-586.

FAO/WHO (1996). Toxicological evaluation of certain food additives and contaminants: Nitrate. Prepared by the Forty-fourth meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), Geneva, World Health Organization, International Programme on Chemical Safety (WHO Food Additive Series 35).

Fan AM, SteinbergVE (1996). Health implications of nitrate and nitrite in drinking water: An update on methemoglobinemia occurrence and reproductive and developmental toxicity. *Regulatory Toxicology and Pharmacology,* 23(1):35-43.

Fewtrell L (2004). Drinking-water nitrate, methemoglobinemia, and global burden of disease: a discussion. *Environmental Health Perspectives,* 112(14):1371-1374.

NOTE: Important general information is contained in PART II, Chapter 6

USEPA (United States Environmental Protection Agency) (1990). Drinking water criteria document on Nitrate/Nitrite. Report TR-1242-608 from ICAIR Life Systems Inc, Cleveland, Ohio. Prepared for Criteria and Standards Division Office of Drinking Water, USEPA, Washington DC.

WHO (World Health Organization) (2007). Nitrate and nitrite in drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Nitrilotriacetic acid (NTA)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of nitrilotriacetic acid in drinking water should not exceed 0.2 mg/L.

GENERAL DESCRIPTION

NTA may be present in drinking water that has been contaminated with sewage, for example by sewage discharge into a river or stream that is then used for drinking water. It is likely to be present in the form of metal complexes rather than the free acid. NTA has been detected in water supplies of municipalities in Canada and the United States at a mean concentration of less than 0.004 mg/L, with a small number of supplies exceeding 0.01 mg/L.

NTA is a chelating agent and forms soluble metal complexes with a number of metal ions including calcium and magnesium. It is used in laundry detergents as a replacement for phosphate, particularly in countries where legislation restricts the use of phosphate-based detergents. It is also used in the treatment of boiler water to prevent scale formation, and in the photographic, metal plating, textile manufacturing, and paper and cellulose industries. It is not widely used in Australia.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

NTA has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

No published reports are available on water treatment procedures to remove NTA from drinking water.

MEASUREMENT

Analysis can be undertaken using gas chromatography with a nitrogen-specific detector after converting NTA to the tri-n-butyl ester (Aue *et al.* 1972, Malaiyandi *et al.* 1979). The limit of determination is 0.0002 mg/L.

HEALTH CONSIDERATIONS

NTA is poorly absorbed by humans compared to experimental animals. It is rapidly excreted unchanged, but may be briefly retained in bone, probably due to the formation of complexes with calcium ions.

Data on the health effects in humans are scarce.

A number of long-term toxicity studies with animals have all shown similar results. No adverse effects are observed with low doses, but higher doses (30 mg/kg body weight per day) can cause some adverse effects to the kidney and urinary tract. The formation of kidney, urinary tract and bladder tumours has been reported in rats after prolonged exposure to high doses, but the tumours are believed to be the result of chelation of metal ions in the urinary tract.

Tests for mutagenic activity using bacteria have been negative; however, the NTA–iron complex is mutagenic in mammalian cells *in vitro*.

The International Agency for Research on Cancer has concluded that NTA is possibly carcinogenic to humans (Group 2B, no data in humans but sufficient evidence in animals) (IARC 1990).

DERIVATION OF GUIDELINE

The assessment of the toxicological data for NTA by the World Health Organization (WHO) has been used without review. The guideline value was determined as follows:

$$0.2 \text{ mg/L} = \frac{10 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.5}{2 \text{ L/day} \times 1000}$$

where:

- 10 mg/kg body weight per day is the no-effect level from a 2-year feeding study using rats (Nixon *et al*. 1972).
- 70 kg is the average weight of an adult.
- 0.5 is the proportion of total daily intake attributable to the consumption of water, based on a WHO assessment of distribution.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for potential carcinogenic effects).

REFERENCES

Aue WA, Hastings CR, Gerhardt KO, Pierce JO, Hill HH, Mosman RF (1972). The determination of part-per-billion levels of citric and nitrilotriacetic acids in tap water and sewage effluents. *Journal of Chromatography*, 72:259–267.

IARC (International Agency for Research on Cancer) (1990). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Some flame retardants and textile chemicals, and exposures in the textile manufacturing industry. World Health Organization, IARC, 48.

Malaiyandi M, Williams DT, O'Grady R (1979). A national survey of nitrilotriacetic acid in Canadian drinking water. *Environmental Science and Technology*, 13:59–62.

Nixon GA, Buehler EV, Niewenhuis RJ (1972). Two-year rat feeding study with trisodium nitrilotriacetate and its calcium chelate. *Toxicology and Applied Pharmacology*, 21:244–252.

NOTE: Important general information is contained in PART II, Chapter 6

N-Nitrosodimethylamine (NDMA)

(endorsed 2011)

GUIDELINE

Based on health considerations, the concentration of NDMA in drinking water should not exceed 0.0001 mg/L (100 ng/L).

Action to reduce NDMA is encouraged, but must not compromise disinfection, as non-disinfected water poses significantly greater risk than NDMA.

GENERAL DESCRIPTION

N-Nitrosodimethylamine ($C_2N_6N_2O$)(CAS No. 62-75-9) is a member of the dialkylnitrosamine family. Other names for the compound include *N*-methyl-*N*-nitrosomethanamine, dimethylnitrosamine, and nitrous dimethylamine. The compound is also referred to by the acronyms NDMA, DMN and DMNA. The most recognisable acronym in the context of water treatment and water recycling is NDMA.

NDMA is a polar compound with a molecular weight of 74.08 g/mol, a water solubility of >10 g/100 ml (at 19ºC) and a Log Octanol/Water partition coefficient of –0.57. Pure NDMA exists as yellow liquid with a density of 1.006 g/cm^3, a boiling point of 151-154ºC and a vapour pressure of 1080 Pa at 25ºC.

NDMA is used as an industrial solvent, an anti-oxidant, a rubber accelerator, and in the preparation of polymers, where it may be used as an initiator or a plasticiser. The compound has been used in the production of rocket fuel, as a biocide for nematodes, and an intermediate for 1,1-dimethylhydrazine to inhibit nitrification of soils.

NDMA is formed under mildly acidic conditions by the reaction of natural and synthetic secondary, tertiary or quaternary amines with nitrate and nitrite. Precursor amines include alkylamines, dimethylamine (DMA), tetramethylthiuram disulfide (thiram) and polyelectrolytes used in water and wastewater treatment. NDMA is also produced as a by-product of chloramination of drinking water (due to the presence of dimethylamine in source waters subject to wastewater discharges or the oxidation of natural organic matter by chlorine in the presence of ammonia) and to a lesser extent by chlorination. NDMA formation can be facilitated in soils by biochemical pathways in micro-organisms, and this compound is resistant to microbial degradation under both aerobic and anaerobic conditions. Ozonation of drinking water contaminated with the fungicide tolyfluamide can also lead to the formation of NDMA.

NDMA can exist in the liquid and vapour phase and may be associated with airborne particulates. The compound has been detected in indoor air contaminated with tobacco smoke at concentrations of up to 240 ng/m^3. Detectable levels in outdoor air have been reported in the immediate vicinity of point sources (e.g. chemical production facilities). NDMA has been detected in preserved foods, such as smoked and salted fish and meat and sausages cured by nitrates. Studies conducted in the 1970s and 1980s found NDMA in foodstuffs at levels up to 17,200 ng/kg for cured meat products such as bacon, 68,000 ng/kg for smoked cheese and 9,200 ng/L for beer; although these levels should be viewed with caution as the concentrations were determined using analytical methods available at the time. Moreover, since that time, efforts have been made to reduce the amount of NDMA in foods by limiting the amount of allowable nitrate in preservation, prohibiting the use of nitrate for certain food groups, and the inclusion of nitrosation inhibitors.

In addition to pre-formed NDMA occurring in some foods, NDMA is generated in the stomach through nitrosation of secondary amines in ingested food, especially fish and meat. This process also involves reaction with nitrate and nitrite from foodstuffs and nitrate formed in the stomach, and is influenced by other food components that may enhance or inhibit nitrosation reactions. For these reasons, it is difficult to estimate the amount of NDMA formed endogenously in the human body.

NDMA is absorbed via the gastrointestinal and respiratory tracts, and may also be absorbed through the skin, but at much lower rates. Distribution in the body is uniform and rapid, and it is metabolised rapidly, with an estimated half life of 4 hours, based on observations in rodents. Excretion is primarily via carbon dioxide in expired air, with only a small percentage persisting as NDMA in the urine.

A worst case estimate for NDMA exposure from contaminated outdoor air and consumption of food and water indicated 5.0-16.0 ng/kg of body weight per day for a 29-50 year old adult (WHO 2006). Drinking water was estimated to account for 0.3-1.0 ng/kg of body weight per day based on a mean NDMA concentration of 12 ng/L and a maximum concentration of 40 ng/L in water. Food was estimated to account for 4.3-11 ng/kg of body weight per day. Cigarette smoking was a more significant source of NDMA exposure, with smokers estimated to have an intake of 1.0-80 ng/kg of bodyweight per day from mainstream smoke, and people with heavy exposure to smoke-contaminated indoor air, an intake of 40-130 ng/kg bodyweight per day from smoke. These estimates did not take into account the endogenous formation of NDMA in the digestive tract, and they indicate that drinking water forms only a minor component of exposure to exogenous NDMA (less than 10%).

Another assessment, incorporating estimates of the possible range of endogenous NDMA formation using data from *in vivo* and *in vitro* studies, indicated that drinking water contributed around 2.7% of daily NDMA intake when only exogenous sources were assessed, but only about 0.02% when endogenous NDMA formation was also taken into account (Fristachi and Rice 2007).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

There are no data in the public domain or peer reviewed literature on NDMA in Australian drinking water distribution systems and water treatment plants. Anecdotal evidence suggests a bi-modal distribution, with several water authorities indicating that NDMA is present at levels at or near the limit of determination of 1 to 2 ng/L, whereas preliminary sampling and analysis by other authorities indicates levels in the range of 60-90 ng/L. A recent report from South Australia has indicated that NDMA may originate from rubber components of newly commissioned drinking water pipelines, regardless of the disinfectant used. This may account at least partly for the divergent results reported by different water suppliers.

MEASUREMENT

Analytical methods for NDMA detection have been developed with a sensitivity at the nanogram per litre (ng/L) level. The methods developed by the United States Environmental Protection Agency (USEPA 2004) and the Ontario Ministry of the Environment (OME 2004) include a concentration and separation step prior to quantification by gas chromatography and mass spectrometry. Internal standards for each method are based on the use of the deuterated analogue of NDMA, NDMA-d6, as the surrogate. The OME method was developed specifically for use in drinking water. In this method, NDMA is extracted onto Ambersorb 572 and eluted using dichloromethane. NDMA is separated from the solvent using capillary column gas chromatography, and quantified by high resolution mass spectrometry at a detection level of 0.4 ng/L, with a reporting detection level of 1.0 ng/L. The USEPA method was developed for large scale surveys and can be used to detect NDMA and seven other nitrosamines. Following solid phase extraction and elution, the nitrosamines may be separated by gas chromatography and quantified via chemical ionisation tandem mass spectrometry, with a detection level of 0.28 ng/L and a limit of determination of 1.6 ng/L.

NOTE: Important general information is contained in PART II, Chapter 6

HEALTH CONSIDERATIONS

NDMA is absorbed through the gastrointestinal tract and subsequently distributes uniformly and rapidly. It can cross the placenta and may be present in breast milk. The metabolic half life in rodents is around 4 hours. It is excreted largely via exhaled carbon dioxide, with limited amounts excreted unchanged in urine.

NDMA is carcinogenic in experimental animals through several exposure routes, including ingestion in drinking water. In 1987 the International Agency for Research on Cancer (IARC) classified NDMA as a Group 2A chemical, probably carcinogenic to humans. The mechanism by which NDMA causes cancer is believed to involve biotransformation in the liver by microsomal enzymes, generating the methyldiazonium ion which subsequently forms DNA adducts.

A number of epidemiological studies have shown an association between NDMA intake from food and increased risks of gastric or colorectal cancer, although the data are not sufficient to derive a quantitative dose-response relationship for cancer risk in humans. These studies did not consider exposure to NDMA from drinking water, or endogenous generation of NDMA in the body.

Various other N-nitrosamine compounds with structures related to NDMA are known to occur in water supplies. The toxicological properties of these compounds have not been well characterised, however it is believed likely that some are also carcinogenic.

DERIVATION OF GUIDELINE VALUE

The World Health Organization has derived a guideline value for NDMA in drinking water based on a study of hepatic biliary cystadenomas in rats that used a wide range of NDMA exposure doses (from 0.033 mg/L to 16.896 mg/L). This dataset was used to derive a tumorigenic dose (TD_{05}) for NDMA corresponding to a dose level that causes a 5% increase in tumour incidence over the background level. The TD_{05} values were used to calculate a unit risk, which represents the increase in risk per unit increase in dose. Using the most sensitive endpoint observed in the animal study and conservative assumptions, a guideline value of 100 ng/L was derived, corresponding to an excess lifetime cancer risk of 1 in 100,000. This methodology treats the risk from exposure to NDMA in drinking water in isolation from other sources of NDMA exposure.

While adopting the same numerical value, a different approach has been taken to derive the guideline value for NDMA in Australian drinking water supplies. In assessing the potential public health benefits associated with regulation of this compound, the following factors were considered:

- NDMA has been demonstrated to be carcinogenic in animals, and is probably carcinogenic in humans.
- NDMA levels in drinking water may be an indicator of the presence of structurally related compounds, some of which may also have carcinogenic properties.
- The current level of exposure to NDMA from food is uncertain, due to lack of recent analytical data; however, even with changes in food preservation techniques since the 1970s, it is probable that exposure through food is at least 5 to 10 times greater than exposure from drinking water.
- There is evidence that exposure from endogenous formation of NDMA in the stomach may greatly exceed dietary exposures from both food and water.

In these circumstances, the adoption of a guideline value corresponding to a 1 in 1,000,000 lifetime cancer risk (the usual ADWG target level for health risks from carcinogens) was deemed inappropriate, as this would impose a disproportionate regulatory burden on water suppliers while having little impact on total population exposures. Nevertheless, it was judged to be prudent to limit levels of NDMA in drinking water, given that this will probably reduce exposure to a range of related but as yet largely uncharacterised *N*-nitrosamine compounds that may pose potential health risks. For these reasons, a guideline value of 100 ng/L has been adopted.

NOTE: Important general information is contained in PART II, Chapter 6

LIMITING FORMATION IN DRINKING WATER

Reducing the occurrence of NDMA in drinking water systems may conflict with the goals of maintaining a persistent chlorine residual in distributions and controlling levels of other disinfection by-products such as trihalomethanes and haloacetic acids. The potential for NDMA formation may be reduced by avoiding chloramination through the removal of ammonia prior to disinfection, or by operating the system for breakpoint chlorination. If NDMA is a problem, treatment using UV irradiation in the prescence of hydrogen peroxide is an option to reduce NDMA levels while maintaining a chloramine residual. NDMA cannot be removed by air stripping or adsorption, due to its vapour pressure, solubility in water and limited partitioning at interfaces. It is only partially removed (<50%) by liquid phase pressure-driven separation processes such as reverse osmosis.

REFERENCES

Fristachi A, Rice G (2007). Estimation of the total daily oral intake of NDMA attributable to drinking water. *Journal of Water and Health* 5(3):341–355.

OME (Ontario Ministry of the Environment) (2004). *The determination of* N-*nitrosodimethylamine (NDMA) in water by gas chromatography-high resolution mass spectrometry (GC-HRMS)*. Toronto, Ontario, Ontario Ministry of the Environment, Laboratory Services Branch (Report No. NDMA-E3291).

USEPA (United States Environmental Protection Agency) (2004). *Method 521: Determination of nitrosamines in drinking water by solid phase extraction and capillary column gas chromatography with large volume injection and chemical ionization tandem mass spectrometry (MS/MS)*. Washington, DC, United States Environmental Protection Agency (EPA Document No. EPA/600/R-05/054; http://www.epa.gov/nerlcwww/m_521.pdf).

WHO (World Health Organization) (2002). *Concise International Chemical Assessment Document (CICAD) 38. N-Nitrosodimethylamine*. Available from www.inchem.org.

WHO (World Health Organization) (2006). N-Nitrosodimethylamine in drinking water. Background document for development of WHO Guidelines on Drinking-water Quality. World Health Organization.

NOTE: Important general information is contained in PART II, Chapter 6

Norflurazon

(endorsed 2011)

GUIDELINE

Based on human health concerns, norflurazon in drinking water should not exceed 0.05 mg/L.

RELATED CHEMICALS

Norflurazon (CAS 27314-13-2) belongs to the pyridazinone class of chemicals. Another pesticide in this class is maleic hydrazide (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, norflurazon would not be a health concern unless the concentration exceeded 0.05 mg/L. Excursions above this level over a short to medium term are of concern as the health-based guideline is based on effects observed in a 3-month study.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Norflurazon is a pre-emergent herbicide for the control of annual grasses, broad-leaf and perennial weeds in agricultural crops.

There is at least one registered product containing norflurazon in Australia. Norflurazon products are intended for professional use and are available as a concentrated solution that is diluted and applied by aerial and ground spray to soil. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to norflurazon and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of norflurazon may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data were found on the occurrence of norflurazon in Australian waters. Norflurazon has been detected in several wells in Florida, USA, at levels approaching or in some cases exceeding 0.03 mg/L (the US Health Advisory Level) (USEPA 1996).

TREATMENT OF DRINKING WATER

No specific data on the treatment of norflurazon in drinking water have been identified.

MEASUREMENT

Norflurazon can be analysed by gas chromatography with a nitrogen–phosphorous specific detector (USEPA Method 645).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for norflurazon is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.5 mg/kg bw/day from a medium-term dietary study in dogs. The NOEL is based on increased liver weights. The ADI incorporates a safety factor of 100 and was first established in 1984.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Norflurazon is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, and is rapidly excreted in urine and faeces, with excretion being complete by 4 days.

Acute effects: Norflurazon has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pigs.

Short-term effects: In short-term (21-day) dietary studies in rats, there was increased testis and kidney weight without accompanying histopathological changes at 100 mg/kg bw/day and above.

In medium-term dietary studies in rats, increased kidney weight without accompanying changes in histopathology was reported at a dose level of 127 mg/kg bw/day. In medium-term dietary studies in dogs, increased liver weight was reported at 10 mg/kg bw/day, with a NOEL of 1.5 mg/kg bw/day. This NOEL is the basis for the current ADI.

Long-term effects: Long-term dietary studies in mice and rats reported decreased bodyweight and food consumption and significant changes in relative organ weights. The overall NOEL was 18 mg/kg bw/day in rats.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for norflurazon.

Genotoxicity: Norflurazon is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and a developmental toxicity study in rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Norflurazon is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.05 mg/L for chemical was determined as follows:

$$0.05 \text{ mg/L} = \frac{1.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.5 mg/kg bw/day is the NOEL based on a medium-term (3-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1996). RED Factsheet – Norflurazon. EPA-738-F-96-012 July 1996. Office of Prevention, Pesticides and Toxic Substances, USEPA.

USEPA Method 645. The determination of certain amine pesticides and lethane in municipal and industrial wastewater. United States Environmental Protection Agency.

NOTE: Important general information is contained in PART II, Chapter 6

Omethoate

(endorsed 2011)

GUIDELINE

Based on human health concerns, omethoate in drinking water should not exceed 0.001 mg/L.

RELATED CHEMICALS

Omethoate (CAS 1113-02-6) belongs to the organophosphate class of chemicals. There are many other pesticides in this class including dimethoate, fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, omethoate would not be a health concern unless the concentration exceeded 0.001 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Omethoate is an insecticide and acaricide (miticide) for the control of aphids, flies, caterpillars, bugs, moths, mites and fleas. Omethoate is a metabolite of dimethoate.

There are registered products containing omethoate in Australia. The products are intended for both professional and home garden use. Omethoate is formulated as a liquid concentrate, to be diluted and sprayed onto plants and bare earth. It can be applied to a variety of crops including pastures, legumes, oilseeds, faba bean, poppies, ornamentals, citrus, apples, cotton, lupins, onions, pears and potatoes. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to omethoate are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice and potential dietary exposure are generally low.

Agricultural use of omethoate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of omethoate in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of omethoate in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Omethoate can be determined in water by hydrophilic interaction liquid chromatography with tandem mass spectrometry, with a limit of quantitation of 0.05 µg/L (Hayama *et al.* 2008). A capillary electrophoresis method has also been developed for the analysis of omethoate with a method detection limit of 50 µg/L (Tao *et al.* 2008). A variety of solid phase extraction materials may be used for the effective extraction of omethoate from water (Geiss and Gebert 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for omethoate is 0.0004 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.04 mg/kg bw/day from 2-year dietary study in rats. The NOEL was based on inhibition of acetylcholinesterase. The ADI incorporates a safety factor of 100 and was established in 2005.

The acute reference dose (ARfD) of 0.003 mg/kg bw/day for omethoate was established in 2005, based on a NOEL of 0.25 mg/kg bw/day from an acute neurotoxicity study in rats. The NOEL was based on inhibition of acetylcholinesterase activity. The ARfD incorporates a safety factor of 100.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Omethoate is absorbed rapidly and extensively from the gastrointestinal tract in rats and is rapidly eliminated in the urine, mainly as unchanged omethoate (88% eliminated after 8 hours). It is widely distributed throughout the body with the highest levels found in the thyroid. Identified metabolites include N-methyl-methyl-sulfinyl-acetamide, O-desmethylated omethoate, O,O dimethyl phosphoric acid and O,O-dimethyl phosphorothioic acid.

Acute effects: Omethoate has high acute oral toxicity and moderate acute dermal toxicity. It can cause skin sensitisation in guinea pigs.

Short-term and long-term effects: Short-term dermal exposure studies and long-term dietary exposure studies in rats reported symptoms indicative of nervous system toxicity. A 2-year dietary study in rats reported inhibition of red blood cell and brain cholinesterase activity at dose levels equivalent to 0.12 mg/kg bw/day and above. The NOEL of 0.04 mg/kg bw/day from this study is the basis of the ADI.

Carcinogenicity: There was evidence of benign thyroid tumours at high dose levels in rats. These levels were well in excess of the expected levels of human exposure.

Genotoxicity: Omethoate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A multigeneration reproduction study in rats reported no evidence of effects on reproductive parameters. Developmental studies in rats and rabbits reported maternal and foetal toxicity as a result of cholinesterase inhibition at 1 mg/kg bw/day in rats and 0.2 mg/kg bw/day in rabbits. There was no evidence of teratogenicity.

Neurotoxicity: Omethoate did not cause delayed neurotoxicity in tests conducted on hens.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Omethoate is in Schedules 5, 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.001 mg/L for omethoate was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.04 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.04 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated amount (maximum) of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Geiss S, Gebert S (2006). Extraction of highly polar organophosphorus pesticides from water. *Acta Hydrochimica et Hydrobiologica,* 34(5):464-473.

Hayama T, Yoshida H, Todoroki K, Nohta H, Yamaguchi M (2008). Determination of polar organophosphorus pesticides in water samples by hydrophilic interaction liquid chromatography with tandem mass spectrometry. *Rapid Communications in Mass Spectrometry,* 22(14):2203-2210.

Tao YG, Wang YM, Ye LB, Li HF, Wang Q (2008). Simultaneous determination of omethoate and dichlorvos by capillary electrophoresis. *Bulletin of Environmnetal Contamination and Toxicology*, 81(2):210-215.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Organotins
dialkyltins
tributyltin oxide

(endorsed 1996)

GUIDELINE

Dialkyltins: Data are inadequate to set a guideline value for drinking water.

Tributyltin oxide: Based on health considerations, the concentration in drinking water should not exceed 0.001 mg/L.

GENERAL DESCRIPTION

The group of compounds known as the organotins comprises a large number of compounds with different properties and applications. Of these the dialkyl and tributyltin compounds have some application in the water industry and are the ones most likely to be found in drinking water supplies.

The dialkyltins are widely used as stabilisers in plastic, and may leach out of PVC water pipes for a short time after installation. In one study, dibutyltin sulfide was detected at a concentration of 0.01 mg/L in water that was in static contact with PVC pipes.

Tributyltins are used as biocides and have occasionally been detected in raw water in Canada, the United States, the United Kingdom and Switzerland, probably because of their use as antifouling agents on boats. The use of tributyl-organotin compounds, particularly tributyltin oxide, in antifouling paints has now been banned in a number of countries because it is extremely toxic to aquatic life. Tributyltin is also used as a biocide in boiler waters. Other organotins are unlikely to be found in water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Organotins have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

No published reports are available on water treatment procedures that can be used to remove organotins from drinking water.

MEASUREMENT

The organotins can be analysed using a solvent extraction procedure (Greaves and Unger 1988). They are extracted using a hexane–tropolone mixture and derivatised to form hexylbutyltins. Analysis is by gas chromatography with flame photometric detection. Limits of determination are less than 0.000002 mg/L (2 ng/L).

HEALTH CONSIDERATIONS

Few data are available on the absorption and distribution of organotins in the body, but animal studies have reported that some of the compounds are poorly absorbed, and distributed primarily to the liver and kidney.

An extensive review and summary of the human and animal toxicity data for tributyltin compounds is

available (IPCS 1990).

The dialkyltins have low general toxicity. A study using rats fed dialkyltin for 3 months reported depressed growth and mild anaemia only at the highest dose used (4mg/kg body weight per day). No toxic effects were observed at lower doses. Other studies with rats and dogs reported similar results. Carcinogenicity bioassays with animals have been inconclusive.

No data are available on the ingestion of tributyltin oxide in humans, although occupational information and dermal exposure are known to cause irritation. A number of long-term animal studies have been undertaken using tributyltin oxide. A 2-year chronic toxicity study using rats concluded that doses of 50 mg/kg body weight per day can induce toxicity to some organs including the thyroid and pituitary glands. A no-effect level of 0.5 mg/kg body weight per day was established from this study (Wester *et al.* 1990). An immunotoxicity study on the suppression of resistance to nematodes in rats identified a no-effect level of 0.025 mg/kg body weight per day (Vos *et al.* 1990). The latter immunotoxicity study is considered more sensitive but the significance to humans is questionable. The no-effect levels agree to about an order of magnitude.

Tributyltin oxide was not mutagenic with bacteria and yeast but caused a significant increase in the number of benign tumours of the pituitary gland when fed to rats for 2 years.

DERIVATION OF GUIDELINE

The guideline value of 0.001 mg/L for tributyltin oxide in drinking water was determined as follows:

$$0.001 \text{ mg/L} = \frac{0.025 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.025 mg/kg body weight per day is the no-effect level from a 18-month immunotoxicity study using rats (Vos *et al.* 1990).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations).

The World Health Organization guideline value of 0.002 mg/L was based on 20% of total daily intake coming from drinking water. The proportion contributed by drinking water to the total Australian intake is probably less.

REFERENCES

Greaves J, Unger MA (1988). A selected ion monitoring assay for tributyltin and its degradation products. *Biomedical and Environmental Mass Spectrometry*, 15:565–569.

IPCS (International Programme on Chemical Safety) (1990). Tributyltin compounds. Environmental Health Criteria, 116. World Health Organization, IPCS.

Vos JG, DeKlerk A, Krajnc EI, VanLoveren H, Rozing J (1990). Immunotoxicity of bis(tri-n-butyltin)oxide in the rat: Effects of thymus dependent immunity and on nonspecific resistance following long-term exposure in young versus aged rats. *Toxicology and Applied Pharmacology*, 105:144–155.

Wester PW, Krajnc EI, van Leeuwen FXR, Loeber JG, van der Heijden CA, Vaessen HAMG, Helleman PW (1990). Chronic toxicity and carcinogenicity of bis(tri-n-butyltin)oxide (TBTO) in the rat. *Food and Chemical Toxicology*, 28:179–196.

NOTE: Important general information is contained in PART II, Chapter 6

Oryzalin

(endorsed 2011)

GUIDELINE

Based on human health concerns, oryzalin in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Oryzalin (CAS 19044-88-3) belongs to the dinitroaniline class of chemicals. Other pesticides in this class include butralin and pendimethalin (Tomlin 2006).

HUMAN RISK STATEMENT

There are currently insufficient data on which to base a human health risk statement.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Oryzalin is a herbicide used for the pre-emergent control of certain annual grasses and broad-leaf weeds in non-bearing and bearing fruit and nut orchards, vineyards, nursery stock, ornamentals and amenity plantings. In addition, it is used for the pre-emergent control of annual ryegrass, phalaris, wireweed (hogweed) and deadnettle in wheat, barley and canola.

There are registered products containing oryzalin in Australia. The products are intended for professional use and are available as a granular formulation, suspension concentrates or emulsifiable concentrates. The concentrates are diluted and applied by ground boom, hand spray or overhead irrigation, and the granules spread by hand or mechanical spreader. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to oryzalin is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of oryzalin may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for oryzalin in Australian waters could be found. In the USA, the highest predicted concentration of oryzalin in surface and groundwater was 90 and 0.9 µg/L, respectively (USEPA 2004). The highest measured concentration in surface and groundwater was 1.9 and 0.018 µg/L, respectively, while concentrations of oryzalin in US drinking water samples ranged from 0.01 to 0.07 µg/L (USEPA 2004).

TREATMENT OF DRINKING WATER

No data on drinking water treatment removal efficiency were found for oryzalin.

MEASUREMENT

Oryzalin in water can be measured by high performance liquid chromatography, with a method detection limit of 0.14 µg/L (USGS 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for oryzalin is 0.1 mg per kg body weight (mg/kg bw), based on a no-observed-effect level (NOEL) of 12 mg/kg bw/day established in 1982. It is assumed this incorporates a safety factor of 100. The basis of this ADI cannot be traced with current records, although, according to available United States reports, it is probably based on a NOEL of 12 mg/kg bw/day from a long-term rat study.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

No evaluation report on the toxicity of oryzalin is currently available from the Office of Chemical Safety and Environmental Health (OCSEH).

Metabolism: No studies have been evaluated in Australia.

Short-term/long-term effects: No studies have been evaluated in Australia.

Carcinogenicity: No studies have been evaluated in Australia. Oryzalin has not been evaluated by International Agency for Research in Cancer.

Genotoxicity: No studies have been evaluated in Australia.

Reproductive and developmental effects: No studies have been evaluated in Australia.

Poisons Schedule: Oryzalin is not scheduled in the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for oryzalin was determined as follows:

$$0.4 \text{ mg/L} = \frac{12 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 12 mg/kg bw/day is the NOEL on which the existing ADI was based; however further details are unknown.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is assumed to be the safety factor applied to the ADI, which is a safety factor typically applied to a NOEL derived from animal studies. The safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation. This is consistent with US reports.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2004). Report of the Food Quality Protection Act (FQPA) Tolerance Reassessment Progress and Risk Management Decision (TRED) for Oryzalin. EPA 738-R-04-009. USEPA.

USGS (United States Geological Survey) (2006). Pesticides in the Nation's Streams and Ground Water, 1992–2001. National Water Quality Assessment Program, USGS.

NOTE: Important general information is contained in PART II, Chapter 6

Oxamyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, oxamyl in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Oxamyl (CAS 23135-22-0) belongs to the carbamate class of chemicals. There are many pesticides in this class including aldicarb, methomyl, carbaryl and methiocarb (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, oxamyl would not be a health concern unless the concentration exceeded 0.007 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on effects on foetal bodyweight following short-term exposure.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Oxamyl is an insecticide for the control of weevil borers and nematodes in agricultural crops.

There is at least one registered product containing oxamyl in Australia. Oxamyl products are intended for professional use and are available as a liquid concentrate formulation applied by stem injection and hand spray to banana crops, and by irrigation to tomato and capsicum crops. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to oxamyl and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of oxamyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data on occurrence of oxamyl in Australian waters could be found. The United States Environmental Protection Agency estimates environmental concentration of 0.001 mg/L in surface waters and 0.004 mg/L in groundwater in the USA (USEPA 2007).

TREATMENT OF DRINKING WATER

No specific data on the treatment of oxamyl in drinking water have been identified.

MEASUREMENT

Oxamyl can be measured by routine high-performance liquid chromatography–mass spectrometry analysis, with a limit of reporting of 0.001 mg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for oxamyl is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a developmental study in rats. The NOEL is based on reduced foetal weights. The ADI incorporates a safety factor of 100 and was established in 1993.

The previous ADI for oxamyl was 0.03 mg/kg bw/day, based on NOELs of 2.5 mg/kg bw/day from long-term dietary studies in rats and dogs. The ADI was amended in 1993 following consideration of the developmental study in rats that demonstrated a lower overall NOEL.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Oxamyl is readily and extensively absorbed in rats. It is not extensively metabolised, and is excreted in the urine almost completely within 168 hours.

Acute effects: Oxamyl has high acute oral toxicity and low dermal toxicity. It is not a skin sensitiser in guinea pigs.

Short-term effects: Short-term (21-day) dermal studies in rabbits and short-term (29-day) dietary studies in rats reported decreases in cholinesterase in plasma, brain, and red blood cells, but no clinical signs of nervous system toxicity at 10 mg/kg bw/day.

Three-month dietary studies in rats and dogs reported decreased bodyweight gain at 15 mg/kg bw/day (rats), and increased serum alkaline phosphatase without associated organ histopathology (dogs), at doses of 15 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies in mice, rats and dogs reported decreased bodyweight gains (all species), hyperactivity and decreased plasma cholinesterase (rats and dogs), and tremors, decreased red blood cell counts, increased absolute brain weights, and inhibition of brain cholinesterase activity (dogs) at 1.3 mg/kg bw/day and above. The lowest overall NOEL was 0.9 mg/kg bw/day (dogs). No other effects were seen in rats and mice.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for oxamyl.

Genotoxicity: Oxamyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In reproduction studies in rats, there was no evidence of effects on reproductive parameters. Developmental studies in rats reported reduced foetal bodyweight at 0.5 mg/kg bw/day and above, and maternotoxicity at 0.8 mg/kg bw/day. The NOEL was 0.2 mg/kg bw/day and is the basis for the current ADI.

Poisons Schedule: Oxamyl is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for oxamyl was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a developmental study in rats using gavage administration.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for oxamyl and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2007). Interim Reregistration Eligibility Decision. Oxamyl – List A, case 0253. USEPA.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Paraquat

(endorsed 2011)

GUIDELINE

Based on human health concerns, paraquat in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Paraquat (CAS 4685-14-7 (paraquat); CAS 1910-42-5 (paraquat dichloride)) belongs to the bipyridinium class of compounds, which also includes the herbicide diquat (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, paraquat would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on an end-point that is common to both short-term and long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Paraquat is a non-selective herbicide for the control of grasses and broad-leaf weeds in agricultural and horticultural crops.

There are registered products that contain paraquat (mainly as the dichloride) in Australia. These products are intended for professional use and are available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to paraquat is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of paraquat may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data for paraquat in Australian water could be found. Paraquat dichloride binds so strongly to soil clay particles that it is extremely unlikely to be found in groundwater supplied as a drinking water source (Health Canada 1986, USEPA 1997).

TREATMENT OF DRINKING WATER

Techniques effective in removing paraquat from water include adsorption by charcoal, ion exchange (66% and 70%) and modified peat (95% to 99%). Chlorine dioxide has been found to oxidize paraquat concentrations of 15 and 30 mg/L within minutes above pH 8.7. The use of bentonite, a clay adsorbent, for 10 minutes, followed by a 15-minute coagulation period, resulted in 90% removal of paraquat present at 1 mg/L (Health Canada, 1986).

MEASUREMENT

Paraquat can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 1 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for paraquat is 0.004 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.45 mg/kg bw/day from a one-year dietary study in the dog. The NOEL is based on lung lesions at 0.93 to 1.0 mg/kg bw/day. The ADI incorporates a safety factor of 100 and was established in 1992.

The acute reference dose (ARfD) of 0.004 mg/kg bw/day for paraquat is based on the same NOEL as the ADI (0.45 mg/kg bw/day from a one-year dietary study in the dog). The NOEL is based on lung lesions at the next highest dose, 0.93 to 1.0 mg/kg bw/day. These lung lesions were evident also for shorter exposures. The ARfD incorporates a safety factor of 100, and was established in 2003.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Paraquat is poorly absorbed via the gastrointestinal tract, and is not extensively metabolised. Within 72 hours after administration, up to 80% appears unchanged in the faeces, up to 20% in urine, and less than 1% remains in the body. The small amount of urinary metabolites has not been identified. At 72 hours, there is no evidence of accumulation in most tissues, although paraquat may persist and accumulate in mouse brain.

Acute effects: Paraquat has moderate acute oral toxicity and low acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 3-6 week dietary studies in mice, rats, rabbits and dogs, pathological changes in the lung, gastrointestinal tract and kidneys at dose levels above 2-4 mg/kg bw/day were reported. Significantly decreased food consumption and decreased bodyweight gain, and a dose-dependent increase in the mortality rate were also common observations. In a 13-week dietary study in dogs, there were macroscopic lung lesions and histopathological signs of alveolitis from 1.5 mg/kg bw/day. Alveolitis was detected in all dogs at 3 mg/kg bw/day.

In a one-year dietary study in dogs, there was an increased incidence of pulmonary lesions associated with chronic pneumonitis from 0.9 mg/kg bw/day. The NOEL was 0.45 mg/kg bw/day and this is the basis for the ADI and ARfD.

Long-term effects: A 2-year dietary study in mice reported clinical signs of toxicity at 1.9 mg/kg bw/day and an increased incidence of pulmonary adenomas at 15 mg/kg bw/day. A 2-year dietary study in rats reported cataracts and lenticular abnormalities of the eye from 2.5 mg/kg bw/day. At higher dose levels, lesions in the lungs included focal subpleural abnormalities, chronic pneumonitis, and proliferative lesions in the alveolar epithelium. Other effects included hydrocephalus, degeneration of the nerve fibres

NOTE: Important general information is contained in PART II, Chapter 6

of the sciatic nerve and an increase in the numbers of cysts or cystic spaces in the spinal cord.

Carcinogenicity: There was evidence of proliferative lesions in the alveolar epithelium in a 2-year rat study, but only at dose levels well in excess of the likely level of human exposure. Long-term studies in mice showed no evidence of carcinogenicity.

Genotoxicity: Paraquat was clastogenic (causes chromosome breaks) in some *in vitro* short-term assays, but there was no evidence of genotoxicity in *in vivo* studies.

Reproductive and developmental effects: Two and three-generation dietary reproduction studies in rats and developmental toxicity studies in mice, rats and rabbits showed no evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: In studies in mice and rats, paraquat produced evidence of neurodegeneration and induced changes in the brain similar to Parkinson's disease. There is equivocal epidemiological evidence in some countries linking pesticide usage and the incidence of Parkinson's disease.

Poisons Schedule: Paraquat is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). The Standard also indicates that aqueous solutions of paraquat must be coloured blue or green, and must contain a stenching agent. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for paraquat was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.45 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.45 mg/kg bw/day is the NOEL based on a long-term (1-year) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1986). Paraquat. Factsheet for the development of the Canadian Guidelines for Drinking Water Quality. Environmental and Workplace Health, Health Canada.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NOTE: Important general information is contained in PART II, Chapter 6

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1997). Reregistration eligibility decision (RED) – Paraquat dichloride. EPA 738-F-96-018. USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Parathion

(endorsed 2011)

GUIDELINE

Based on human health concerns, parathion in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Parathion (ethyl parathion)(CAS 298-00-0) belongs to the organophosphate class of chemicals. Other pesticides in this class include methyl parathion, disulfoton, ethion, and chlorpyrifos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, parathion would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Parathion is an insecticide and acaricide (miticide) for the control of aphids, mites and caterpillars in commercial vegetable, fruit, tobacco and cotton crops.

There are currently no registered products containing parathion in Australia, but de-registered compounds could still be detected in water. Previously registered products were intended for professional use; these products were soluble concentrates intended to be diluted and applied by aerial and ground spray.

Exposure sources: If used in the future, the main source of public exposure to parathion and its metabolites, based on the previous use pattern, would be through residues in food. Residue levels in food produced according to good agricultural practice are generally low.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Parathion is typically not detected in Australian drinking waters and is not reported in drinking waters internationally under conditions of good agricultural practice. Its presence in drinking waters has not been reported by the World Health Organization (WHO 2004a).

TREATMENT OF DRINKING WATER

No specific data on the treatment of parathion in drinking water have been identified.

MEASUREMENT

Parathion is effectively extracted from water using liquid-liquid extraction with dichloromethane followed by gas chromatographic analysis employing nitrogen-phosphorus detection. The typical limit of detection using this procedure is 10 ng/L (Culea and Gocan 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for parathion is 0.005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.05 mg/kg bw/day for a reduction in erythrocyte cholinesterase activity in a 3-week oral toxicity study in humans. The ADI incorporates a safety factor of 10. It was first set in 1967 and reaffirmed in 1997.

The acute reference dose (ARfD) of 0.01 mg/kg bw for parathion was established in 2000, based on a NOEL of 0.125 mg/kg bw/day for erythrocyte cholinesterase inhibition from a 35-day oral toxicity study in humans. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Parathion is readily and extensively absorbed via the gastrointestinal tract. It is extensively metabolised, and is rapidly excreted in the urine and faeces, almost completely within 48 hours. The main metabolite is paraoxon-ethyl, which is of similar acute toxicity to the parent compound.

Acute effects: Parathion has high acute oral and dermal toxicity in animals and humans. The potential for skin sensitisation is unknown, due to its high dermal toxicity. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: An dietary study in humans for up to 21 days reported reduced erythrocyte cholinesterase activity at the highest dose tested, 0.1 mg/kg bw/day. The NOEL was 0.05 mg/kg bw/day and this NOEL is the basis for the current ADI.

Long-term effects: Long-term dietary studies in rats and dogs reported reduced plasma and erythrocyte cholinesterase activity at doses of 0.03 mg/kg/day and above. In rats, reduced brain cholinesterase activity, retinal atrophy, and myelin degeneration in optical nerves were seen at the highest dose tested, 1.9 mg/kg bw/day.

Carcinogenicity: The evidence from several long-term rodent studies is inadequate to assess carcinogenicity for parathion.

Genotoxicity: Parathion is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two-generation reproduction studies in rats up to 2.8 mg/kg bw/day and developmental studies in rats and rabbits up to 1.5 mg/kg bw/day found no evidence for effects on reproduction or foetal development.

Neurotoxicity: In a 10-day study in hens, there was no evidence of delayed neurotoxicity due to parathion.

Poisons Schedule: Parathion is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for parathion was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.05 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.05 mg/kg bw/day is the NOEL based on a short-term (3-week) dietary study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from human studies. This safety factor covers intraspecies variation.

The World Health Organization has not established a health-based guideline value for parathion and it is excluded from the list of agricultural chemicals guideline value derivation because it "occurs in drinking-water at concentrations well below those at which toxic effects may occur" (WHO 2004b).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Culea M, Gocan S (2000). Organophosphates. In: Nollet LM, Dekker M (eds). *Handbook of Water Analysis*, New York, pp571-608.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004a). Parathion in Drinking-water; Background Document for Development of WHO Guidelines for Drinking Water.

WHO (World Health Organization) (2004b). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

Parathion-methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, parathion-methyl in drinking water should not exceed 0.0007 mg/L.

RELATED CHEMICALS

Parathion-methyl (CAS 298-00-0) belongs to the organophosphate class of pesticides, and is structurally related to parathion. This is one of the largest classes of pesticides, and other members include acephate, chlorpyrifos, ethion, phorate, and terbufos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, parathion-methyl would not be a health concern unless the concentration exceeded 0.0007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Parathion-methyl is an insecticide for the control of pests in agricultural crops.

There are registered products that contain parathion-methyl in Australia. The products are intended for professional use on vegetables, citrus, pome and stone fruit, grapevines, and cotton crops. Products are capsule suspensions or liquid concentrates that are applied in diluted form to crops by ground boom or aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to parathion-methyl and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of parathion-methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of parathion-methyl in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Parathion methyl is completely removed from water by chlorination when the chlorine dose is adjusted to match chlorine demand (Ormad *et al.* 2008).

Ozonation and activated carbon adsorption for parathion-methyl removal has also been reported with moderate success (Ormad *et al.* 2008). Conventional coagulation/flocculation provides a relatively low removal rate (30%). Jar testing is recommended to optimise dose rates if parathion-methyl is detected.

MEASUREMENT

Parathion-methyl can be measured in drinking water by gas chromatography–mass spectrometry. The typical limit of detection is 0.1 µg/L (QFSS 2009).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for parathion-methyl is 0.0002 mg per kg of bodyweight (mg/kg bw), based on a NOEL of 0.02 mg/kg bw/day from long-term (1-year and 2-year) dietary studies in rats. The NOEL is based on time- and dose-dependent neuro-pathological effects in the form of neuronal degeneration and abnormal gait. The ADI incorporates a safety factor of 100, and was established in 1997.

The previous ADI was 0.03 mg/kg bw/day. It was established in 1990 based on a NOEL of 0.3 mg/kg bw/day for erythrocyte cholinesterase inhibition in a 30-day dietary study in humans. The ADI was amended in 1997 to its current level after submission of long-term studies in rats showing effects at levels below this NOEL.

The ARfD of 0.03 mg/kg bw for parathion-methyl was established in 2000, based on the aforementioned NOEL of 0.3 mg/kg bw/day for erythrocyte cholinesterase inhibition in a 30-day dietary study in humans. The ARfD incorporates a safety factor of 10.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Parathion-methyl is readily and extensively absorbed from the gastrointestinal tract. Metabolism was extensive and proceeded by sulfonation, conjugation, and oxidation. The metabolites are of similar or lower toxicity to parathion-methyl. Excretion of parent compound and metabolites is rapid, proceeding through urine and being almost complete by 24 hours.

Acute effects: Parathion-methyl has high acute oral and dermal toxicity in rats. Parathion-methyl is not a skin sensitiser in guinea pigs. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: A five-day dermal study in rats reported brain cholinesterase inhibition at 1 mg/kg bw/day and above. Clinical symptoms were observed at higher dose levels. A 28-day dermal study in rats reported brain and erythrocyte cholinesterase inhibition at 0.3 mg/kg bw/day and above. A 13-week dietary study in rats reported erythrocyte cholinesterase inhibition at 0.23 mg/kg bw/day and brain cholinesterase inhibition at higher doses. There were no associated histopathological changes. The NOEL in this study was 0.02 mg/kg bw/day.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: Long-term dietary studies were undertaken in rats, dogs, and mice. Effects were typical of organophosphates and in rats included neuronal degeneration and lethargy at doses of 0.1 mg/kg bw/day and above. In mice and rats, effects included behavioural changes and brain and erythrocyte cholinesterase inhibition at doses of 0.5 mg/kg bw/day. Brain demyelination also was seen in rats by 6 months at doses of 2 mg/kg bw/day. In dogs, erythrocyte cholinesterase was inhibited at the lowest dose tested, 0.03 mg/kg bw/day.

The lowest overall NOEL was 0.02 mg/kg bw/day based on neuronal degeneration and decreased physical activity in the rat. This NOEL is the basis for the current ADI.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for parathion-methyl.

Genotoxicity: Parathion-methyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats reported reduced pup survival during weaning at 1 mg/kg bw/day, without evidence of adverse effects in dams. Developmental studies in rats and rabbits did not identify any effects on foetal development.

Neurotoxicity: Short-term dietary studies in rats, mice and dogs reported symptoms indicative of nervous system toxicity but only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Parathion-methyl is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.0007 mg/L for parathion-methyl was determined as follows:

$$0.0007 \text{ mg/L} = \frac{0.02 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.02 mg/kg bw/day is the NOEL based on long-term (1-year and 2-year) dietary studies in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for parathion-methyl based on the shown evidence that the chemical "occurs in drinking-water at concentrations well below those at which toxic effects may occur" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71: 97–106.

QFSS (Queensland Forensic and Scientific Services) (2009). Personal communication

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Pebulate

(endorsed 2011)

GUIDELINE

Based on human health concerns, pebulate in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

Pebulate (CAS 1114-71-2) belongs to the thiocarbamate class of chemicals. Other pesticides in this class include molinate, butylate, and EPTC (ethyl dipropylthiocarbamate) (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pebulate would not be a health concern unless the concentration exceeded 0.03 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pebulate is a selective herbicide for the control of grassy weeds in tobacco and tomato crops.

There is at least one registered product containing pebulate in Australia. Pebulate products are intended for professional use and are available as a concentrated solution to be applied in diluted form by roller application to foliage, and by boomspray to soil. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to pebulate and its metabolites are residues in food and in tobacco used for cigarettes and cigars. Residue levels in food and tobacco produced according to good agricultural practice are generally low.

Agricultural use of pebulate may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on pebulate occurrence in Australian drinking water supplies were found.

TREATMENT OF DRINKING WATER

No specific data on the treatment of pebulate in drinking water have been identified.

MEASUREMENT

United States Environmental Protection Agency method 525.2 (USEPA 1995) for the determination of organic compounds in drinking water by liquid-solid extraction and capillary column gas chromatography–mass spectrometry (GC-MS) can achieve a limit of quantitation (LOQ) of 0.08 µg/L to 0.11 µg/L for pebulate. Pebulate can be extracted from water by liquid/liquid extraction with dichloromethane and analysed by GC-MS in selected ion monitoring mode, with a LOQ of 0.5 µg/L. USEPA method 634 for the determination of thiocarbamate pesticides in industrial and municipal wastewaters by gas chromatography is also approved for the analysis of pebulate in water (USEPA 1993). USEPA method 507 for the determination of nitrogen and phosphorus containing pesticides in water by gas chromatograpy (GC) with a nitrogen-phosphorus detector can achieve a LOQ of 0.05 µg/L (Munch 1995). Solid-phase microextraction (SPME) followed by gas–liquid chromatography employing a nitrogen-phosphorus detector can achieve a LOQ of 0.04 µg/L, and SPME-GC employing mass spectrometry can achieve a LOQ of 0.01 µg/L (Choudhury *et al.* 1996).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pebulate is 0.007 mg per kg of bodyweight (mg/kg bw), based on no-observed-effect levels (NOELs) of 0.7 and 0.75 mg/kg bw/day from a long-term dietary study and a reproduction study in rats, respectively. The NOELs are based on decreased bodyweight and slight lens disjunction in eyes at 7 mg/kg bw/day in the long-term study, and decreased bodyweight in parents and offspring at 7.5 mg/kg bw/day in the reproduction study. The ADI incorporates a safety factor of 100, and was established in 1990.

The previous ADI of 0.01 mg/kg bw was set in 1982.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pebulate was rapidly absorbed and extensively metabolised in oral dosing studies in rats. Excretion was mainly through expired air and to a lesser extent by urine and faeces, and was complete by 24 hours. The major metabolites were mercapturic acids.

Acute effects: Pebulate has low acute oral and dermal toxicity. Pebulate is not a skin sensitiser.

Short-term effects: In medium-term dietary studies in rats and dogs, there was reduced cholinesterase activity in brain and red blood cells, as well as reduced red blood cell count, at doses of 16 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in rats (2-years) and dogs (1-year). In rats, there was decreased bodyweight and slight lens disjunction in the eyes at doses of 7 mg/kg bw/day. In dogs, minor decreases in bodyweight and increases in serum platelet count were seen at doses of 50 mg/kg bw/day. The overall NOEL was 0.7 mg/kg bw/day in the rat study, which is partly the basis for the ADI.

Carcinogenicity: Based on a 2-year study in mice and rats, there is no evidence of carcinogenicity for pebulate.

Genotoxicity: Only short-term *in vitro* studies are available; these report no evidence that pebulate is genotoxic.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A two-generation study in rats found no effects on reproductive parameters. Decreased bodyweights in parents and offspring were seen at doses of 7.5 mg/kg bw/day. The NOEL was 0.75 mg/kg bw/day, which is partly the basis for the ADI. Development studies in rats and rabbits found no effects on foetal development.

Poisons Schedule: Pebulate is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for pebulate was determined as follows:

$$0.03 \text{ mg/L} = \frac{0.7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.7 mg/kg bw/day is the NOEL based on a 2-generation studies and long-term (2-year) dietary studies in rats. The effects at higher levels were decreased bodyweights and lens disjunction.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Choudhury TK, Gerhardt KO, Mawhinney TP (1996). Solid-phase microextraction of nitrogen- and phosphorus-containing pesticides from water and gas chromatographic analysis. *Environmental Science and Technology,* 30(11):3259–3265.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Munch JW (1995). *Method 507 Determination of nitrogen and phosphorus containing pesticides in water by gas chromatography with a nitrogen phosphorus detector*. National Exposure Research Laboratory, Office of Research and Development, United States Environmental Protection Agency, Cincinnati, Ohio.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1993). *Method 634. Methods for the Determination of Nonconventional Pesticides in Municipal and Industrial Wastewater.* USEPA United States Environmental Protection Agency, Washington DC,

NOTE: Important general information is contained in PART II, Chapter 6

USEPA (United States Environmental Protection Agency) (1995). *Method 525.2 Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and capillarity Columns Gas Chromatography/ Mass Spectrometry Revision 2.0*. Environmental Monitoring Systems Laboratory Office of Research and Development, USEPA, Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Pendimethalin

(endorsed 2011)

GUIDELINE

Based on human health concerns, pendimethalin in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Pendimethalin (CAS 40487-42-1) belongs to the dinitroaniline class of chemicals. Other pesticides in this class include trifluralin and oryzalin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pendimethalin would not be a health concern unless the concentration exceeded 0.4 mg/L. While the health-based guideline is based on long-term effects, there is evidence for maternotoxicity after short-term exposure at levels near the no-observed-effect level (NOEL) used to derive the health-based guideline. Therefore, excursions above the health-based guideline even for a short period are of concern.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pendimethalin is a selective herbicide for the control of broad-leaf weeds and annual grasses in soybean, cotton, wheat, barley, vegetable and turf crops.

There are registered products that contain pendimethalin in Australia. Pendimethalin products are intended for professional use. They are in emulsifiable concentrate or granular formulations, and are applied by aerial spray or ground spray to crops or soil, or mixed with irrigation water for use on fields. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to pendimethalin and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of pendimethalin may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of pendimethalin in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of pendimethalin in drinking water have been identified.

MEASUREMENT

Pendimethalin can be determined in drinking waters by gas chromatography-mass spectrometry. The typical limit of detection is 0.01 µg/L (WHO 2004).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pendimethalin is 0.1 mg per kg of bodyweight (mg/kg bw), based on a NOEL of 12.5 mg/kg bw/day from a long-term (2-year) dietary study in dogs. The NOEL is based on decreased bodyweight gain and evidence of mild effects on the liver. The ADI incorporates a safety factor of 100, and was first established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pendimethalin is poorly absorbed in the gastrointestinal tract, with most excreted unchanged in the faeces and the remainder being extensively metabolised and rapidly excreted in urine. Excretion is complete by 96 hours.

Acute effects: Pendimethalin has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pigs.

Short-term effects: In 30-day dietary studies in rats and dogs, decreased bodyweight gain, and increased absolute liver weights and blood glucose levels were seen in rats at doses of 320 mg/kg bw/day and above. Toxic effects were not seen in dogs even up to high exposure levels of 1000 mg/kg bw/day.

In a 3-month dietary study with pendimethalin in dogs, the only effect was a small decrease in bodyweight gain at the highest dose tested, 1000 mg/kg bw/day.

Long-term effects: In long-term dietary studies in mice, rats and dogs, decreased bodyweight gain and mild liver effects (increased serum alkaline phosphatase, mild inflammation, biliary hyperplasia, hepatic haemosiderosis) were seen in all three species at various doses. The lowest overall NOEL was 12.5 mg/kg/day from a 2-year dietary study in dogs. This NOEL is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for pendimethalin.

Genotoxicity: Pendimethalin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In 2- and 3-generation studies in rats, and in developmental studies in rats and rabbits, there was no evidence of effects on reproductive parameters or on foetal development. However, maternotoxicity in these studies at 30 mg/kg bw/day is close to the NOEL used as a basis for the ADI.

Poisons Schedule: Pendimethalin is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for pendimethalin was determined as follows:

$$0.4 \text{ mg/L} = \frac{12.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 12.5 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a health-based guideline value of 0.02 mg/L for pendimethalin (WHO 2004). This was based on an ADI of 5 mg/kg bw and an uncertainty factor of 1000 (100 for inter- and intra-species variation and 10 for a combination of the use of a lowest-observed-adverse-effect level instead of a no-observed-adverse-effect level and limitations of the database).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Pentachlorophenol

(endorsed 2011)

GUIDELINE

Based on human health concerns, pentachlorophenol in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Pentachlorophenol (CAS 87-86-5) belongs to the chlorinated hydrocarbon class of chemicals. Other pesticides in this class include endosulfan (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pentachlorophenol would not be a health concern unless the concentration exceeded 0.01 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pentachlorophenol is an insecticide and fungicide used formerly as a wood preservative. It has also been used formerly as a pre-harvest defoliant, pre-emergence herbicide and an aquatic biocide.

There are no registered products containing pentachlorophenol in Australia, but de-registered compounds may still be detected in water. When used previously, products containing pentachlorophenol or its salt variant were intended for professional use only.

Exposure sources: If used in the future, the main source of public exposure to pentachlorophenol and its metabolites would be through residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of pentachlorophenol, in the future, may potentially lead to contamination of source waters through processes such as leaching from treated wood, run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data in Australian water could be found. Although pentachlorophenol has been banned in most countries, it can still often be found in surface and groundwater at low µg/L concentrations and in drinking water usually in the range 0.01-0.1 µg/L (Health Canada 1987, WHO 2003).

TREATMENT OF DRINKING WATER

Available data indicate that concentrations of chlorophenols are not reduced significantly during conventional drinking water treatment processes. Although few relevant data are available, it is likely that concentrations below 1 µg/L can commonly be achieved by packed tower aeration and granular activated carbon adsorption (Health Canada 1987).

MEASUREMENT

Pentachlorophenol can be measured by routine gas chromatography mass spectrometry analysis, with a limit of reporting of 0.25 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

No acceptable daily intake (ADI) or acute reference dose (ARfD) values have been established for pentachlorphenol in Australia.

An ADI for pentachlorophenol was set at 0.003 mg per kg of bodyweight (mg/kg bw) by the United States National Academy of Sciences. This ADI was based on a no-observed-effect level (NOEL) of 3 mg/kg bw/day from a long-term (2-year) dietary study in rats. The NOEL is based on decreased bodyweight, liver effects and liver tumours. The ADI incorporates a safety factor of 1000 and was established in 1977.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pentachlorophenol is readily absorbed via the gastrointestinal tract and through the skin in animals and humans. After exposure, the highest tissue levels were found in the liver and kidneys. Pentachlorophenol is excreted rapidly, either unchanged or as the metabolites, tetrachlorhydroquinone and glucuronides. The half-life for elimination in humans is 17 days.

Acute effects: Pentachlorophenol has moderate to high acute oral toxicity and moderate dermal toxicity. The skin sensitisation potential of pentachlorophenol is unknown.

Short-term effects: In short-term dietary studies in mice, rats and pigs, the main effects observed were on the liver in the form of increased relative liver weight and increased enzyme activity (particularly aryl hydrocarbon hydroxylase), together with histopathological changes. In mice, these effects, as well as increased mortality, were observed from 30 mg/kg bw/day. In pigs, liver weight changes were observed at 10 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice and rats. The liver was the main organ affected, with changes in enzyme levels and liver weight commonly noted at the lower doses, and histopathological changes and in some cases tumour formation at higher dose levels. In mice, decreased bodyweight, liver lesions, hepatocellular adenomas and carcinomas and benign adrenal medulla pheochromocytomas were observed at the lowest dose, 15 mg/kg bw/day. Female mice also showed haemangiosarcomas of the spleen and liver at 30-45 mg/kg bw/day. In rats, reduced bodyweight gain and pigment accumulation in the liver were observed from 10 mg/kg bw/day.

Carcinogenicity: Studies in mice showed an increased incidence of tumours in the spleen, liver and adrenal gland from 15 mg/kg bw/day. This dose level is well in excess of the likely level of pentachlorophenol in drinking water.

Genotoxicity: Pentachlorophenol is not considered to be genotoxic, based on *in vitro* and *in vivo* short-

term studies, despite some weak evidence for chromosomal effects.

Reproductive and developmental effects: In a one-generation reproduction study in rats, there was evidence of foetotoxicity at high dose levels (25 mg/kg bw/day) only.

In a developmental toxicity study in rats, delayed foetal development was observed at 4 mg/kg bw/day, and embryonic death and maternotoxicity were seen at doses of 43 mg/kg bw/day.

Poisons Schedule: Pentachlorophenol is included in Schedule 6 and 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for pentachlorophenol was determined as follows:

$$0.01 \text{ mg/L} = \frac{3 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 3 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation, with an additional safety factor of 10 due to the limitation of the toxicological data available at the time the ADI was set.

The World Health Organization has a health-based guideline value of 0.009 mg/L for pentachlorophenol (WHO 2004). This guideline value is considered provisional because of the variations in metabolism between experimental animals and humans.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1987). Chlorophenols. Background document for the development of the Guidelines for Canadian Drinking Water Quality. Feb 1987. Health Canada.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

WHO (World Health Organization) (2003). Pentachlorophenol in drinking water: Background document for the development of the WHO Guidelines for Drinking Water Quality. WHO.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Permethrin

(endorsed 2011)

GUIDELINE

Based on human health concerns, permethrin in drinking water should not exceed 0.2 mg/L.

RELATED CHEMICALS

Permethrin (CAS 52645-53-1) is in the pyrethroid class of chemicals. Other pesticides in this class include cyfluthrin, cypermethrin, alpha-cypermethrin and deltamethrin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, permethrin would not be a health concern unless the concentration exceeded 0.2 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Permethrin is an insecticide used for the control of mosquitoes and flies in domestic and agricultural settings, seed infestation in food industry settings, and fleas on dogs.

There are many registered products containing permethrin in Australia. The products are intended for both professional and home garden use. Use patterns include sprays and drenches for livestock in agricultural settings, paint formulations on timber as a preservative in industrial settings, shampoos for dogs, sprays for insects in domestic settings, and sprays for application directly to clothing. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to permethrin are home garden use as an insecticide, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on the concentrations of permethrin in Australian drinking waters or in drinking water overseas.

TREATMENT OF DRINKING WATER

There are no identified reports of the treatment of permethrin in drinking water.

MEASUREMENT

Several methods have been reported for the analysis of permethrin in water, including gas chromatography with micro-electron capture detection (limit of detection [LOD] 2.18 ng/L, Casas *et al*. 2006; LOD 3 ng/L, Mekebri *et al*. 2008), liquid chromatography with electrospray ionisation mass spectrometry (LOD 0.4 ng/L in groundwater and 0.7 ng/L in sea water) (Gil-Garcia *et al*, 2006), and gas chromatography–high resolution mass spectrometry (LOD 0.15ng/L) (Woudneh and Oras 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for permethrin is 0.05 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day from a 2-year dietary study in rats and a 1-year oral dosing study in dogs. This NOEL is based on neurotoxic effects including tremors, incoordination and convulsions. The ADI incorporates a safety factor of 100 and was established in 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Permethrin is rapidly absorbed from the gastrointestinal tract and widely distributed in the body. It is rapidly eliminated, mainly in the urine and faeces as polar metabolites in the form of glucuronide conjugates, benzyl alcohols, and weak acids. Permethrin has a low potential for bioaccumulation. The primary metabolites are *m*-phenoxybenzyl alcohol and *m*-phenyxybenzoic acids.

Acute effects: Permethrin has low acute oral and dermal toxicity. Symptoms of acute poisoning were indicative of nervous system poisoning and included tremors, hyperexcitability, salivation and paralysis. It is not a skin sensitiser.

Short-term effects: A 10-day dietary study in rats produced clinical symptoms indicative of an effect on the nervous system (impaired muscle relaxation) at 30 mg/kg bw/day. Recovery from these effects was observed one week after dosing. Medium-term studies were conducted in rats and dogs. Rats were most sensitive to permethrin, with liver centrilobular hypertrophy observed in males at 7.5 mg/kg bw/day. Dogs reported increased liver weight from 50 mg/kg bw/day and decreased bodyweight at 500 mg/kg bw/day.

Long-term effects: A long-term (2-year) dietary study with permethrin in rats, and a 1-year oral dosing study in dogs showed the main effects to be on the liver, and central and peripheral nervous systems. Symptoms included increased serum glucose levels and absolute liver weight at the highest dose tested of 25 mg/kg bw/day (rats), and tremors, incoordination and convulsions at doses of 100 mg/kg bw/day and above (dogs). The NOEL in both studies was the next lowest dose levels tested, 5 mg/kg bw/day, and this is the basis for the current ADI.

Carcinogenicity: Based on long-term studies in rats, there is no evidence of carcinogenicity for permethrin.

Genotoxicity: Permethrin is not considered to be genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproductive and developmental effects: In a 3-generation reproduction study in rats and developmental toxicity studies in rats and rabbits, there was no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Permethrin is either exempt from scheduling or in Schedule 4, 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.2 mg/L for permethrin was determined as follows:

$$0.2 \text{ mg/L} = \frac{5.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 5.0 mg/kg bw/day is the NOEL based on a long-term (1-year) oral dosing study in dogs and a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has established a guideline value of 0.3 mg/L for permethrin (WHO 2006).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Casas V, Llompart M, Garcia-Jares C, Cela R, Dagnac T (2006). Multivariate optimisation of the factors influencing the solid-phase microextraction of pyrethroid pesticides in water. *Journal of Chromatography A*, 1124:148-156.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Gil-Garcia MD, Barranco-Martinez D, Martinez-Galera M, Parrilla-Vazquez P (2006). Simple, rapid solid-phase extraction procedure for the determination of ultra-trace levels of pyrethroids in ground and sea water by liquid chromatography/electrospray ionisation mass spectrometry. *Rapid Communications in Mass Spectrometry*, 20:2395-2403.

Mekebri A, Crane DB, Blondina GJ, Oros DR, Rocca JL (2008). Extraction and analysis methods for the determination of pyrethroid insecticides in surface water, sediments and biological tissues at environmentally relevant concentrations. *Bulletin of Environmnetal Contamination and Toxicology*, 80:455-460.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, First addendum WHO, Geneva, Switzerland.

Woudneh MB, Oros DR (2006). Quantitative determination of pyrethroids, pyrethrins and piperonyl butoxide in surface water by high-resolution gas chromatography/high resolution mass spectrometry. *Journal of Agricultural and Food Chemistry*, 54(19):6957-6962.

NOTE: Important general information is contained in PART II, Chapter 6

pH

(endorsed 1996)

GUIDELINE

Based on the need to reduce corrosion and encrustation in pipes and fittings, the pH of drinking water should be between 6.5 and 8.5.

New concrete tanks and cement-mortar lined pipes can significantly increase pH and a value up to 9.2 may be tolerated, provided monitoring indicates no deterioration in microbiological quality.

GENERAL DESCRIPTION

pH is a measure of the hydrogen ion concentration of water. It is measured on a logarithmic scale from 0 to 14. A pH of 7 is neutral, greater than 7 is alkaline, and less than 7 is acidic.

One of the major objectives in controlling pH is to minimise corrosion and encrustation in pipes and fittings. Corrosion can be reduced by the formation of a protective layer of calcium carbonate on the inside of the pipe or fitting, and the formation of this layer is affected by pH, temperature, the availability of calcium (hardness) and carbon dioxide. If the water is too alkaline (above pH 8.5), the rapid deposition and build-up of calcium carbonate that can result may eventually block the pipe.

When pH is below 6.5 or above 11, the water may corrode plumbing fittings and pipes. This, however, will depend on other factors such as the material used, the concentration and type of ions in solution, the availability of oxygen, and the water temperature. Under some conditions, particularly in the presence of strong oxidising agents such as chlorine, water with a pH between 6.5 and 7 can be quite corrosive.

Chlorine disinfection efficiency is impaired above pH 8.0, although the optimum pH for monochloramine disinfectant formation is between 8.0 and 8.4. In chloraminated supplies chlorine can react with ammonia to form odorous nitrogen trichloride below pH 7.

Chlorination of water supplies can decrease the pH, while it can be significantly raised by lime leached from new concrete tanks or from pipes lined with asbestos cement or cement mortar. Values of pH above 9.5 can cause a bitter taste in drinking water, and can irritate skin if the water is used for ablutions.

MEASUREMENT

pH can be determined potentiometrically using a standard glass electrode and reference (APHA Method 4500-H^+ 1992).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies pH ranges between 6 and 10.8.

CONTROL IN DRINKING WATER SUPPLIES

The pH of water can be adjusted by the addition of acid or alkali. Usually lime, soda ash, sodium hydroxide, or a combination of lime and carbon dioxide are used (AWWA 1990).

HEALTH CONSIDERATIONS

A direct relationship between pH and human health is difficult to determine, as pH is closely associated with other aspects of water quality. Consumption of food and beverages with quite low or high pH is common and does not result in adverse health effects. Some carbonated soft drinks, for instance, have a

pH of 2.5, orange fruit juice has a pH of about 3.8, and the pH of milk is 6.7.

In humans, extreme values of pH result in irritation of the eyes, skin and mucous membranes. Eye irritation and exacerbation of skin disorders have been associated with pH values above 11. Gastrointestinal irritation may occur in sensitive individuals at pH values above 10. Below pH 4, redness and irritation of the eyes have been reported, with the severity increasing with decreasing pH. Below pH 2.5, damage to the epithelium is irreversible and extensive.

pH may have an indirect effect on bacteriological quality through its effects on disinfection processes. It can affect the solubility of heavy metals, particularly lead and copper from pipes, and the formation of trihalomethanes (see Section 6.3.2 in Chapter 6 on disinfection by-products) (USEPA 1989).

In studies using animals, solutions of differing pH have been injected into the abdominal skin of mice, resulting in irritation at pH 10 after 6 hours. In rabbits, eye irritation was reported at pH 10 but not at pH 4.5.

Chromosome aberrations and gene mutations have been reported in cultured mammalian and invertebrate cells using different acids between pH 4 and 6.5.

The effect of pH on health will depend on the buffering capacity of the water used. This is related to the nature and amount of dissolved inorganic and organic material. Water with a low buffering capacity can change pH rapidly, but water with a high buffering capacity is resistant to pH change. Extreme values of pH in association with highly buffered water are of greater concern than when the water has a low buffering capacity.

DERIVATION OF GUIDELINE

The guideline value is based on minimising corrosion and encrustation of plumbing fittings and pipes. Water with a pH between 6.5 and 8.5 should deposit a protective coating of calcium carbonate and prevent corrosion. High pH can cause scaling and encrustation problems, while lower pH can result in corrosion.

New concrete tanks and cement–mortar lined pipes can significantly increase pH and a value up to 9.2 may be tolerated, provided microbiological monitoring indicates no deterioration in bacteriological quality.

Insufficient data are available to set a health-based guideline value for pH.

GUIDELINES IN OTHER COUNTRIES

The Canadian Guidelines, United States Regulations, European Economic Community Standards, and 1984 World Health Organization (WHO) Guidelines all recommend a pH range of 6.5 to 8.5. The 2006 WHO Guidelines do not provide a specific range of pH values.

REFERENCES

APHA Method 4500-H^+ (1992). pH value: B. Electrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

AWWA (American Water Works Association) (1990). *Water Quality and Treatment: A handbook of community water supplies*. AWWA, 4th edition, McGraw-Hill Inc.

USEPA (United States Environmental Protection Agency) (1989). Health effects of drinking water treatment technologies. USEPA, Lewis Publ. Inc.

WHO (World Health Organization) (2006). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Picloram

(endorsed 2011)

GUIDELINE

Based on human health concerns, picloram in drinking water should not exceed 0.3 mg/L.

RELATED CHEMICALS

Picloram (CAS 2545-60-0) belongs to the pyridinecarboxylic acid class of chemicals. Other pesticides in this class include clopyralid, fluroxypyr and triclopyr (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, picloram would not be a health concern unless the concentration exceeded 0.3 mg/L. Excursions above this level over a short to medium period are of concern, as the health-based guideline is based on effects observed in a 3-month study.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Picloram is a post-emergent herbicide for the control of woody and herbaceous weeds and rhizomatous plants in cereal crops, conservation areas, reserves and parks, and in home gardens.

There are registered products that contain picloram as an ester or a salt in Australia. The products are intended for professional and home garden use. They are generally available as emulsifiable concentrates that are applied by aerial and ground spray, or by direct brushing onto foliage or pouring into the trunks of herbaceous and woody weeds. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to picloram and its metabolites are the use of home garden products, contact with treated weeds in parklands, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The use of picloram in parkland areas and agriculture may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of picloram in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Picloram is completely removed when water undergoes advanced oxidation with iron catalysed ultraviolet irradiation and peroxide (Fenton reaction) (Huston and Pignatello 1999). More research into the removal of picloram is recommended.

MEASUREMENT

Picloram may be measured in drinking waters by liquid chromatography–mass spectrometry, with a typical detection limit of 0.01 µg/L (QFSS 2009 pers comm).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for picloram is 0.07 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 7 mg/kg bw/day from a short-term (3-month) dietary study in dogs. The NOEL is based on increased liver weight. The ADI incorporates a safety factor of 100 and was first established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Picloram is readily absorbed in the gastrointestinal tract. It is not extensively metabolised, and is rapidly excreted in the urine, almost completely within 48 hours.

Acute effects: Picloram and its esters and salts have low acute oral dermal toxicity. There is some evidence that picloram is a skin sensitiser in humans. Picloram esters and salts are skin sensitisers in guinea pigs.

Short-term effects: A 3-month oral toxicity study conducted in rats reported no adverse effects up to maximal levels of exposure relevant to humans. A 6-month dietary study in dogs reported increased liver weight at 35 mg/kg bw/day. The lowest overall NOEL was 7 mg/kg bw/day in dogs. This NOEL is the basis for the current ADI.

Long-term effects: Long-term dietary studies were conducted in rats and dogs. In both rats and dogs, the studies reported increases in liver weight, hepatocellular enlargement, and liver discolouration at doses of 60 mg/kg bw/day and above. The lowest overall NOEL was 20 mg/kg bw/day in the rat.

Carcinogenicity: There is limited evidence of picloram (technical grade) carcinogenicity in rats.

Genotoxicity: Picloram is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two- and 3-generation reproduction studies in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Picloram is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.3 mg/L for picloram was determined as follows:

$$0.3 \text{ mg/L} = \frac{7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 7 mg/kg bw/day is the NOEL based on a medium-term (3-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Huston PL, Pignatello JJ (1999). Degradation of selected pesticide active ingredients and commercial formulations in water by the photo-assisted Fenton reaction 1999. *Water Research,* 33(5):1238-1246.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

QFSS (Queensland Forensic and Scientific Services) (2009). Personal communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Piperonyl butoxide

(endorsed 2011)

GUIDELINE

Based on human health concerns, piperonyl butoxide in drinking water should not exceed 0.6 mg/L.

RELATED CHEMICALS

Piperonyl butoxide (CAS 51-03-6) is a derivative of safrole and does not belong to a recognised chemical class. There are no chemically-related pesticides (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, piperonyl butoxide would not be a health concern unless the concentration exceeded 0.6 mg/L. Minor excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Piperonyl butoxide is a synergist for pyrethrin and pyrethroid insecticides and parasiticides for the control of parasites and insects.

There are many registered products that contain piperonyl butoxide in Australia. It is only ever used in combination with insecticides and parasiticides to increase their efficacy. The products are intended for professional and domestic use. Products containing piperonyl butoxide are available as skin ointments, throat/ear drops, aerosol sprays, powders, shampoos and soluble concentrates. Products are applied topically when used as a parasiticide on pets or livestock, and are sprayed as aerosols or diluted solutions when used as an insecticide on fruit, cereal, and vegetable crops. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to piperonyl butoxide and its metabolites are dermal and inhalation exposure from the use of domestic insecticide products, and residues in foods. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of piperonyl butoxide may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater. The veterinary use of piperonyl butoxide provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of piperonyl butoxide in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of piperonyl butoxide in drinking water have been identified.

MEASUREMENT

Piperonyl butoxide may be measured in drinking water by gas chromatography–mass spectrometry. The limit of reporting for this method is typically 0.1 µg/L (QFSS 2009 pers comm).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for piperonyl butoxide is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 16 mg/kg bw/day from a long-term study (1-year dietary study) in dogs. The NOEL is based on increased liver weight and associated hepatocellular hypertrophy, increased serum ALP activity, and increased thyroid weight. The ADI incorporates a safety factor of 100, and was established in 1997.

The previous ADI for piperonyl butoxide was 0.03 mg/kg bw, based on a NOEL of 3 mg/kg bw/day from the same long-term study in dogs. The ADI was amended in 1997 after a re-evaluation of this study.

The previous *Australian Drinking Water Guidelines* health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Piperonyl butoxide is poorly but rapidly absorbed from the gastrointestinal tract. The absorbed fraction is mostly distributed to the liver and fat, and undergoes extensive metabolism. Excretion occurs relatively rapidly, through the urine (30%) and faeces and is essentially complete within 7 days.

The metabolites of piperonyl butoxide inhibit detoxification enzymes in the liver and are responsible for the synergising effects.

Acute effects: Piperonyl butoxide has low acute oral and dermal toxicity, and is not a skin sensitiser. In humans, no adverse effects were seen after a single oral dose of 0.7 mg/kg bw

Short-term effects: A 4-week dietary study in rats reported histopathological changes in the liver at the lowest dose, 62.5 mg/kg bw/day. An 8-week dietary study in dogs reported decreased bodyweight, increased liver weight and hypertrophy, and decreased testis weights at 62 mg/kg bw/day. Medium-term studies mice reported decreased bodyweight gain, increased liver weight and hypertrophy, and clinical chemistry changes 100 mg/kg bw/day.

Long-term effects: In long-term dietary studies in dogs (1 year), mice (18 months), and rats (up to 22 months), changes included decreased bodyweight gain, increased liver and thyroid weight, and changes indicative of mild liver toxicity at doses of 62 mg/kg bw/day and above. Liver hyperplasia and increased kidney weight were also observed in rats and mice at higher doses. The lowest overall NOEL was 16 mg/kg bw/day in the 1-year study in dogs, and this is the basis for the ADI.

Carcinogenicity: Based on 18- to 22-month studies in mice and rats, there is no evidence of carcinogenicity for piperonyl butoxide.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Piperonyl butoxide is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Piperonyl butoxide is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.6 mg/L for piperonyl butoxide was determined as follows:

$$0.6 \text{ mg/L} = \frac{16 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 16 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

QFSS (Queensland Forensic and Scientific Services) (2009). QFSS, Personal communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Pirimicarb

(endorsed 2011)

GUIDELINE

Based on human health concerns, pirimicarb in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Pirimicarb 9 CAS 23103-98-2) belongs to the carbamate class of chemicals. There are many pesticides in this class, including aldicarb, bendiocarb, carbaryl, carbofuran, methiocarb, methomyl and oxamyl (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pirimicarb would not be a health concern unless the concentration exceeded 0.007 mg/L. Excursions above this level even for a relatively short period are of concern, as the health-based guideline is based on short- to medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pirimicarb is a post-emergent insecticide for the control of aphids in pastures and a variety of agricultural crops and ornamentals.

There are registered products that contain pirimicarb in Australia. The products are intended for professional use and are available as concentrated solutions, powders and granular formulations. Products are intended for application as a concentrated or dilute spray using high volume application spray equipment such as aircraft or ground boom. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to pirimicarb and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of pirimicarb may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Pirimicarb has been routinely monitored by water utilities in Australia. No detections above analytical limits of detection have been reported in the reviewed reports.

TREATMENT OF DRINKING WATER

Pirimicarb undergoes photochemical and metabolic degradation. Greater than 99.5% removal of pesticides has been reported with the ozonation, biological activated carbon filtration and reverse osmosis treatment (Bonne et al. 2000). Reverse osmosis alone was able to remove 99% of the influent concentration of pirimicarb in a challenger test conducted using two different type of membranes (Bonne *et al.* 2000).

MEASUREMENT

Pirimicarb can be analysed by on-line solid phase extraction followed by high-performance liquid chromatography-tandem mass spectrometry. The method can achieve a limit of quantitation (LOQ) of 0.4 µg/L. Solid-phase microextraction followed by gas chromatography–mass spectrometry can achieve a LOQ of 0.1 µg/L (Carabias-Martinez *et al.* 2005). Solid-phase extraction followed by high-performance liquid chromatography coupled to atmospheric pressure electrospray ionisation mass spectrometry can also achieve a LOQ of 0.1 µg/L (Nogueira *et al.* 2003). Solid-phase extraction followed by liquid chromatography with diode array detection can achieve a LOQ of 0.02 µg/L (Van Hoof *et al.* 2002). Single-drop microextraction followed by gas chromatography–mass spectrometry can achieve a LOQ of 50 ng/L (Saraji and Esteki 2008). Dispersive liquid–liquid microextraction coupled with high-performance liquid chromatography–diode array detection can achieve a LOQ of 0.6 ng/mL

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pirimicarb is 0.002 mg per kg of bodyweight (mg/kg bw), based on a lowest-observed-effect level (LOEL) of 0.4 mg/kg bw/day from a short-term (90-day dietary) study in dogs. The LOEL is based on haemotoxicity. The ADI incorporates a safety factor of 200, and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pirimicarb is readily absorbed via the gastrointestinal tract of mammals. It is readily metabolised and rapidly excreted, mostly in expired air, with the remainder excreted in urine and faeces. It has a low potential for bioaccumulation.

Acute effects: Pirimicarb has moderate acute oral toxicity and moderate acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: Short-term dietary studies conducted in mice, rats, dogs and monkeys reported haematological effects in dogs as the most sensitive toxicological endpoint. A study in rats reported decreased bodyweight gain and food consumption at the highest dose of 75 mg/kg bw/day. A study in dogs reported increased incidence of megaloblasts in bone marrow at 0.4 mg/kg bw/day. This LOEL is the basis for the current ADI. Effects in dogs at higher doses included plasma cholinesterase inhibition, decreased bodyweight gain and effects indicative of bone marrow dysfunction. A study in monkeys reported plasma cholinesterase inhibition at doses of 7 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies have been conducted in mice, rats and dogs. In mice, no effects were reported up to doses of 60 mg/kg bw/day. A two-year study in rats reported decreased bodyweight gain at 25 mg/kg bw/day and above. Plasma cholinesterase was inhibited at the highest dose of 75 mg/kg bw/day. A study in dogs reported increased erythroid/myeloid ratio in females at the highest dose tested of 4 mg/kg bw/day.

Carcinogenicity: There was some evidence of an increased incidence of lung tumours in mice but only at very high dose levels, well in excess of the likely level of human exposure.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: Pirimicarb is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: One and three-generation reproductive studies in rats did not produce any evidence of reproductive effects. Developmental studies in mice, rats and rabbits reported foetotoxicity and maternotoxicity at high dose levels that are well in excess of the likely level of human exposure.

Neurotoxicity: Special neurotoxicity studies in rats by short-term oral administration found no evidence of delayed neurotoxicity at doses up to 25 mg/kg bw/day.

Poisons Schedule: Pirimicarb is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for pirimicarb was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.4 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 0.4 mg/kg bw/day is the LOEL based on a short-term (90-day) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is the safety factor applied to the LOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation, with an additional safety factor of 2 for the use of a LOEL instead of a no-observed-effect level.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bonne PAC, Beerendonk EF, van der Hoek JP, Hofman JAMH (2000). Retention of herbicides and pesticides in relation to aging of RO membranes. *Desalination,* 132(1-3):189-193.

Carabias-Martinez R, Garcia-Hermida C, Rodriguez-Gonzalo E, Ruao-Miguel L (2005). Behaviour of carbamate pesticides in gas chromatography and their determination with solid-phase extraction and solid-phase microextraction as preconcentration steps. *Journal of Separation Science*, 28(16):2130-8.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Nogueira JM, Sandra T, Sandra P (2003). Considerations on ultra trace analysis of carbamates in water samples. *Journal of Chromatography A,* 996(1-2):133-40.

NOTE: Important general information is contained in PART II, Chapter 6

Saraji M, Esteki N (2008). Analysis of carbamate pesticides in water samples using single-drop microextraction and gas chromatography-mass spectrometry. *Analytical and Bioanalytical Chemistry*, 391(3):1091-100.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Van Hoof F, Van Wiele P, Acobas F, Guinamant J-L, Bruchet A, Schmitz I, Bobeldijk I, Sacher F, Ventura F, Boleda R (2002). Multiresidue determination of pesticides in drinking and related waters by solid-phase extraction and liquid chromatography with ultraviolet detection: interlaboratory study. *Journal of AOAC International,* 85(2):375-83.

NOTE: Important general information is contained in PART II, Chapter 6

Pirimiphos methyl

(endorsed 2011)

GUIDELINE

Based on human health concerns, pirimiphos methyl in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Pirimiphos methyl (CAS 29232-93-7) belongs to the organophosphate class of chemicals. There are many other pesticides in this class including acephate, dichlorvos, fenthion, malathion, omethoate and trichlorfon (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pirimiphos methyl would not be a health concern unless the concentration exceeded 0.09 mg/L. Excursions above this level even for a relatively short period are of concern, as the health-based guideline is based on short- to medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pirimiphos methyl is an insecticide for the control of pests such as cockroaches, fleas, ants, mosquitoes and flies in domestic, public, commercial and industrial areas, and agricultural buildings. It is also used as a fumigant to treat stored grain and peanuts.

There are registered products that contain pirimiphos methyl in Australia. The products are intended for professional and domestic use and are available as concentrated solutions to be applied in diluted form using pressurised hand-held spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The possible sources of public exposure to pirimiphos methyl and its metabolites are the use of domestic products, residues found in publicly accessible areas, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The use pattern of pirimiphos methyl for treatment of mosquito larvae involves direct application to water that may harbour larvae, and which may then enter source waters for drinking water. Other insecticidal uses of pirimiphos methyl may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on pirimiphos methyl occurrence in Australian drinking water supplies were found. Pirimiphos-methyl was considered by the World Health Organization (WHO) for addition to drinking water in containers as a mosquito larvicide treatment, particularly to control dengue fever. However, the

WHO does not recommended their use for direct application to drinking water unless no other effective and safe treatment is available (WHO 2008).

TREATMENT OF DRINKING WATER

Pirimiphos methyl can be completely mineralised by hydroxyl radicals (HO) generated by Electro-Fenton process (Guivarch *et al.* 2003) and it can be readily degraded in water by ozone-forming polar phenol derivatives (Chiron *et al.* 1998). Powdered activated carbon filtration and reverse osmosis have been demonstrated to be highly effective for the removal of organic chemicals including pesticides in water (Heijman and Hopman 1999).

MEASUREMENT

Pirimiphos methyl can be extracted from water by liquid/liquid extraction with dichloromethane. The extract is dried with sodium sulfate, concentrated, and analysed by gas chromatography–mass spectrometry in selected ion monitoring mode. The method can achieve a limit of quantitation (LOQ) of 0.06 µg/L. Solid-phase microextraction using ceramic/carbon materials followed by gas chromatography with a flame thermionic detector can achieve a LOQ of 15.6 ng/L (Zeng *et al.* 2008). Enzyme-linked immunosorbent assay can achieve pirimiphos detection limits of 0.01 µg/mL (Tang *et al.* 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pirimiphos methyl is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.25 mg/kg bw/day from short-term studies on human volunteers. The NOEL is based on the absence of adverse effects at the highest dose tested. The ADI incorporates a safety factor of 10, and was established in 1991.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pirimiphos methyl is readily absorbed via the gastrointestinal tract of rats and dogs, and is extensively metabolised to non-phosphorylated derivatives. In rats, pirimiphos methyl was excreted rapidly in both urine (85%) and faeces (15%), with 12 metabolites detected in urine but not identified.

Acute effects: Pirimiphos methyl has low acute oral and dermal toxicity. It methyl is not a skin sensitiser.

Short-term effects: Short-term dietary studies in rats reported decreased bodyweight gain and food consumption, and inhibition of brain and plasma cholinesterase, at doses of 4 mg/kg bw/day and above. Clinical signs of cholinesterase inhibition and haematological effects were reported at 200 mg/kg bw/day. A 13-week dietary study in dogs reported inhibition of erythrocyte cholinesterase at doses of 2 mg/kg bw/day. At doses of 10 mg/kg bw/day and above, clinical signs consistent with cholinesterase inhibition were observed, as well as decreased bodyweight gain and evidence of mild liver toxicity.

In two human volunteer studies (0.25 mg/kg bw/day for 28 or 56 days), there was no significant change in plasma and erythrocyte cholinesterase activity or liver function parameters. The NOEL from these studies was 0.25 mg/kg bw/day and is the basis of the current ADI.

NOTE: Important general information is contained in PART II, Chapter 6

Long-term effects: Long-term dietary studies in mice, rats and dogs reported cholinesterase inhibition to be the most sensitive toxicological effect. In mice, plasma and erythrocyte cholinesterase were inhibited at 25 mg/kg bw/day, with no associated clinical signs. In rats, plasma and brain cholinesterase were inhibited at 2.5 mg/kg bw/day, with slight anaemia at higher doses. In dogs, inhibition of plasma cholinesterase with associated clinical signs was reported at 2 mg/kg bw/day.

Carcinogenicity: Based on 2-year studies in dogs, mice and rats, there is no evidence of carcinogenicity for pirimiphos methyl.

Genotoxicity: Pirimiphos methyl is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation study in rats and developmental studies in rats and rabbits did not indicate any adverse effects on reproductive parameters or foetal development.

Neurotoxicity: Special neurotoxicity studies in rats by dietary administration found no evidence of delayed neurotoxicity.

Poisons Schedule: Pirimiphos methyl is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.09 mg/L for pirimiphos methyl was determined as follows:

$$0.09 \text{ mg/L} = \frac{0.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.25 mg/kg bw/day is the NOEL based on two short-term (28- and 56-day) studies involving human volunteers.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from human studies, to allow for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Chiron S, Rodriguez A, Fernandez-Alba A (1998). Application of gas and liquid chromatography-mass spectrometry to the evaluation of pirimiphos methyl degradation products in industrial water under ozone treatment. *Journal of Chromatography A*, 823(1-2):97-107.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Guivarch E, Oturan N, Oturan MA (2003). Removal of organophosphorus pesticides from water by electrogenerated Fenton's reagent. *Environmental Chemistry Letters,* 1(3):165-168.

NOTE: Important general information is contained in PART II, Chapter 6

Heijman SGJ, Hopman R (1999). Activated carbon filtration in drinking water production: model prediction and new concepts. *Colloids and Surfaces A: Physicochemical and Engineering Aspects,* 151:303–310.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tang J, Zhang M, Cheng G, Li A, Lu Y (2008). Development of IC-ELISA for detection of organophosphorus pesticides in water. *Journal of Environmental Science and Health B*, 43(8):707-12.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2008). *Guidelines for Drinking-water Quality*. 3rd Edition including 1st and 2nd addenda, WHO, Geneva, Switzerland.

Zeng J, Yu B, Chen W, Lin Z, Zhang L, Chen X, Wang X (2008). Application of ceramic/carbon composite as a novel coating for solid-phase microextraction. *Journal of Chromatography A,* 1188(1):26-33.

NOTE: Important general information is contained in PART II, Chapter 6

Plasticisers
Di(2-ethylhexyl) phthalate (DEHP)
Di(2-ethylhexyl) adipate (DEHA)

(endorsed 1996)

GUIDELINE

Di(2-ethylhexyl) phthalate: Based on health considerations, concentrations in drinking water should not exceed 0.01 mg/L.

Di(2-ethylhexyl) adipate: The data are inadequate to determine a guideline value.

GENERAL DESCRIPTION

DEHP and DEHA are commonly used plasticisers in flexible polyvinyl chloride products. They may be present in drinking water that has been in contact with these products for long periods of time, or as the result of industrial spills. Overseas studies have detected DEHP in drinking water on a few occasions at concentrations from 0.00005 mg/L (50 ng/L) to 0.01 mg/L. DEHA has been detected at concentrations between 0.000001 mg/L (1 ng/L) to 0.0001 mg/L (100 ng/L) in treated drinking water.

DEHP is the most widely used plasticiser. It is also used as a replacement for polychlorinated biphenyls (PCBs) in electrical capacitors. DEHA is used as a lubricant and in hydraulic fluids. Exposure to DEHP and DEHA is widespread because of the broad range of products using these plasticisers. Food is the major source of exposure, and it has been estimated that adult daily intake of DEHP and DEHA, as a result of consumption of food in contact with plastic products, is 0.2 mg to 16 mg.

People receiving kidney dialysis treatment may be exposed to much higher amounts of these plasticisers. In the United States it has been estimated that each dialysis patient could be receiving up to 90 mg of DEHP per treatment.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on DEHP or DEHA concentrations in Australian drinking waters. It is unlikely that concentrations would exceed those reported overseas.

TREATMENT OF DRINKING WATER

There are no published reports on methods for the removal of DEHP or DEHA from drinking water.

MEASUREMENT

Measurement can be undertaken using a liquid extraction procedure (USEPA Draft Method 506 1990). The water sample is extracted with a ternary solvent consisting of methylene chloride, hexane and ethyl acetate. The extract is concentrated and analysed by gas chromatography with photoionization detection. The limit of determination is lower than 0.01 mg/L.

HEALTH CONSIDERATIONS

In animals, DEHP and DEHA are efficiently absorbed from the gastrointestinal tract, although marked differences in absorption are seen between species. Metabolism also differs markedly between species. Highest concentrations of metabolites are seen in the liver and adipose tissue.

An extensive review and summary of the human and animal toxicity data for DEHP is available

(IPCS 1992).

Human volunteers fed up to 10 g of DEHP have experienced mild gastric disturbances, which occurred only at the highest dose. Dialysis patients receiving 150 mg per week intravenously showed no liver changes after one month, but had higher peroxisome numbers after a year. No data exist on the effects of ingested DEHA in humans.

Exposure to DEHP and DEHA can result in a significant increase in peroxisome proliferation in the liver cells of rats. An increase in peroxisome proliferation has been linked to the development of liver tumours in rodents. Humans are regarded as being less sensitive to chemically induced peroxisomal proliferation than rodents.

Long-term gavage (measured force-feeding) studies in rats using DEHP have reported that doses of 100 mg/kg body weight increased the activity of peroxisomal-associated enzymes, with higher doses resulting in depression of growth and enlargement of the liver and kidneys. Very high doses resulted in increased incidence of liver tumours. Short-term studies have reported increases in liver peroxisomal activity at lower doses (from 25 mg/kg body weight per day).

DEHP adversely affects reproduction in mice at 140 mg/kg body weight per day, and it is teratogenic and fetotoxic in mice with a no-effect level of 35 mg/kg body weight per day.

A short-term study using DEHA in rats and mice reported peroxisomal proliferation with a no-effect level of 100 mg/kg body weight per day. Longer-term studies are available, but have used much higher doses. DEHA adversely affects reproduction in rats at doses from 128 mg/kg body weight per day.

Neither DEHP nor DEHA exhibited mutagenic activity when applied to bacteria or to mammalian cells.

The International Agency for Research on Cancer has concluded that DEHA is not classifiable as to its carcinogenicity to humans (Group 3, no adequate evidence in humans and limited evidence in animals) and that DEHP is possibly carcinogenic to humans (Group 2B, no adequate evidence in humans but sufficient evidence in animals) (IARC 1982).

Derivation of guidelThe guideline values were determined as follows:

i) DEHP

$$0.01 \text{ mg/L} = \frac{25 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.01}{2 \text{ L/day} \times 1000}$$

where:

- 25 mg/kg body weight per day is the lowest effect level based on a 14-day study using rats and hamsters (IPCS 1992). Although longer-term studies are available, they report no-effect levels at higher doses.
- 70 kg is the average weight of an adult.
- 0.01 is the proportion of total daily intake attributable to the consumption of water. Sufficient data are available to indicate that food is by far the major source of exposure, and that drinking water contributes approximately 1% of total daily intake.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 because effects were observed at the lowest dose).

An additional safety factor for carcinogenic effects was not applied, as rats are by far the most sensitive species with respect to the sensitive end-point of peroxisomal proliferation.

The World Health Organization (WHO) guideline value of 0.008 mg/L was based on an adult body

weight of 60 kg. The difference in guideline values is not significant.

ii) DEHA

The WHO has calculated a guideline value of 0.08 mg/L for DEHA based on a short-term developmental toxicity study (ICI 1988). The data are not considered to be adequate to determine an Australian guideline value.

REFERENCES

IARC (International Agency for Research on Cancer) (1982). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans: Some industrial chemicals and dyestuffs. World Health Organization, IARC, 29.

ICI (1988). Di(2-ethylhexyl)adipate: teratogenicity study in the rat. ICI Report No. CTL/P/2119.

IPCS (International Programme on Chemical Safety) (1992). Diethylhexyl phthalate. Environmental Health Criteria, 131. World Health Organization, IPCS.

USEPA Draft Method 506 (1990). Determination of phthalate and adipate esters in drinking water by liquid–liquid extraction or liquid–solid extraction and gas chromatography with photoionization detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

Polihexanide

(endorsed 2011)

GUIDELINE

Based on human health concerns, polihexanide in drinking water should not exceed 0.7 mg/L.

RELATED CHEMICALS

Polihexanide (CAS 50641-36-6) is a polymer of chlorhexidine. There are no related pesticides (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, polihexanide would not be a health concern unless the concentration exceeded 0.7 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Polihexanide is a disinfectant used to control microorganisms in veterinary hospitals and animal accommodations, and as a sanitiser for milk-handling equipment.

There is at least one registered product containing polihexanide in Australia. Polihexanide products are intended for professional use and are available as a concentrate. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to polihexanide are residues in food (dairy products), and exposure to treated surfaces.

Use of polihexanide as a disinfectant may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of polihexanide in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of polihexanide in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

No suitable analytical techniques have been identified, but the use of high performance liquid chromatography–tandem mass spectrometry is expected to be suitable for residue levels of this pesticide in water.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) and acute reference dose (ARfD) for polihexanide is 0.2 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 20 mg/kg bw/day from a rabbit developmental study. The NOEL was based on maternotoxicity in pregnant rabbits. The ADI and ARfD incorporate a safety factor of 100, and were established in 2007.

An *Australian Drinking Water Guidelines* health value has not been previously established for polihexanide.

HEALTH CONSIDERATIONS

Metabolism: Polihexanide is poorly absorbed via the gastrointestinal tract and is rapidly excreted mainly in the faeces (94%), with the absorbed portion excreted in urine. The metabolic fate of polihexanide is unknown.

Acute effects: Polihexanide has a low acute oral and dermal toxicity. It is a skin sensitiser in guinea pigs.

Short-term effects: Repeat-dose dermal exposure in rats caused reddening and inflammation of the skin at 200 mg/kg bw/day. Ninety-day dietary studies were conducted in rats and dogs. In rats, there was reduced bodyweight gain and haemosiderin deposits in the liver at 68 mg/kg bw/day. In dogs, there were haemosiderin deposits in the spleen at 68 mg/kg bw/day.

Long-term effects: Long-term studies were conducted in mice (2-years), rats (2-years) and dogs (1-year). In mice, there were treatment-related effects in the liver (increase in haemangiosarcomas) and recto-anal junction (inflammation, hyperplasia and squamous cell carcinomas) at 180 mg/kg bw/day. In rats, there was some evidence of an increase in haemangioma/haemangiosarcomas in the liver at 100 mg/kg bw/day. In dogs, there was evidence of systemic toxicity at 45 mg/kg bw/day.

Carcinogenicity: Polihexanide induced an increased incidence in haemangiomas/haemangiosarcomas in the liver of mice at a high dose level that is well in excess of the normal level of human exposure. The increased incidence of squamous cell carcinomas in the recto-anal junction was considered to be a consequence of irritation/inflammation at the high dose level and not relevant to human exposure.

Genotoxicity: Polihexanide is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproduction and developmental effects*:* A reproduction study in rats and developmental studies in rats and rabbits did not show any evidence of effects on reproductive parameters or on foetal development. The NOEL for maternal toxicity in rabbits was 20 mg/kg bw/day, and this was the basis for the ADI.

Poisons Schedule: Polihexanide is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.7 mg/L for polihexanide was determined as follows:

$$0.7 \text{ mg/L} = \frac{20 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 20 mg/kg bw/day is the NOEL based on a developmental study in rabbits.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor applied to the NOEL from a developmental study conducted in rabbits. The safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variations.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Polycyclic aromatic hydrocarbons (PAHs)

(endorsed 2011)

GUIDELINE

Based on health considerations, the concentration of benzo[a]pyrene in drinking water should not exceed 0.00001 mg/L (10 ng/L). Data are inadequate to set guidelines for other PAHs, however comparative carcinogenic potency can be used to determine an approximate risk when complex mixtures of PAHs are present in drinking water.

GENERAL DESCRIPTION

The polycyclic aromatic hydrocarbons are a large group of organic compounds with two or more fused aromatic rings. Several hundred have been identified in air, emitted from various combustion and pyrolysis sources. The principal PAHs include phenanthrene, fluoranthene, pyrene, anthracene, benzo(a) pyrene, benzofluoranthene, chrysene, anthanthrene and naphthalene.

PAHs are widespread throughout the environment. They are formed in forest fires and in the combustion of fossil fuels, and are present in emissions from coke ovens, aluminium smelters and motor vehicles. Contamination of drinking water can occur by direct atmospheric deposition and by leaching from bituminous liners in water distribution systems.

There are very few data on concentrations of PAHs in drinking water supplies. The few data that exist are mainly for benzo(a)pyrene (BaP). The typical concentration of BaP in drinking water in the United States is estimated to be 0.00000055 mg/L (0.55 ng/L).

Background levels of PAHs in drinking water range from 4 to 24 ng/L (0.0004 to 0.0024 µg/L) (ATSDR 2008). The typical level of BaP in drinking water in the United States is estimated to be 0.55 ng/L (0.00055 µg/L) (WHO 1996, WHO 1998). The median value for daily intake for PAHs from exposure via drinking water has been estimated to be 0.006 µg/day (Menzie *et al.* 1992). Daily intake of total PAHs has also been estimated to be 0.027 µg (Santodonato *et al.* 1981), while the daily intake of BaP in drinking water is estimated to range from 0.1 to 1 ng (Santodonato *et al.* 1981, WHO 1996). In general it is suggested that PAHs in drinking water only contribute no more than 1% of total PAH intake (WHO 1996).

Food is the major source of intake of PAHs. Highest concentrations occur in smoked foods, leafy vegetables and the burnt fat of meats. Intake from foods is extremely variable but significantly higher (by at least an order of magnitude) than from drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

PAHs have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas. One potential contributor to PAHs in drinking water in Australia would be periodic intense and extensive bush fires in water storage catchments.

TREATMENT OF DRINKING WATER

The conventional water treatment processes of coagulation, settling and filtration are capable of reducing the BaP concentration of raw waters to less than 0.000001 mg/L (1 ng/L), even if the influent concentration is high. It is likely that other PAHs would be similarly reduced. Granular activated carbon would also be effective in the removal of these compounds.

MEASUREMENT

Concentrations of a wide range of PAHs in water can be determined by the use of gas chromatography–mass spectrometry (GC-MS) combined with suitable extraction and pre-concentration procedures. Alternatively, the use of fluorescence detection combined with high performance liquid chromatography (HPLC) provides a sensitive method for PAHs in water (USEPA Draft Method 550, 1990). Typically, solvent extraction employing dichloromethane or solid phase extraction using resins or commercially available adsorption cartridges or discs are used for extraction and concentration, prior to final determination by GC-MS or HPLC.

HEALTH CONSIDERATIONS

Most of the toxicological literature deals specifically with BaP. Few studies are available for the other PAHs. Some PAH compounds have been found to be carcinogenic by non-oral routes, but others are known to have low potential for carcinogenicity.

BaP is absorbed principally through the gastrointestinal tract and the lungs. The rate of absorption increases with increased intake of polyunsaturated fatty acids. BaP is rapidly distributed to the organs and may be stored in mammary and adipose tissue. Metabolism occurs mainly in the liver.

The International Agency for Research on Cancer (IARC) has upgraded BaP from group 2B to group 1 (carcinogenic to humans), based on mechanistic considerations and other relevant data. This evaluation is presented in full in IARC monograph 92. In addition, dibenz[a,h]anthracene and dibenzo[a,l]pyrene have been upgraded to group 2A (probably carcinogenic to humans), with supporting evidence from other relevant data. Certain substances containing complex mixtures of PAHs are classified as known human carcinogens (Group 1) by IARC. These include: coal tar pitches, coal tars and cigarette smoke condensate.

In experiments with animals, many PAH mixtures have been associated with an increased incidence of cancer. BaP is one of the most potent carcinogenic compounds, with primary tumours having been reported in a variety of studies, using different administration techniques, in mice, rats, hamsters, guinea pigs, rabbits, ducks and monkeys. Tumours have mostly appeared only at the site of administration.

BaP is metabolically activated to a series of dihydrodiol expoxides by the mixed function oxidases, particularly cytochrome P450 1A1, in combination with epoxide hydrolase. One particular dihydrodiol epoxide (+ anti isomer) is highly mutagenic and in its ultimate carcinogenic form (a carbonium ion), will bind to nucleophilic sites on DNA to product covalent DNA adducts. If these adducts are misrepaired, they may produce mutations in tumour suppression genes and cancer-causing oncogenes, which can lead to cancer. Other carcinogenic PAHs operate in a similar manner through metabolic activation and DNA adduct formation. The sensitive determination of DNA adducts can be useful in determination of human exposure to carcinogenic

DERIVATION OF GUIDELINE

Data are insufficient to set guideline values for PAHs except for BaP. The use of relative potencies of other PAHs can, however, give guidance to their relative contribution to risk caused by their presence in drinking water. Relative potencies of the most commonly found PAHs are given in Table 1.

NOTE: Important general information is contained in PART II, Chapter 6

The relative carcinogenic potencies of a number PAHs have been determined and are reported in the literature. Table 1 presents those data with summarised relative potencies sourced from the World Health Organization (WHO 1998) and additional data as referenced.

Table 1: *Relative carcinogenic potency of selected PAHs*

PAH	Relative potency with BaP as unity
Benz[a]anthracene	0.1*
Benzo[a]pyrene	1*
Benzo[b]fluoranthene	0.1*
Benzo[j]flouranthene	0.1*
Benzo[k]flouranthene	0.1*
Dibenz[a,h]anthracene	1*
Indeno[1,2,3,c,d]pyrene	0.1*
Benzo[g,h,i]perylene	0.01**
Benz[a]anthracene	0.1** and ***
Chrysene	0.01** and ***
Dibenz[a,h]anthracene	5**
Dibenz[a,e]pyrene	1***
Dibenz[a,h]pyrene	10***
Dibenz[a,i]pyrene	10***
Dibenz[a,l]pyrene	10***

* Sourced from WHO (1998)

** Sourced from Nisbet and LaGoy (1992)

*** Sourced from CA EPA (2002)

A number of points in relation to Table 1 are worth noting. PAHs with relative carcinogenic potencies of less than 0.01 (100 times less carcinogenic than BaP) are not reported due to the lower level of risk they pose in water. There are three PAHs listed in Table 1 with relative carcinogenic potencies ten times that of BaP, and these PAHs should be given attention when risk assessments of PAHs in drinking water are undertaken. Slope factors for carcinogenic risk assessment of PAHs are provided by the California Environmental Protection Agency (CA EPA 2002). Additional data on slope factors and drinking water unit risk for BaP are given in the IRIS database (USEPA 2008).

While it is recognised that a guideline is available only for BaP, this can be used in conjunction with relative potencies given in Table 1 to gain an estimate of acceptable levels when complex mixtures of PAHs are present.

On the basis of a feeding study using mice (Neal and Rigdon 1967), the excess risk of lifetime consumption of water with a BaP concentration of 0.00007 mg/L (70 ng/L) was conservatively estimated by WHO, using a linear multistage model, at one additional cancer per million people.

The guideline value has been set at the limit of determination because this is slightly less than the value derived using a risk assessment calculation, and provides an adequate degree of protection. This is consistent with the general approach adopted for genotoxic carcinogens (see Section 6.3.3).

The WHO guideline value of 0.0007 mg/L was based on an oral carcinogenicity study in mice and calculated using a 2-stage birth-death mutation model, and on carcinogenicity studies in mice following oral administration (WHO, 2004).

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

ATSDR (Agency for toxic Substances and Disease Registry) (2008). Public Health Statement for Polycyclic Aromatic Hydrocarbons (PAHs). ATSDR. Available from: http://www.atsdr.cdc.gov/toxprofiles/phs69.html

CEPA (California Environmental Protection Agency) (2002). Air toxic hotspots program risk assessment guidelines. Part II. Technical support document for describing available cancer potency factors. Available from: http://www.oehha.org/air/cancer_guide/TSD2.html

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IARC (2010) *Monograph on the Evaluation of Carcinogenic Risks to Humans. Volume 92: Some non-heterocyclic, polycyclic aromatic hydrocarbons and some related exposures.* World Health Organization, International Agency for Research on Cancer, Lyon, France

Menzie CA, Potocki BB, Santodonato J (1992). Exposure to carcinogenic PAHs in the environment. *Environmental Science and Technology*, 26:1278-1284.

Neal J, Rigdon RH (1967). Gastric tumors in mice fed benzo(a)pyrene: a quantitative study. *Texas Reports on Biology and Medicine*, 25:553–557.

Nisbet ICT, Lagoy PK (1992). Toxic equivalency factors (TEF's) for polycyclic aromatic hydrocarbons (PAHs). *Regulatory and Toxicological Pharmacology,* 16:290-300.

Santodonato J, Howard P, Basu D (1981). Health and ecological assessment of polynuclear aromatic hydrocarbons. *Journal of Environmental Pathology and Toxicology*, 5:1-364.

USEPA (United States Environmental Protection Agency) (2008). Integrated Risk Information system (IRIS), Benzo[a]pyrene CASRN 50-32-8. Accessed at http://cfpub.epa/ncea/iris/.

USEPA Draft Method 550 (1990). Determination of polycyclic aromatic hydrocarbons in drinking water by liquid–liquid extraction and HPLC with coupled ultraviolet and fluorescence detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

WHO (World Health Organization) (1996). *Guidelines for Drinking-Water Quality*, 2nd Edition, Vol. 2 – Health criteria and other supporting information. WHO, Geneva, Switzerland.

WHO (World Health Organization) (1998). *Guidelines for Drinking-Water Quality*, 2nd edition, Addendum to Volume. 2 – Health criteria and other supporting information. WHO, Geneva, Switzerland.

WHO (World Health Organization) (2004) *Guidelines for Drinking-Water Quality*, 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Profenofos

(endorsed 2011)

GUIDELINE

Based on human health concerns, profenofos in drinking water should not exceed 0.0003 mg/L.

RELATED CHEMICALS

Profenofos (CAS 41198-08-7) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes temephos, fenthion, and methidathion (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, profenofos would not be a health concern unless the concentration exceeded 0.0003 mg/L. Excursions above this level even for a short period are of concern as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Users: Profenofos is used as an insecticide and acaricide (miticide) for the control of certain insects on cotton crops.

There are registered products containing profenofos in Australia. The products are intended for professional use and are applied as concentrated or diluted solutions by either ground boom spray or aerial application methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to profenfos is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of chemical may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of profenfos in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of profenfos in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Profenofos can be measured in drinking waters by gas chromatography–mass spectrometry, with a practical limit of detection of 0.1 µg/L (QHFSS 2008 pers comm).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for profenofos is 0.0001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.0072 mg/kg bw/day from a 6-month dog study. The NOEL is based on inhibition of plasma cholinesterase. The ADI incorporates a safety factor of 100, and was established in 1982.

The previous *Australian Drinking Water Guidelines* health value was 0.0003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Profenofos is rapidly absorbed in the gastro-intestinal tract and is completely metabolised to four metabolites, which are excreted mainly in urine.

Acute effects: Profenofos has a moderate oral and low dermal toxicity. Profenofos is a skin sensitiser. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: Medium-term toxicity studies in rats (3 months) and dogs (6 months) reported decreased plasma, red blood cell and brain cholinesterase, with plasma cholinesterase the most sensitive, at dose levels above 0.3 mg/kg bw/day and 0.007 mg/kg bw/day, respectively.

Long-term effects: Long-term studies in mice and rats reported decreased cholinesterase as the most sensitive toxicity endpoint. Decreased plasma cholinesterase was noted at dose levels above 0.015 mg/kg bw/day in the rat.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for profenofos.

Genotoxicity: There is equivocal evidence of genotoxicity from *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a reproduction study in rats and developmental studies in rats and rabbits, there was no evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Profenofos is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.0003 mg/L for profenofos was determined as follows:

$$0.0003 \text{ mg/L} = \frac{0.0072 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.0072 mg/kg bw/day is the NOEL based on a medium-term (6-month) study in dogs.
- 70 kg is taken as the average weight of an adult
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.

NOTE: Important general information is contained in PART II, Chapter 6

- 2 L/day is the estimated maximum amount of water consumed by an adult
- 100 is a safety factor applied to the NOEL from a study conducted in dogs. The safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) T***he Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

QHFSS (Queensland Health Forensic and Scientific Services) (2008). Personal communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Promecarb

(endorsed 2011)

GUIDELINE

The health concerns associated with promecarb have not been fully evaluated and therefore a health value for promecarb in drinking water cannot be set.

RELATED CHEMICALS

Promecarb (CAS 2631-37-0) belongs to the carbamate class of chemicals. There are many other pesticides in this class, which includes aldicarb, carbaryl and methomyl (Tomlin 2006).

HUMAN RISK STATEMENT

There are currently insufficient data on which to base a human risk statement.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Promecarb is an insecticide formerly used to control lepidopterous pests, leaf miners of fruits, Colorado potato beetle and common rootworm, in agriculture and the home garden.

There are no registered products containing promecarb in Australia, but de-registered compounds may still be detected in water. Previously registered products were intended for both professional and home garden use.

Exposure sources: If used in the future, the main source of public exposure to promecarb and its metabolites would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of promecarb in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No occurrence data on promecarb in Australian waters could be found.

TREATMENT OF DRINKING WATER

No information on efficiency of drinking water treatment to remove promecarb could be found.

MEASUREMENT

Promecarb can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 1 µg/L (Queensland Health, 2007).

HISTORY OF THE HEALTH VALUES

There is currently no acceptable daily intake (ADI) or acute reference dose (ARfD) value for promecarb.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L.

HEALTH CONSIDERATIONS

Metabolism: Promecarb is well absorbed from the gastrointestinal tract, extensively metabolised and rapidly excreted, mainly via the urine.

Acute effects: Promecarb has high acute oral toxicity and moderate acute dermal toxicity. It is unknown whether promecarb is a skin sensitiser. In rats, inhibition of plasma, brain and red blood cell cholinesterase was observed after a single oral dose of 10 mg/kg bw. Recovery was seen within 24 hours.

Short-term effects: In 3-month oral studies in mice, there was increased mortality, decreased bodyweight gain, severe hyperglycemia, decreased spleen and ovarian weights, and liver degeneration at 8.9 mg/kg bw/day and above.

In 3-month dietary studies in rats and dogs, no treatment-related effects were observed up to 20 mg/kg bw/day in rats and up to 5 mg/kg bw/day in dogs. These were the highest doses tested for each species.

Long-term effects: In an 18-month dietary study in rats, there were no clinical signs of toxicity at 20 mg/kg bw/day, the highest dose tested (cholinesterase activity was not measured).

Carcinogenicity: In an 18-month study in rats, there was no evidence of carcinogenicity for promecarb.

Genotoxicity: Only short-term *in vitro* studies are available; these report no evidence that promecarb is genotoxic.

Reproductive and developmental effects: There were no reproduction studies available. In a developmental study in rats, maternotoxicity and delayed ossification in the foetus occurred at 5 mg/kg bw/day.

Poisons Schedule: Promecarb was removed from Schedule 6 of the Standard for the Uniform Scheduling of Drugs and Poisons in August 1993 due to concerns over the lack of toxicity and residue data. Current versions of the Standard for the Uniform Scheduling of Medicines and Poisons (the Poisons Standard) (DoHA 2010) should be consulted for further information. The Poisons Standard are available from the ComLaw website: http://www.comlaw.gov.au/Details/F2010L02386/Download.

DERIVATION OF THE HEALTH-BASED GUIDELINE

There are currently insufficient data on which to establish a health-based guideline for promecarb in drinking water in Australia.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Propachlor

(endorsed 2011)

GUIDELINE

Based on human health concerns, propachlor in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Propachlor (CAS 1918-16-7) belongs to the chloroacetamide class of chemicals. Other pesticides in this class include metolachlor and S-metolachlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propachlor would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propachlor is a herbicide for the control of annual grasses and certain broad-leaf weeds in sorghum and selected vegetable crops.

There is at least one registered product that contains propachlor in Australia. Propachlor products are intended for professional use, and are available as an aqueous concentrate to be diluted and applied as a surface spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to propachlor and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of propachlor may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of propachlor in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Propachlor can be effectively oxidised in drinking water by suitable dosing chemical oxidants such as ozone, or by photocatalytic advanced oxidation processes (Konstantinou *et al.* 2002, Liu *et al.* 2008).

MEASUREMENT

No suitable analytical methods have been identified for the analysis of propachlor in drinking waters. However, sensitive liquid chromatography–tandem mass spectrometry methods are available for the analysis of the major environmental degradation products of propachlor and other related herbicides in drinking waters (Fuhrman and Allan 2002, Shoemaker and Bassett 2006).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propachlor is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.0 mg/kg bw/day established in two long-term studies (an 18-month study in mice and a 2-year study in rats). The NOELs were based on increased relative liver weights in mice and increased absolute and relative weights of the thyroid and parathyroid glands in rats. The ADI incorporates a safety factor of 100, and was established in 1988.

Earlier ADIs established for propachlor were 0.1 and 0.002 mg/kg bw, set in 1972 and in 1987 respectively. The former ADI was based on a 90-day rat study in which the NOEL was 10 mg/kg bw/day; the latter was based on a NOEL of 3 mg/kg bw/day obtained in a 2-generation rat reproduction study. This NOEL was chosen in the absence of a NOEL derived from long-term toxicity studies.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Propachlor is absorbed readily and extensively in the gastrointestinal tract, with maximum blood concentration in 1 hour. It is metabolized rapidly and the major metabolites are excreted in the urine (68%) and faeces (19%). Biliary excretion followed by gut microflora metabolism and re-absorption is significant.

Acute effects: Propachlor has low acute oral and dermal toxicity. It is a skin sensitiser.

Short-term effects: Ninety-day dietary studies in mice, rats and dogs reported no clinical or pathological signs of toxicity at 930 mg/kg bw/day in mice, at 310 mg/kg bw/day in rats, or at 155 mg/kg bw/day in dogs.

Long-term effects: An 18-month dietary study in mice reported increases in liver weight at 7.5 mg/kg bw/day. A two-year dietary studies in rats reported increases in thyroid and parathyroid weights at 25 mg/kg bw/day. A 1-year dietary study in dogs reported a significant decrease in bodyweight in male dogs at 25 mg/kg bw/day. No clinical or pathological signs of toxicity were noted in any of these studies. The NOEL of 2 mg/kg bw/day from the long-term mouse and rat studies is the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for propachlor.

Genotoxicity: Propachlor is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a 2-generation reproduction study in rats and in a developmental study in rats, there was no evidence of reproductive or developmental effects. A developmental study in rabbits revealed post-implantation foetal losses and a subsequent decrease in the number of viable foetuses at 15 mg/kg bw/day, which was below the dose level causing maternal toxicity.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Propachlor is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for propachlor was determined as follows:

$$0.07 \text{ mg/L} = \frac{2.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.0 mg/kg bw/day is the NOEL based on long-term dietary studies in mice (18 months) and rats (2 years)
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Fuhrman JD, Allan JM (2002) Determination of acetanilide degradates in ground and surface waters by direct aqueous injection LC/MS/MS. In: Ferrer I, Thurman EM (eds), *Symposium on Analysis of Emerging Contaminants Using Liquid Chromatography, Mass Spectrometry and MS held at the 225th National Meeting of the American-Chemical-Society,* Orlando, Florida, pp. 256-272.

Konstantinou IK, Sakkas VA, Albanis TA (2002). Photocatalytic degradation of propachlor in aqueous TiO2 suspensions. Determination of the reaction pathway and identification of intermediate products by various analytical methods. *Water Research,* 36(11):2733-2742.

Liu C, Qiang ZM, Tian F, Zhang T (2008). Reactivity of several classes of pesticides with UV, ozone and permanganate. In: Wang X, Chen R (eds), *International Conference on Advances in Chemical Technologies for Water and Wastewater Treatment,* Xian, Peoples Republic of China, pp. 279-280.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Shoemaker JA, Bassett MV (2006). Development of EPA method 535 for the determination of chloroacetanilide and other acetamide herbicide degradates in drinking water by solid-phase extraction and liquid chromatography/tandem mass spectrometry. *Journal of AOAC International,* 89(1):201-209.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Propanil

(endorsed 2011)

GUIDELINE

Based on human health concerns, propanil in drinking water should not exceed 0.7 mg/L.

RELATED CHEMICALS

Propanil (CAS 709-98-8) belongs to the anilide class of chemicals. Another chemical in this class is pentanochlor (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propanil would not be a health concern unless the concentration exceeded 0.7 mg/L. Minor excursions above this level would need to occur over a significant period to be of health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propanil is a herbicide for the post-emergent control of barnyard grass in rice.

There are registered products that contain propanil in Australia. The products are intended for professional use and are available as concentrated solutions to be applied in diluted form using boom, aerial and knapsack spray application methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to propanil and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of propanil on rice fields may lead to contamination of source waters through entry into groundwater and processes such as run-off and spray drift.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Propanil has been tested but not detected in the Ord River Irrigation Area, Western Australia (Oliver and Kookana 2005). It was detected in one of 53 finished drinking water samples collected at different sites in the USA at 0.7 µg/L (Li *et al.* 2006). Propanil has been detected in the drainage channels of the main rice cultivating countries in Europe. The highest concentration reported was 16.82 µg/L (Kuster *et al.* 2008).

TREATMENT OF DRINKING WATER

Propanil can be removed from drinking water by granular activated carbon (Ayranci and Hoda 2004) and reverse osmosis treatment.

MEASUREMENT

Propanil can be measured in water by online solid-phase extraction (SPE) liquid chromatography mass spectrometry (LC-MS) or direct injection on a triple quadrapole LC-MS instrument in multiple reaction monitoring mode. The method can achieve a limit of quantitation (LOQ) of 1 µg/L. SPE followed by high performance liquid chromatography using photochemically-induced fluorescence detection can achieve a LOQ in the range of 0.07 to 0.7 µg/L (de la Pena *et al.* 2003). Li *et al.* (2006) reported a propanil LOQ in drinking water of 0.02 µg/L using liquid chromatography–electrospray ionization–mass spectrometry.

Propanil can also be detected at very low concentrations in water. A fully automated method using on-line solid-phase extraction–liquid chromatography–electrospray-tandem mass spectrometry can achieve a LOQ of 0.5 ng/L (Kampioti *et al.* 2005). A solid-phase microextraction-gas chromatography-mass spectrometry method can achieve a LOQ of 2 ng/L (Natangelo *et al.* 1999) and on-line solid-phase extraction-liquid chromatography–electrospray–tandem mass spectrometry can achieve a LOQ of 0.4 ng/L (Kuster *et al.* 2008). Moreover, a method without any sample pre-treatment and without pre-concentration using a fully automated immunoassay for detection of propanil in aqueous samples can achieve a LOQ of 0.6 ng/L (Tschmelak *et al.* 2004).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propanil is 0.2 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 20 mg/kg bw/day from a 2-year oral study in rats. The NOEL is based on decreased bodyweight gain, mild anaemia and organ weight changes. The ADI incorporates a safety factor of 100, and was established in 1981.

The previous *Australian Drinking Water Guidelines* health value was 0.5 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Propanil is readily and extensively absorbed via the gastrointestinal tract. It is metabolised and eliminated within 48 hours. The major metabolites are 3,4-dichloroaniline (DCA) and 3,3 ,4,4 -tetrachloroazobenzene (TCAB) (WHO 2004). These metabolites in addition to tetrachloroazoxybenzene (TCAOB) have been reported as impurities of toxicological concern in propanil. The Australian Pesticides and Veterinary Medicines Authority have set impurity limits of 20 mg/kg for TCAB and 2 mg/kg for TCAOB in propanil active constituent.

Acute effects: Propanil has low oral and dermal acute toxicity. It is not a skin sensitiser.

Short-term effects: A short-term dietary study in rats reported an increase in number of abnormal red blood cells at doses of 25 mg/kg bw/day. At higher dose levels, effects reported included haemolytic anaemia, decreased bodyweight gain and increased mortality. In a 4-week dietary study in dogs, decreased bodyweight gain and food consumption was reported at 375 mg/kg bw/day.

Long-term effects: Long-term dietary studies with propanil in rats and dogs showed the main toxicological effect to be haemotoxicity. A 2-year dietary study in rats reported decreased bodyweight gain, decreased haemoglobin and haematocrit levels, and increased spleen, liver and testes weights at 80 mg/kg bw/day. In dogs, there was decreased bodyweight gain and decreased haemoglobin levels at 225 mg/kg bw/day and above. The lowest overall NOEL was 20 mg/kg bw/day in the rat study and this is the basis for the current ADI.

Carcinogenicity: Based on 2-year studies in rats and dogs, there is no evidence of carcinogenicity for propanil.

NOTE: Important general information is contained in PART II, Chapter 6

Genotoxicity: The genotoxicity of propanil has not been evaluated in Australia, but studies evaluated by the World Health Organization are reported as negative.

Reproductive and developmental effects: A 3-generation reproduction study in rats did not show any evidence of effects on reproductive parameters or foetal development. No specific developmental toxicity study was available.

Poisons Schedule: Propanil is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.7 mg/L for propanil was determined as follows:

$$0.7 \text{ mg/L} = \frac{20 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 20 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for propanil. The following reason is given: "Although a health-based value for propanil can be derived, this has not been done, because propanil is readily transformed into metabolites that are more toxic. Therefore, a guideline value for the parent compound is considered inappropriate, and there are inadequate data on the metabolites to allow the derivation of a guideline value for them. Authorities should consider the possible presence in water of more toxic environmental metabolites." (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Ayranci E, Hoda N (2004). Adsorption of bentazon and propanil from aqueous solutions at the high area activated carbon-cloth. *Chemosphere,* 57(8):755-762.

de la Pena AM, Mahedero MC, Bautista-Sanchez A (2003). Monitoring of phenylurea and propanil herbicides in river water by solid-phase-extraction high performance liquid chromatography with photoinduced-fluorimetric detection. *Talanta,* 60(2-3):279-85.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Kampioti AA, Borba da Cunha AC, Lopez de Alda M, Barcelo D (2005). Fully automated multianalyte determination of different classes of pesticides, at picogram per litre levels in water, by on-line solid-phase extraction-liquid chromatography-electrospray-tandem mass spectrometry. *Analytical and Bioanalytical Chemistry,* 382(8):1815-25.

NOTE: Important general information is contained in PART II, Chapter 6

Kuster M, Lopez de Alda MJ, Barata C, Raldua D, Barcelo D (2008). Analysis of 17 polar to semi-polar pesticides in the Ebro river delta during the main growing season of rice by automated on-line solid-phase extraction-liquid chromatography-tandem mass spectrometry. *Talanta,* 75(2):390-401.

Li Y, George JE, McCarty CL, Wendelken SC (2006). Compliance analysis of phenylurea and related compounds in drinking water by liquid chromatography/electrospray ionization/mass spectrometry coupled with solid-phase extraction. *Journal of Chromatography A,* 1134(1-2):170-6.

Natangelo M, Tavazzi S, Fanelli R, Benfenati E (1999). Analysis of some pesticides in water samples using solid-phase microextraction-gas chromatography with different mass spectrometric techniques. *Journal of Chromatography A,* 859(2):193-201

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Oliver D, Kookana R (2005). Pesticide use in the 6th Creek Sub-catchment, Mt. Lofty Ranges, S.A. and Assessment of Risk of Off-site MOVEMENT using Pesticide Impact Rating Index (PIRI). Adelaide, CSIRO Land and Water Technical Report

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Tschmelak J, Proll G, Gauglitz G (2004). Ultra-sensitive fully automated immunoassay for detection of propanil in aqueous samples: steps of progress toward sub-nanogram per liter detection. *Analytical and Bioanalytical Chemistry,* 379(7-8):1004-12.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Propargite

(endorsed 2011)

GUIDELINE

Based on human health concerns, propargite in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Propargite (CAS 2312-35-8) is a sulfite ester acaricide. There are no other pesticides in this chemical class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propargite would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propargite is an acaricide (miticide), used on a wide variety of food crops, ornamentals and cotton for the control of mites.

There are registered products that contain propargite in Australia. The products are for professional use. Some are emulsifiable concentrates used on cotton, to be diluted and typically applied by ground rig. Others are a water-dispersible granule formulation used on various fruits and vegetable crops and ornamentals. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to propargite and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of propargite may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Propargite was detected in 2000 at Carole Creek, at Mungindi Road bridge, New South Wales, at a concentration of 1.10 µg/L (Muschal 2001).

TREATMENT OF DRINKING WATER

Measured removal of propargite by coagulation–flocculation during drinking water treatment ranged from less than 1% to 17% of the initial concentration (Ballard and Mackay 2005).

MEASUREMENT

Propargite can be extracted from water by liquid/liquid extraction with dichloromethane. The extract is then dried with sodium sulfate, concentrated, and analysed by gas chromatography–mass spectrometry (GC-MS) in selected ion monitoring mode (SIM). The method can achieve a limit of quantitation (LOQ) of 15 µg/L (Yu *et al*. 1997). Akhtar (1988) achieved a LOQ for propargite in groundwater of 0.1 µg/L using GC-MS-SIM. A multi-residue analysis of the pesticides involving extraction and clean-up using gel permeation chromatography and solid-phase extraction (SPE), and subsequent identification and quantification by GC-MS can achieve a LOQ of 2.5 µg/mL for propargite (Huang *et al*. 2007). SPE and gas chromatography ion-trap mass spectrometry can achieve a LOQ of 0.05 µg/L (Deger *et al*. 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propargite is 0.002 mg per kg of bodyweight (mg/kg bw), based on a NOEL of 2 mg/kg bw/day from a long-term dietary study in rats. The NOEL is based on proliferation of cells in the small intestine (increased jejunal smooth muscle cells). The ADI was established in 1999 and incorporates a safety factor of 1000. The additional 10-fold safety factor was applied to address the uncertainty due to the narrow margin between the NOEL and the dose level at which jejunal tumours were observed (3 mg/kg bw/day).

The first ADI of 0.2 mg/kg bw was set in 1988 and was based on a NOEL of 22.5 mg/kg bw/day in a long-term (2-year) dietary study in dogs. This ADI was revised in 1992 to 0.02 mg/kg bw, based on a NOEL of 2 mg/kg bw/day in a rabbit developmental study.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Propargite is partially absorbed from the gastrointestinal tract, extensively metabolised, and distributed evenly in the tissues. Excretion is approximately equal between urine and faeces, with biliary excretion contributing to the faecal excretion. Little unchanged propargite was found in the bile or plasma.

Acute effects: Propargite has low to moderate acute oral toxicity and low dermal toxicity. It is a skin sensitiser in guinea pigs.

Short-term effects: A 90-day dietary study in rats reported clinical signs of toxicity, reduced bodyweight gain and haematological and clinical chemistry changes at 50 mg/kg bw and above.

A 13-week dietary study in dogs reported decreased food consumption, reduced bodyweight gain, increased pigmentation in the reticuloendothelial cells in the liver and increased haemosiderin deposits in the spleen at 50 mg/kg bw/day and above.

Long-term effects: A 2-year dietary study in rats reported a moderate decrease in bodyweight gain, clinical chemistry changes and relative liver and kidney weight changes at 19 mg/kg bw/day. There was also a dose-related increase in undifferentiated sarcomas of the jejunum, often associated with ulceration, at a dose level of 3 mg/kg bw/day and above in females and 19 mg/kg bw/day and above in males. In some animals, there were metastases to the mesentery and lungs. A more detailed long-term study examining the changes in the jejunum in rats showed significant cell proliferation and increased jejunal mass at 14 mg/kg bw/day in males and 21 mg/kg bw/day in females. The NOEL for cell proliferation was 2 mg/kg bw/day and this is the basis of the ADI.

An 18-month study in mice reported no increase in jejunal tumours at the highest dose level of 150 mg/kg bw/day.

NOTE: Important general information is contained in PART II, Chapter 6

Carcinogenicity: There was evidence of carcinogenicity in rats but not mice. In rats, sarcomas of the jejunum are likely to be the result of increased cell proliferation. This effect occurred at dose levels well in excess of the likely level of human exposure to propargite.

Genotoxicity: Propargite is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Propargite is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for propargite was determined as follows:

$$0.007 \text{ mg/L} = \frac{2.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 2.0 mg/kg bw/day is the NOEL based on a long-term (20-month) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation, 10 for intraspecies variation, and 10 for the narrow margin between the NOEL for cell proliferation (2 mg/kg bw/day) and the dose at which tumours occurred (3 mg/kg bw/day) in rats.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Akhtar M (1988). Determination of Propargite and its Major Product Omite Glycol Ether in Ground Water. UNIROYAL: 8774, File No. OMIT- 83.

Ballard BD, MacKay AA (2005). Estimating the removal of anthropogenic organic chemicals from raw drinking water by coagulation flocculation. Journal of Environmental Engineering, 131(1):108-118.

Deger AB, Gremm TJ, Frimmel FH (2000). Problems and solutions in pesticide analysis using solid-phase extraction (SPE) and gas chromatography ion-trap mass spectrometry detection (GC-MS). *Acta Hydrochimica et Hydrobiologica,* 28(6):292-299.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Huang Z, Li Y, Chen B, Yao S (2007). Simultaneous determination of 102 pesticide residues in Chinese teas by gas chromatography-mass spectrometry. *Journal of Chromatography B,* 853(1-2):154-62.

NOTE: Important general information is contained in PART II, Chapter 6

Muschal M (2001). *Central and North West Regions' Water Quality Program. 1999-2000 Report on pesticides monitoring*. Parramatta, NSW Centre for Natural Resources , Department of Land and Concervation

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Yu L, Schoen R, Dunkin A, Firman M, Cushman H (1997). Rapid identification and quantitation of diphenylamine, o-phenylphenol, and propargite pesticide residues on apples by gas chromatography/ mass spectrometry. *Journal of Agricultural and FoodChemistry,* 45(3): 748–752.

NOTE: Important general information is contained in PART II, Chapter 6

Propazine

(endorsed 2011)

GUIDELINE

Based on human health concerns, propazine in drinking water should not exceed 0.05 mg/L.

RELATED CHEMICALS

Propazine (CAS 139-40-2) belongs to the triazine class of chemicals. There are many pesticides in this class, including atrazine, symazine and cyanazine (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propazine would not be a health concern unless the concentration exceeded 0.05 mg/L. Minor excursions above this level would need to occur over a relatively long period to be a health concern, as the health-based guideline is based on medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propazine is a herbicide used for the control of broad-leaf weeds in agricultural crops.

There are currently no products containing propazine registered in Australia, but de-registered conpounds may still be detected in water.

Exposure sources: The main source of public exposure to propazine and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of propazine may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of propazine in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

Relatively high removal rates of propazine have been achieved using conventional flocculation, adsorption onto activated carbon and ozonoation (Ormad *et al.* 2008). More research into the reliable removal of propazine is recommended. If propazine is detected, jar testing with a matrix of multiple oxidants, adsorbents, and coagulants is recommended.

MEASUREMENT

Analysis for propazine in drinking water now typically employs solvent extraction or solid phase extraction with gas chromatography-mass spectrometry determination (QHFSS, 2009 pers comm). Limits of detection are normally 10 ng/L using this methodology.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propazine is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.5 mg/kg bw/day from a medium-term (90-day) dog study. The NOEL is based on reduced bodyweight gain. The ADI incorporates a safety factor of 100, and was established in 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: No metabolism studies are available for propazine.

Acute effects: Propazine has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea pig.

Short-term effects: Medium-term (3-month) dietary studies have been conducted in rats and dogs. Decreased bodyweight gain and food consumption was observed in rats at doses of 14 mg/kg bw/day and above, and in dogs at doses of 6 mg/kg bw/day and above. At higher doses, effects on biochemical and haematological parameters as well as changes in urinalysis and histopathology, and increased mortality were reported in both rats and dogs. The lowest overall NOEL was 1.5 mg/kg bw/day based on decreased bodyweight gain in dogs, and this is the basis for the current ADI.

Long-term effects: In long-term studies in mice and rats, the most sensitive toxicological effect was decreased bodyweight gain and food consumption. A long-term dietary study in mice reported decreased food consumption and a slight increase in mortality at 480 mg/kg bw/day and above. In a 2-year rat study, decreased bodyweight gain and food consumption were observed at doses of 60 mg/kg bw/day and above. The NOEL for this study was 6 mg/kg bw/day.

Carcinogenicity: There was some evidence of carcinogenicity in rats; however, the mode of action is not considered relevant to humans at the normal levels of human exposure.

Genotoxicity: Only short-term *in vitro* studies are available; these report no evidence that propazine is genotoxic.

Reproductive and developmental effects: A 3-generation reproduction study in rats and developmental studies in rats did not produce any evidence of effects on reproductive parameters or on the developing foetus.

Poisons Schedule: Propazine is not included in the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.05 mg/L for propazine was determined as follows:

$$0.05 \text{ mg/L} = \frac{1.5 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 1.5 mg/kg bw/day is the NOEL based on a short-term (3-month) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71: 97–106.

QHFSS (Queensland Health Forensic and Scientific Services) (2009). Personal communication.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Propiconazole

(endorsed 2011)

GUIDELINE

Based on human health concerns, propiconazole in drinking water should not exceed 0.1 mg/L.

RELATED CHEMICALS

Propiconazole (CAS 60207-90-1) is in the DMI: triazole class of chemicals. Other pesticides in this class include flutriafol, myclobutanil, paclobutrazol, and triadimefon (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propiconazole would not be a health concern unless the concentration exceeded 0.1 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propiconazole is a broad-spectrum fungicide used to control a variety of fungi in food crops, and as turf and timber treatments.

There are registered products containing propiconazole in Australia. The products are intended for professional use only and are available as emulsifiable concentrate formulations, to be diluted and applied using ground or aerial spray methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to propiconazole and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of propiconazole in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of propiconazole in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Propiconazole can be measured in drinking water by the use of high performance liquid chromatography–tandem mass spectrometry. The limit of detection for this approach has been reported as 0.05 ng/L (Van De Steene and Lambert 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propiconazole is 0.04 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 4.0 mg/kg bw/day from a 2-year dietary study in rats. The NOEL is based on decreased food consumption and decreased bodyweight gain. The ADI incorporates a safety factor of 100 and was established in 1983.

The previous *Australian Drinking Water Guidelines* health value was 0.0001 mg/L and previous health value was 0.1 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Propiconazole is rapidly and extensively absorbed via the gastrointestinal tract and widely distributed in various tissues. It is readily metabolised and excreted in faeces and urine within 144 hours (97%), and has low potential for bioaccumulation. The major metabolites of propiconazole are triazole derivatives of the parent compound and include 1,2,4-triazole, triazole alanine and triazole acetic acid.

Acute effects: Propiconazole has low oral and dermal acute toxicity. It is not a skin sensitiser.

Short-term effects: In medium-term dietary studies in rats, there was reduced bodyweight gain at 80 mg/kg bw/day. In medium-term studies in dogs, there were no adverse effects at dose levels up to 36 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice and rats. In mice, there were increased liver weights and associated changes in blood plasma enzymes at 50 mg/kg bw/day. In rats, there was reduced bodyweight gain at 20 mg/kg bw/day.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for propiconazole.

Genotoxicity: Propiconazole is not considered to be genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproduction and developmental effects: Two 3-generation reproduction studies in rats produced decreased bodyweight gain at high dose levels only. There was no evidence of effects on reproductive parameters. In developmental studies in rats and rabbits, there was no evidence of effects on foetal development in the absence of maternal toxicity.

Poisons Schedule: Propiconazole is included in Schedule 5 or 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.1 mg/L for propiconazole was determined as follows:

$$0.1 \text{ mg/L} = \frac{4 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 4 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Van De Steene JC, Lambert WE (2008). Validation of a solid-phase extraction and liquid chromatography-electrospray tandem mass spectrometric method for the determination of nine basic pharmaceuticals in wastewater and surface water samples. *Journal of Chromatography A,* 1182(2):153-160.

Propyzamide

(endorsed 2011)

GUIDELINE

Based on human health concerns, propyzamide in drinking water should not exceed 0.07 mg/L.

RELATED CHEMICALS

Propyzamide (CAS 23950-58-5) belongs to the benzamide class of chemicals. Another herbicide in this class is isoxaben (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, propyzamide would not be a health concern unless the concentration exceeded 0.07 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Propyzamide is a pre- and post-emergent herbicide for the control of certain grasses and broad-leaf weeds in sports turf and grazing pastures, home garden lawns, and agricultural legume and lettuce crops.

There are registered products that contain propyzamide in Australia. The products are intended for professional and home garden use, and are available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays to soil or established crops. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to propyzamide and its metabolites are residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of propyzamide may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of propyzamide in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of propyzamide in drinking water have been identified.

MEASUREMENT

Analysis of propyzamide in drinking water may be undertaken by high performance liquid chromatography–mass spectrometry (HPLC-MS). The analytical detection limit for this method is 8 ng/L (Di Corcia *et al.* 2000).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for propyzamide is 0.02 mg per kg bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.9 mg/kg bw/day from a long-term (2-year) dietary study in mice. The NOEL is based on evidence of significant liver damage and an increased incidence of hepatocellular tumours. The ADI incorporates a safety factor of 100, and was first established in 1994. The safety factor does not include the evidence that the compound is a carcinogen for several organs.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Propyzamide is readily absorbed via the gastrointestinal tract and extensively metabolised. The major metabolite is chloromethylbutane. Excretion occurred evenly between faeces (as metabolites and unabsorbed compound) and urine (as metabolites) and is complete within 6 days.

Acute effects: Propyzamide has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 4-week dietary studies in rats and dogs, there was slight hepatocellular hypertrophy and increased absolute and relative liver weight at 37 mg/kg bw/day in rats and 62 mg/kg bw/day in dogs. Other changes observed in dogs were also indicative of liver damage.

In 3-month dietary studies in rats and dogs, increased absolute liver weights were seen at doses of 7.5 mg/kg bw/day and above in rats. Other effects, including reversible hormonal effects, indicative of thyroid toxicity, were observed 50 mg/kg bw/day in rats.

Long-term effects: Two-year dietary studies were conducted in mice, rats and dogs. In mice, there was increased liver weight, evidence of bile duct obstruction associated with necrosis, and an increased incidence of liver carcinomas at 10 mg/kg bw/day. In rats, there was an increased incidence of thyroid adenocarcinoma, testicular adenomas and ovarian hyperplasia at 50 mg/kg bw/day. In dogs, there was an increase in kidney and heart weights, and decreased spleen weights (all without histological changes) at 7.5 mg/kg bw/day, and liver hypertrophy at 35 mg/kg bw/day. The lowest NOEL was 1.9 mg/kg bw/day in mice, and this is the basis of the current ADI.

Carcinogenicity: Propyzamide is associated with an increased tumour incidence in rodents, however, the tumours are considered to be rodent-specific or resulting from regenerative hyperplasia or hormonal changes, and not relevant to humans at the likely levels of exposure to propyzamide.

Genotoxicity: Propyzamide is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two- and 3-generation reproductive studies in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Endocrine effects: Special studies on thyroid function and endocrine regulation in the testes found tumour production was secondary to increased thyroxine turnover and perturbation of the pituitary-testicular endocrine axis, respectively.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Propyzamide is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.07 mg/L for propyzamide was determined as follows:

$$0.07 \text{ mg/L} = \frac{1.9 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.9 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Di Corcia A, Nazzari M, Rao R, Samperi R, Sebastiani E (2000). Simultaneous determination of acidic and non-acidic pesticides in natural waters by liquid chromatography-mass spectrometry. *Journal of Chromatography A,* 878(1):87-98.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Pyrasulfotole

(endorsed 2011)

GUIDELINE

Based on human health concerns, pyrasulfotole in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Pyrasulfotole (CAS 365400-11-9) belongs to the benzoylpyrazole class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pyrasulfotole would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pyrasulfotole is a herbicide used on cereal grains including wheat, barley, oats, rye and triticale.

There is at least one registered product containing pyrasulfotole in Australia. Pyrasulfotole products are intended for professional use only and is applied using ground boom apparatus. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to pyrasulfotole is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of pyrasulfotole may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of pyrasulfotole in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of pyrasulfotole in drinking water have been identified.

MEASUREMENT

No methods have been identified for the analysis of pyrasulfotole in drinking water.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pyrasulfotole is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a long-term (2-year) study in rats. This NOEL is based on corneal and retinal lesions, and centrilobular hepatocellular hypertrophy at 10 mg/kg bw/day. The ADI incorporates a safety factor of 100, and was established in 2007.

The acute reference dose (ARfD) of 0.2 mg/kg bw/day was established in 2007, based on a LOEL of 200 mg/kg bw/day for clinical signs of toxicity. The ARfD incorporates a safety factor of 1000.

An *Australian Drinking Water Guidelines* health value has not been previously established for pyrasulfotole.

HEALTH CONSIDERATIONS

Metabolism: Pyrasulfotole is readily absorbed via the gastrointestinal tract, and approximately 60% is excreted in the urine within 6 hours, mainly unchanged. There is also excretion in the faeces, partially via the bile. By 48 hours, there is <2% remaining in the body, indicating a low potential for bioaccumulation. The main metabolite is a desmethyl derivative.

Acute effects: Pyrasulfotole has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 28-day studies, there were histopathological changes in the urinary bladder at 961 mg/kg bw/day in mice, and increased serum triglyceride levels and elevated liver weights at 171 mg/kg bw/day in dogs. In a 90-day study in rats, increased liver weight, corneal effects and microscopic renal abnormalities were reported at 77 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, an increased incidence of gallstones was observed at 14 mg/kg bw/day. A 2-year study in rats reported corneal and retinal lesions, increased liver weight, centrilobular hepatocellular hypertrophy and elevated plasma cholesterol at 10 mg/kg bw/day. In dogs, tubular dilatation of the kidneys was seen at 101 mg/kg bw/day. The NOEL of 1 mg/kg bw/day in the rat study is the basis for the current ADI.

Carcinogenicity: Pyrasulfotole caused an increased incidence of neoplasms in the urinary tract of mice and in the eye of rats at high dose levels only. These dose levels were considered to be well in excess of the normal levels of human exposure.

Genotoxicity: Pyrasulfotole is not considered genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In multigeneration reproduction studies in rats, there was no evidence of effects on reproductive parameters. In developmental studies in rabbits, there was evidence of foetotoxicity in the absence of maternal toxicity at 10 mg/kg bw/day.

Poisons Schedule: Pyrasulfotole is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for pyrasulfotole was determined as follows:

$$0.04 \text{ mg/L} = \frac{1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1 mg/kg bw/day is the NOEL from a long-term (2-year) rat study.
- 70 kg is taken as the average weight of an adult.

NOTE: Important general information is contained in PART II, Chapter 6

- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Pyrazophos

(endorsed 2011)

GUIDELINE

Based on human health concerns, pyrazophos in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Pyrazophos (CAS 13457-18-6) belongs to the phosphorothiolate class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pyrazophos would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pyrazophos is a fungicide for the control of powdery mildew in vegetable crops.

There are no registered products containing pyrazophos in Australia, but de-registered compounds may still be detected in water. When used previously, products containing pyrazophos were available as concentrated solutions and applied in diluted form using ground and aerial sprays.

Exposure sources: The main source of public exposure to pyrazophos, if used in the future, would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on pyrazophos occurrence in Australian drinking water supplies were found.

TREATMENT OF DRINKING WATER

Activated carbon can be added in a powder form to the coagulation processes or in a granular form as part of the filtration process for the removal of pyrazophos. Effective removal of organophosphorous pesticides has also been observed with softening, disinfection, membrane treatments, and in some cases air-stripping (USEPA 2001).

MEASUREMENT

Pyrazophos can be extracted from the water by liquid/liquid extraction with dichloromethane. The extract is dried with sodium sulfate, concentrated, and analysed by gas chromatography coupled with a nitrogen phosphorus detector and flame photometric detector. The method can achieve a limit of quantitation (LOQ) of 0.05 µg/L. A fully automated at-line solid-phase extraction–gas chromatography procedure can achieve a LOQ of 0.05 µg/L (Hankemeier *et al*. 1996). Solid-phase microextraction with high resolution gas chromatography and mass spectrometry can achieve a LOQ of 10 ng/mL (Souza *et al*. 2003).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pyrazophos is 0.007 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.07 mg/kg bw/day from a 10-day oral study in humans. The NOEL is based on headaches and decreased plasma cholinesterase activity at 0.15 mg/kg bw/day. The ADI incorporates a safety factor of 10, and was first established in 1991.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Pyrazophos is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, primarily through sulfoxidation to form organophosphate compounds. Excretion is rapid, being almost complete by 72 hours and proceeding through urine.

Acute effects: Pyrazophos has moderate acute oral toxicity and low acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: In a 28-day dietary study in rats, there was serum cholinesterase inhibition at 0.25 mg/kg bw/day, and erythrocyte cholinesterase inhibition at 0.75 mg/kg bw/day. In a 10-day oral study in humans, plasma cholinesterase activity was decreased at 0.15 mg/kg bw/day. The NOEL was 0.07 mg/kg bw/day and this is the basis of the ADI.

Three-month dietary studies were conducted in rats and dogs. In rats, there was decreased erythrocyte cholinesterase activity at 0.48 mg/kg bw/day and an increase in absolute adrenal and spleen weights at 8 mg/kg bw/day. In dogs, there was decreased erythrocyte and plasma cholinesterase activity at 0.25 mg/kg bw/day and decreased physical activity and anaemia at 16 mg/kg bw/day.

Long-term effects: Long-term dietary studies in mice, rats and dogs showed decreased plasma and erythrocyte cholinesterase activity at 0.7 mg/kg bw/day in mice, 1 mg/kg bw/day in rats, and 0.25 mg/kg bw/day in dogs.

Carcinogenicity: Based on a 2-year study in rats and an 18-month study in mice, there is no evidence of carcinogenicity for pyrazophos.

Genotoxicity: Pyrazophos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and development studies in rats and rabbits did not produce any evidence for effects on reproductive parameters or foetal development.

Neurotoxicity: A delayed neurotoxicity study in hens using doses up to 150 mg/kg bw found no evidence for delayed neurotoxicity from pyrazophos.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Pyrazophos is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for pyrazophos was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.07 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.07 mg/kg bw/day is the NOEL based on a short-term (10-day) oral dosing study in human volunteers.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from a study in humans, to allow for variation of response within the human population.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Hankemeier T, Steketee E, Vreuls JJ, Brinkman UAT (1996). Automated at-line solid-phase extraction-gas chromatographic analysis of micropollutants in water using the PrepStation. *Journal of Chromatography A,* 750:161-174.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Souza DA, Lancas FM (2003). Solventless sample preparation for pesticides analysis in environmental water samples using solid-phase microextraction-high resolution gas chromatography/mass spectrometry (SPME-HRGC/MS). *Journal of Environmental Science and Health B,* 38(4):417-28,

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2001). FIFRA Scientific Advisory Panel Meeting. Session VI- A Set of Scientific Issues Being Considered by the Environmental Protection Agency Regarding: Progress Report on Estimating Pesticide Concentrations in Drinking Water and Assessing Water Treatment Effects on Pesticide Removal and Transformation: A Consultation. Arlington, Virginia.

NOTE: Important general information is contained in PART II, Chapter 6

Pyroxsulam

(endorsed 2011)

GUIDELINE

Based on human health concerns, pyroxsulam in drinking water should not exceed 4 mg/L.

RELATED CHEMICALS

Pyroxsulam (CAS 422556-08-9) is in the triazolopyrimidine class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, pyroxsulam would not be a health concern unless the concentration exceeded 4 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Pyroxsulam is a post-emergence herbicide used to control a wide range of grass and broad-leaf weeds in wheat.

There is at least one registered product containing pyroxsulam in Australia. Pyroxsulam products are for professional use and are available as an oil-dispersible liquid to be applied by ground boom spray for one post-emergent (early season) application to wheat. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to pyroxsulam is residues in food. Residue levels in food produced according to good agricultural practice are generally low

Agricultural use may potentially lead to residues in source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of pyroxsulam in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of pyroxsulam in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

No suitable analytical techniques have been identified, but the use of high performance liquid chromatography–tandem mass spectrometry is expected to be suitable for residue levels of this pesticide in water.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for pyroxsulam is 1.0 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 100 mg/kg bw/day from an 18-month dietary study in mice. The NOEL is based on effects on the liver. The ADI incorporates a safety factor of 100, and was established in 2008.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Pyroxsulam is rapidly absorbed from the gastrointestinal tract. It is rapidly eliminated, mainly in the urine, largely unchanged. It has low potential for bioaccumulation. The primary metabolite of pyroxsulam is 2'-demethyl-pyroxsulam.

Acute effects: Pyroxsulam has low to moderate oral acute toxicity, and low dermal toxicity. It is a skin sensitiser in guinea pigs.

Short-term effects: Medium-term dietary studies were conducted in mice, rats and dogs. The only effect observed was an increase in serum cholesterol levels at 1000 mg/kg bw/day; however, these returned to normal after cessation of treatment and were likely be adaptive and non-adverse.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, there was an increase in liver weight at the highest dose, 1000 mg/kg bw/day. No adverse effects were observed in rat and dog studies.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for pyroxsulam.

Genotoxicity: Pyroxsulam is not considered to be genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproductive and developmental effects: In a 2-generation reproduction study in rats and developmental studies in rats and rabbits, there was no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Pyroxsulam is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 4 mg/L for pyroxsulam was determined as follows:

$$4 \text{ mg/L} = \frac{100 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 100 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in mice.
- 70 kg is taken as the average weight of an adult.

NOTE: Important general information is contained in PART II, Chapter 6

FACT SHEETS

- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Quintozene

(endorsed 2011)

GUIDELINE

Based on human health concerns, quintozene in drinking water should not exceed 0.03 mg/L.

RELATED CHEMICALS

Quintozene (CAS 82-68-8) belongs to the nitroaniline class of chemicals. There are no other registered pesticides in this chemical class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, quintozene would not be a health concern unless the concentration exceeded 0.03 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Quintozene is a soil fungicide for the control of pathogenic fungi on turf, ornamentals, cotton seedlings, peanuts, and vegetable agricultural crops.

There are registered products that contain quintozene in Australia. The products are intended for professional use and are available as concentrated solutions to be applied directly to soil in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to quintozene and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of quintozene may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of quintozene in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of quintozene in drinking water have been identified.

MEASUREMENT

No suitable techniques for the analysis of quintozene in drinking water have been identified. However, it is expected that a suitable method using high performance liquid chromatography–tandem mass spectrometry could be developed if required.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for quintozene is 0.007 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.7 mg/kg bw/day from a long-term (2-year) dietary study in dogs. The NOEL is based on evidence of mild liver toxicity (increased absolute and relative liver weights, hepatocyte enlargement and granulosis, and increased serum alkaline phosphatase and cholesterol). The ADI incorporates a safety factor of 100, and was first established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Quintozene is readily absorbed from the gastrointestinal tract of rats. The compound is extensively metabolised, mostly via nitrogen reduction to form acetyl pentachlorophenyl cysteine and pentachloroaniline. Excretion is rapid, occurring mostly via urine and being complete by three days, with small amounts of unabsorbed parent compound also excreted in faeces.

Acute effects: Quintozene has low acute oral and dermal toxicity. It is a skin sensitiser in humans.

Short-term effects: A 28-day dietary study in dogs reported increased relative liver weights at a dose of 64 mg/kg bw/day, but with no histological examination of the liver. Medium-term dietary studies in rats and dogs did not show any evidence of toxicity up to 100 mg/kg bw/day in rats and up to 25 mg/kg bw/day in dogs.

Long-term effects: In long-term studies in rats (2-year) and dogs (1- and 2-year), there was decreased bodyweight gain and increased liver and kidney weight in both species at 6 mg/kg bw/day. There was also evidence of liver toxicity in rats (necrosis) at this dose level. In dogs, there was increased serum alkaline phosphatase, serum cholesterol, hepatocyte size and granulosis at 40 mg/kg bw/day. The lowest overall NOEL was 0.7 mg/kg bw/day in dogs.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for quintozene.

Genotoxicity: Quintozene is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a 2-generation reproduction study in rats, and in developmental studies in rats, mice and rabbits, there was no evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Quintozene is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.03 mg/L for quintozene was determined as follows:

$$0.03 \text{ mg/L} = \frac{0.7 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.7 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has not established a health-based guideline value for quintozene and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Radionuclides (Other beta- and gamma-emitting)

(endorsed 2001)

GUIDELINE

No specific guideline values are set for beta- or gamma-emitting radionuclides.

Specific beta- or gamma-emitting radionuclides should be identified and determined only if gross beta radioactivity (after subtracting the contribution of potassium-40) exceeds 0.5 Bq/L (27.6 Bq of beta activity per gram of stable potassium).

GENERAL DESCRIPTION

Several radionuclides that are classified as beta-particle or gamma-ray emitters may occasionally be present in drinking water. The significant long-lived nuclides in this group are the naturally occurring isotopes potassium-40, lead-210 and radium-228, and artificial radionuclides caesium-137 and strontium-90. Tritium, another nuclide in this group, is present in the environment both from natural sources and as a result of nuclear fall-out and nuclear power generation.

Levels of strontium-90 and caesium-137 in the Australian environment have decreased substantially since atmospheric testing of nuclear weapons ceased, and these radionuclides are not detectable in drinking water. In the absence of a nuclear power industry in Australia, these nuclides are likely to be present in significant concentrations in drinking water only as a result of transient contamination following an event such as a nuclear accident.

Potassium-40 occurs naturally in a fixed ratio to stable potassium. Potassium is an essential element for humans, and is absorbed mainly from ingested food. Potassium-40 does not accumulate in the body but is maintained at a constant level independent of intake. The average concentration of potassium in an adult male is about 2 g/kg of bodyweight, which gives an activity mass concentration of potassium-40 of 60 Bq per kg of bodyweight. The corresponding value for females is slightly less.

Lead-210, like radium-226, is a decay product of the uranium-238 series. Food is the most important route by which lead-210 enters the human body, and the annual intake depends on diet: highest concentrations are found in fish and other aquatic species. Generally, lead-210 concentrations in drinking water are considerably less than concentrations of either radium-226 or radium-228.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Concentrations of potassium-40 in Australian drinking water supplies vary widely, from below 0.05 Bq/L in surface water sources to more that 1 Bq/L in some supplies drawn from groundwater.

There are only limited data on concentrations of other beta- or gamma-emitting radionuclides such as lead-210, strontium-90 and caesium-137 in Australian drinking water supplies. Lead-210 concentrations are probably below 0.05 Bq/L and concentrations of artificial radionuclides are negligible.

TREATMENT OF DRINKING WATER

Treatment processes involving ion exchange or reverse osmosis will effectively remove radionuclides such as lead-210, strontium-90 and caesium-137. There is no suitable treatment to remove tritium.

ANALYSIS

For initial screening, gross beta activity is determined by evaporation of the sample and beta measurement of the residue (AS 2531 1982, ISO 1991). The limit of determination is approximately 0.02 Bq/L, but will vary with the mass of residue.

For potassium-40, the most suitable method is to determine the stable potassium concentration by atomic absorption spectrophotometry. The beta activity due to potassium-40 is then calculated using the ratio of 27.6 Bq of beta activity per gram of stable potassium.

Specific determination of lead-210 and strontium-90 in drinking water is carried out by beta counting after radiochemical isolation of the radionuclide (EML 1990, USEPA 1980). The limit of determination for each nuclide is approximately 0.02 Bq/L by this method. High-resolution gamma spectrometry is the most suitable method for the determination of caesium-137.

Tritium is determined by distillation and liquid scintillation counting (ISO 1989).

HEALTH CONSIDERATIONS

Lead-210 can concentrate in bone, where it remains for many years. The radiation dose from lead-210 is due mainly to the emission of alpha particles from its progeny, polonium-210.

In principle, lead-210 may increase the risk of bone cancers; however, no link has been demonstrated, either in animal studies or epidemiological studies.

Much of what is known of the health effects of ingested strontium-90 and caesium-137 comes from animal studies. Caesium-137, when ingested, is distributed throughout the body, mainly to soft tissues. The organ most at risk is the liver. Dogs exposed to high concentrations of caesium-137 showed an increased incidence of liver cancer. The risks of bone cancer have been estimated from extensive life-span studies of dogs injected intravenously with strontium-90. The studies showed the dose-response relationship to be non-linear for chronic exposure to strontium-90.

Potassium-40 is not considered to be of significance to health because it is present naturally with the stable potassium isotope. The average contribution of this nuclide to the annual effective dose from background radiation is estimated to be 0.18 mSv (UNSCEAR 2000).

ESTIMATION OF DOSE

The dose from beta- or gamma-emitting radioisotopes should be estimated using the method described in Section 7.5.

REFERENCES

AS 2531 (1982). Australian Standard for Waters – determination of gross alpha and gross beta activities. Standards Association of Australia, Sydney, New South Wales.

EML (1990). EML Procedures Manual, 27th edition. Environmental Measurements Laboratory, Department of Energy, New York, United States.

ISO (International Organization for Standardization) (1989). Water quality – determination of tritium activity concentration – liquid scintillation counting method. ISO, International Standard ISO 9698:1989(E), Geneva, Switzerland.

ISO (International Organization for Standardization) (1991). Water quality – measurement of gross beta activity in non-saline water – Thick source method. ISO, International Standard ISO 9695, Geneva, Switzerland.

UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (2000). Sources, effects and risks of ionising radiation. UNSCEAR, report ISBN 92-1-142143-8, New York.

USEPA (United States Environmental Protection Agency) (1980). Prescribed procedures for measurement of radioactivity in drinking water. USEPA, Environmental Monitoring and Support Laboratory, EPA-600/4-80-032, Cincinnati, United States.

NOTE: Important general information is contained in PART II, Chapter 6

Radium-226 and radium-228

(endorsed 2001)

GUIDELINE

Radium-226 and Radium-228 should be determined if the gross alpha radioactivity in drinking water exceeds 0.5 Bq/L, or the gross beta activity (with the contribution of potassium-40 subtracted) exceeds 0.5 Bq/L.

GENERAL DESCRIPTION

Radium isotopes are formed as a result of radioactive decay of uranium-238 and thorium-232, both of which occur naturally in the environment. The two most significant isotopes in this process, in terms of radiological health, are radium-226 (uranium series) and radium-228 (thorium series), which have half-lives of 1620 years and 5.8 years, respectively.

Radium-226 is an alpha emitter. It has been used, separated from its parent uranium, in cancer therapy.

Of the radionuclides that comprise the natural thorium and uranium series, radium-226 and radium-228 are those most likely to be found in drinking water, and this occurs more commonly in supplies derived from groundwater. Concentrations in surface water are likely to be extremely low. Concentrations of radium isotopes in groundwater vary according to the type of aquifer minerals and dissolved anions such as chloride, carbonate, and sulfate anions, which tend to increase the mobility of radium.

Radium is widespread in the environment and trace amounts are found in many foods. The average dietary intake is estimated to be 15 Bq per year (UNSCEAR 2000).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In supplies derived from groundwater sources, radium-226 and radium-228 concentrations vary considerably depending on the aquifer, and it is not uncommon in small supplies to find concentrations up to, or exceeding, 0.5 Bq/L. Radium concentrations in Australian surface water supplies are generally below 0.02 Bq/L.

TREATMENT OF DRINKING WATER

Lime softening, reverse osmosis and ion exchange all remove both radium-226 and radium-228 very efficiently from water. Aeration may be effective in certain circumstances.

ANALYSIS

Generally, analysis for radium isotopes is only required if gross alpha and beta activities exceed 0.5 Bq/L (see Chapter 7).

Radium-226 can be determined by several methods involving radiochemistry, by radon emanation, or by liquid scintillation counting (USEPA 1980, ASTM 1989, Cooper and Wilks 1981, EML 1990, APHA 1992). The estimated limit of determination is 5 mBq/L.

Radiochemical techniques are necessary to determine radium-228 (USEPA 1980). The estimated limit of determination is 20 mBq/L.

HEALTH CONSIDERATIONS

The metabolic behaviour of radium is similar to that of calcium, and an appreciable fraction of ingested radium is deposited in bone tissue, where it is retained for a long time.

High levels of exposure to radium have been shown to be carcinogenic. Epidemiological studies of 2000 radium dial painters, and studies of the medical use of the short-lived isotope radium-224, have both shown an increased incidence of bone sarcomas. Animal experiments have also established an association between radium exposure and bone sarcoma.

Studies of populations in the United States exposed to radium in drinking water have produced no conclusive evidence linking cancer with ingestion of radium.

Apart from cancer, the only other health effect resulting from ingestion of radium observed in the studies of radium dial painters was bone necrosis.

DERIVATION OF GUIDELINE

The dose from radium-226 and radium-228 should be estimated using the method described in Section 7.5.

REFERENCES

APHA Method 7500-Ra C (1992). Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

ASTM, Vol. 11.02 (1989). 1989 Annual book of ASTM standards. American Society for Testing and Materials, Philadelphia, United States.

Cooper MB, Wilks MJ (1981). An analytical method for radium (^{226}Ra) in environmental samples by the use of liquid scintillation counting. Australian Radiation Laboratory report, ARL/TR040.

EML (Environmental Measurements Laboratory) (1990). EML Procedures Manual, 27th edition. EML, Department of Energy, New York, United States.

UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (2000). Sources, effects and risks of ionising radiation. UNSCEAR, report ISBN 92-1-142143-8, New York.

USEPA (United States Environmental Protection Agency) (1980). Prescribed procedures for measurement of radioactivity in drinking water. USEPA, Environmental Monitoring and Support Laboratory, EPA-600/4-80-032, Cincinnati, United States.

NOTE: Important general information is contained in PART II, Chapter 6

Radon-222

(endorsed 1996)

GUIDELINE

Based on a consideration of the potential health impact from radon released from tap water to the air inside a dwelling, the activity concentration of radon-222 in drinking water should not exceed 100 Bq/L.

The guideline value applies to the concentration of radon at the point of use of the water, not at the source, because of the significant decrease in concentration which can occur due to radioactive decay during storage, treatment and reticulation.

GENERAL DESCRIPTION

Radon-222 is a radioactive gas produced from the decay of radium-226 in soil and minerals. It has a half-life of 3.8 days.

Elevated concentrations of radon-222 may occur in drinking water derived from groundwater, due to the release of radon from aquifer rocks and minerals, particularly in granitic areas. In Finland, for example, the weighted average radon concentration in drinking water is 25 Bq/L.

Radon concentrations in surface water supplies are very low because the gas is rapidly lost to the atmosphere.

Studies from Canada, Finland and the United States have shown that dissolved radon-222 in drinking water may be released to air during domestic use and contribute to indoor radon concentrations.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

The data on the concentrations of radon-222 in Australian drinking water supplies are limited, but sufficient to indicate that radon may be significant in some rural supplies.

TREATMENT OF DRINKING WATER

The most effective way of eliminating dissolved radon-222 from water is by aeration, either actively by processes such as spraying, or by passive processes such as open-air storage. Radon concentrations will also decrease by radioactive decay in water stored before use.

ANALYSIS

The concentration of radon-222 in drinking water can be determined by liquid scintillation counting of a small volume of water, or by a de-emanation of radon into a Lucas Cell chamber for counting (EPA 1987, EPA 1991). The limits of determination for these methods are around 1–2 Bq/L and 0.1–0.5 Bq/L respectively. The former method is preferable because because it is quicker and easier to use.

HEALTH CONSIDERATIONS

The main health risk from radon arises from inhalation of the gas, particularly when it accumulates inside dwellings. Radon-222 has several short-lived radioactive progeny that can give rise to an increased risk of lung cancer.

Epidemiological studies of underground miners in the uranium mining industry overseas have established a relationship between the incidence of lung cancer and occupational exposure to radon.

No link has been demonstrated, however, in either experimental or epidemiological studies, between ingestion of radon in drinking water and increased cancer rates.

DERIVATION OF GUIDELINE

The guideline value was determined following consideration of these points:

- Ingestion of radon in water does not pose a sufficient risk to health to warrant consideration of this pathway in setting a guideline value (UNSCEAR 2000).
- The main sources of radon in indoor air are the subjacent ground and building materials, with radon in tap water being normally only a small contributor. The release of radon from tap water into household air will depend upon the volume and nature of water usage in the dwelling. The overall radon concentration in air will be influenced by factors such as the construction of the dwelling, ventilation rates and domestic practices.
- Given the indirect nature of the exposure pathway and the number of assumptions that must be made to assess the dose to an individual arising from the inhalation of radon released to household air from tap water, it is not appropriate to use a level of dose as the basis for a guideline value for radon in drinking water.
- The ratio between the radon concentration in tap water and the concentration in air is commonly estimated at 10,000 to 1 (UNSCEAR 2000). On this basis, a concentration of radon in tap water of 100 Bq/L would give rise to a concentration in air of 10 Bq/m^3, which is 5% of the present NHMRC action level for radon in air in a dwelling. A guideline value of 100 Bq/L would ensure that radon in drinking water would not be a significant contributor to indoor radon.

REFERENCES

NHMRC (1990). Report of the 109th Session. National Health and Medical Research Council, May 1990.

UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (2000). Sources, effects and risks of ionising radiation. UNSCEAR, report ISBN 92-1-142143-8, New York.

USEPA (United States Environmental Protection Agency) (1987). Two test procedures for radon in drinking water, inter-laboratory collaborative study. USEPA, EPA-600/2-87-082, Washington, United States.

NOTE: Important general information is contained in PART II, Chapter 6

Selenium

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of selenium in drinking water should not exceed 0.01 mg/L.

GENERAL DESCRIPTION

Selenium and selenium salts are widespread in the environment. Selenium is released from natural and human-made sources, with the main source being the burning of coal. Selenium is also a by-product of the processing of sulfide ores, chiefly in the copper refining industry.

The major use of selenium is in the manufacture of electronic components. It is used in several other industries, and selenium compounds are used in some insecticides, in hair shampoos as an anti-dandruff agent, and as a nutritional feed additive for poultry and livestock.

Selenium concentrations in source waters are generally very low and depend on local geochemistry, pH and the presence of iron salts. Concentrations in drinking water supplies overseas are generally below 0.01 mg/L but groundwater concentrations as high as 6 mg/L have been reported in the United States.

Food is the major source of intake for Australians. Cereal and grain products contribute most to intake, while fish and liver contain the highest selenium concentrations. Average daily intakes for Australian adults are between 0.06 mg and 0.13 mg.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, selenium concentrations are less than 0.005 mg/L. Selenium concentrations in groundwater are not a problem in Australia, as they are in some overseas supplies.

TREATMENT OF DRINKING WATER

Selenium concentrations in drinking water can be reduced by coagulation with ferric chloride and by lime softening. Coagulation with alum is much less effective. Activated alumina absorption is the most effective means of treatment, but only at low pH.

MEASUREMENT

The selenium concentration in drinking water can be determined by hydride generation followed by atomic absorption spectroscopy (APHA Method 3500-Se Part C 1992). The limit of determination is 0.001 mg/L.

HEALTH CONSIDERATIONS

Selenium is an essential element for many species, including humans. Signs of selenium deficiency in humans are not well established but may include a chronic disorder of the heart muscle, other heart diseases and cancer. The Australian recommended dietary intake to maintain health is approximately 0.001 mg/kg body weight per day.

Most water-soluble selenium compounds are effectively absorbed by the gastrointestinal tract. Selenium is then distributed to most organs, with highest concentrations found in the kidney, liver and spleen.

The toxicity of selenium varies considerably among the different selenium compounds. Selenite and selenate are much more toxic than selenium sulfide.

An extensive review and summary of the human and animal toxicity data for selenium is available (IPCS 1987).

There have been a number of reports of ill effects attributed to short- and long-term exposure to selenium; most of these have resulted from occupational exposure or accidental poisoning; acute or chronic nutritional toxicity is comparatively rare. Intakes above about 1 mg/day over prolonged periods may produce nail deformities characteristic of selenosis. Other features of excess selenium intake include nonspecific symptoms such as gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath.

A 2-year study on 140 people with an average selenium intake of 0.24 mg/day reported no effect associated with the level of selenium intake.

Domestic animals developed a symptom known as 'blind staggers' when fed plants that had accumulated selenium. The animals had impaired vision, depressed appetite and a tendency to wander in circles. This led to paralysis and death from respiratory failure.

Except for selenium sulfide, experiments with laboratory animals indicate that selenium compounds are not carcinogenic, with some selenium compounds displaying an anticarcinogenic effect. Results for selenium sulfide indicate that it causes liver and skin tumours in mice.

Tests for mutagenic activity using bacteria have reported both positive and negative results. Studies indicate that selenite can cause chromosome damage to mammalian cells.

The International Agency for Research on Cancer has concluded that selenium is not classifiable as to its carcinogenicity in humans (Group 3, inadequate evidence in humans and in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value for selenium in drinking water was derived as follows:

$$0.01 \text{ mg/L} = \frac{0.24 \text{ mg/day} \times 0.1}{2 \text{ L/day}}$$

where:

- 0.24 mg per day is the acceptable daily intake (Longnecker *et al*. 1991).
- 0.1 is the proportion of daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.

REFERENCES

APHA Method 3500-Se Part C (1992). Selenium: Continuous hydride generation/Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1987). Selenium. Environmental Health Criteria, 58. World Health Organization, IPCS.

Longnecker MP, Taylor PR, Levander OA, Howe M, Veillon C, McAdam PA, Patterson KY, Holden JM, Stampfer MJ, Morris JS, Willet WC (1991). Selenium in diet, blood, and toenails in relation to human health in a seleniferous area. *American Journal of Clinical Nutrition*, 53:1288–94.

NOTE: Important general information is contained in PART II, Chapter 6

Silica

(endorsed 2011)

GUIDELINE

To minimise an undesirable scale build up on surfaces, silica (SiO_2) within drinking waters should not exceed 80 mg/L.

GENERAL DESCRIPTION

Silica present in water is usually referred to as amorphous silica (i.e. lacking any crystalline structure). When silica is dissolved within water it forms monosilicic acid:

$$SiO_2 + 2H_2O \rightarrow Si(OH)_4$$

When the concentrations of monosilicic acid increase, polymerisation of the silica occurs, forming polysilicic acids followed by formation of colloidal silica. Monosilicic acid and polysilicic acids are the forms of silica analysed when determining dissolved silica content.

The deposition of silica from solutions can occur via various mechanisms. The deposition of silica that can cause the most problems for the water industry is via silica's ability to deposit on solid surfaces that have hydroxyl (OH) groups present. Surfaces that commonly have hydroxyl groups present are glass and metallic surfaces. For example, dissolved silica will react with the surfaces of glass and begin to form a white precipitate. The silica forms silicates on the surface, resulting in silica build-up. In cases where customer complaints occur due to scale build-up, water hardness and silica concentrations should be investigated to determine the cause.

Silica can be a problem in water treatment due to its ability to cause fouling of reverse osmosis (RO) membranes (Sheikholeslami and Tan, 1999, Ning 2002, Sahachaiyunta and Sheikholeslami 2002). This occurs when the dissolved silica of the concentrate becomes super-saturated, causing silicates to form in the presence of metals, and these deposit on the membrane surface. The silicate then dehydrates, forming hard layers on the membrane that reduce the effectiveness of the process.

Fouling of membranes can occur in two ways:

- precipitation fouling – monosilicic acid polymerises at the membrane surface forming a deposit similar to silica scale on glass surfaces;
- particulate fouling – accumulation of colloidal silica within the solution is then deposited on the membrane surface.

A suggested industry standard guideline is to limit the concentration of silica within the RO concentrate to ~ 120 mg/L at 25°C to limit fouling of RO membrane (Freeman and Majerle 1995).

Colloidal silica may affect ion-exchange processes in water treatment. The stability of colloidal silica as an un-ionised compound causes problems in removal using ion-exchange resins. High concentrations may also cause fouling of ion exchange units.

TYPICAL VALUES IN AUSTRALIAN RAW WATER

Dissolved silica from various source can range between 0.6 mg/L in some surface waters to 110 mg/L in ground waters.

MEASUREMENT

Silica can be determined by spectrophotometric techniques upon the addition of ammonium molybdate (EPA method 370.1, APHA Method 4500.F-SiO_2) (Clesceri *et a,* 1998).

TREATMENT OF WATER

The removal of silica from waters involves the use of cold lime softeners, hot process softeners, macroreticular anion resin, up-flow filters with chemical feed, cross-flow microfiltration with chemical feed, and nanofiltration.

Emerging processes in water treatment such as electrodialysis removal (EDR) and high efficiency reverse osmosis (HERO), as a by-product, have some success in silica removal. EDR is based on applying electrochemical separation processes within the water that allow for the removal of charged ions from the water. This process may offer some level of silica removal if the silica is in a charged form; however colloidal silica, which is likely to be present at large concentrations, will not be removed due to its stability as an un-ionised compound. HERO is based on normal RO processes; however pre-treatment removes divalent metals that form scale, and the operating pH has been increased to 11 to support the process. At this pH, silica is ionised, which increases its solubility and hence eliminates scaling of the membranes. It has also been reported that, at higher pH values, there is improved rejection of silica.

HEALTH CONSIDERATIONS

No health guideline has been set for silica as there are no data linking silica to adverse health outcomes.

DERIVATION OF GUIDELINES

The suggested guideline is based on the solubility of amorphous silica being between 100 and 140 mg/L at 25°C, so that, based on current date, limiting the value to below 80 mg/L should limit the formation of silica scaling.

REFERENCES

Clesceri LS, Greenberg AE, Eaton AD (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington.

Freeman SDN, Majerle RJ (1995). Silica fouling revisited. *Desalination*, 103:113 – 115.

Ning R (2002). Discussion of silica speciation, fouling, control and maximum reduction. *Desalination*, 151:67-73.

Sahachaiyunta P, Sheikholeslami R (2002). Effect of several inorganic species on silica fouling in RO membranes. *Desalination*, 144: 373-378.

Sheikholeslami R, Tan S (1999). Effects of water quality on silica fouling of desalination plants. *Desalination,* 126:267-280.

NOTE: Important general information is contained in PART II, Chapter 6

Silver

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of silver in drinking water should not exceed 0.1 mg/L.

GENERAL DESCRIPTION

Silver concentrations in natural source waters are generally very low, less than 0.0002 mg/L. In some countries silver and silver salts are used for disinfection and preservation of water, and this can result in higher silver concentrations.

Silver is a precious metal and is used in the production of tableware, jewellery and coins. It is also used in batteries, mirrors, as a chemical catalyst, and as an antiseptic agent.

Traces of silver can be found in most foods. The daily dietary intake has been estimated at between 0.03 mg and 0.09 mg.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for silver.

TREATMENT OF DRINKING WATER

Silver can be readily removed from drinking water by conventional coagulation or lime softening.

MEASUREMENT

The concentration of silver in drinking water can be determined by graphite furnace atomic absorption spectroscopy or inductively coupled plasma emission spectroscopy (APHA Method 3500-Ag Parts B or C 1992). The limits of determination are 0.001 mg/L and 0.01 mg/L respectively.

HEALTH CONSIDERATIONS

Although silver can be found in many biological substances, it is not considered an essential trace element for mammals.

It has been estimated that less than 10% of dietary silver is absorbed by the gastrointestinal tract. Silver is stored mainly in the liver and skin and is capable of binding to amino acids and proteins.

The best-known clinical condition of silver intoxication is argyria, which results in a bluish-grey metallic discolouration of the skin, hair, mucous membranes, mouth and eye. Most cases have been associated with self-administration of silver preparations, or occupational exposure to silver and silver compounds.

Experiments with laboratory rats and mice have reported similar results. Very high concentrations of silver in drinking water (over 600 mg/L) for a lifetime caused discolouration in the thyroid and adrenal glands, the choroids of the brain and eye, and the liver and kidney. Some hypoactive behaviour was also reported.

No data are available on the carcinogenicity of silver. Silver salts are not mutagenic in tests with bacteria, but can induce damage in mammalian DNA.

DERIVATION OF GUIDELINE

The guideline value for silver in drinking water was derived as follows:

$$0.1 \text{ mg/L} = \frac{0.4 \text{ mg/day} \times 0.5}{2 \text{ L/day}}$$

where:

- 0.4 mg/day is derived from a human lifetime no-effect level of 10 g (Hill and Pillsbury 1939).
- 0.5 is the proportion of total daily intake attributable to the consumption of drinking water.
- 2 L/day is the average amount of water consumed by an adult.

No additional safety factors were used, as the calculation was based on a human no-effect level.

It is unlikely that silver concentrations in drinking water would ever reach a concentration that could cause adverse effects. Silver or silver salts should not be used as antimicrobial agents unless no other disinfectants are available.

REFERENCES

APHA Method 3500-Ag Part B (1992). Silver: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Ag Part C (1992). Silver: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

Hill WR, Pillsbury DM (1939). *Argyria, the Pharmacology of Silver*. The Williams and Wilkins Co., Baltimore, Maryland.

Simazine

(endorsed 2011)

GUIDELINE

Based on human health concerns, simazine in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Simazine (CAS 122-34-9) belongs to the triazine class of chemicals. There are many other pesticides in this class, including atrazine and cyanazine (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, simazine would not be a health concern unless the concentration exceeded 0.02 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Simazine is a pre- and post-emergent herbicide for the control of annual grasses and broad-leaf weeds in a range of agricultural crops such as in citrus, pomefruits, grapes, chickpeas and canola crops. It is also used as an algicide in swimming pools.

There are registered products that contain simazine in Australia. The products are intended for professional and home garden use and are available as concentrated solutions to be applied in diluted form using ground and aerial sprays directly onto soil, or added to swimming pools. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to simazine are residues in food and use in swimming pools. Residue levels in food produced according to good agricultural practice are generally low. The concentrations in swimming pools, when the product is used correctly, are also low.

Agricultural use of simazine may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on occurrence of simazine in Australian drinking water could be found. Simazine has been reported at 0.04 µg/L in the Brisbane River (Bengston Nash *et al.* 2006) and at levels as high as 18 µg/L in an agricultural drainage channel in New South Wales (Tran *et al.* 2007).

Levels of 1-2 µg/L have been reported in groundwater in the USA (WHO 2003). Simazine has also been reported in private and public drinking water supplies in Canada, with a maximum concentration of 23 µg/L (Health Canada 1986).

TREATMENT OF DRINKING WATER

Chlorination, activated carbon or ozonation may be only partially effective at removing simazine from drinking waters, depending on precise operational conditions (Ormad *et al.* 2008).

MEASUREMENT

Simazine can be measured by routine gas chromatography–mass spectrometry analysis, with a limit of reporting of 0.01 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for simazine is 0.005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.5 mg/kg bw/day from a long-term (2-year) dietary study in rats. The NOEL is based on decreased survival, decreased bodyweight gain, and evidence of anaemia. The ADI incorporates a safety factor of 100, and was established in 1990.

The previous ADI for simazine was set in 1985 at 0.003 mg/kg bw, based on a NOEL of 6 mg/kg bw/day from a 2-year study in rats and a safety factor of 2000.

The previous *Australian Drinking Water Guidelines* health value was 0.02 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Simazine is readily absorbed via the gastrointestinal tract, and is extensively metabolised via oxidative N-dealkylation to over 20 metabolites. The major metabolite is desethyl-desisopropyl-atrazine. Excretion in urine is rapid and almost complete within 24 hours.

Acute effects: Simazine has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In a 2-week dietary study in rats, there was a dose-dependant increase in oestrous cycle length, as well as an increase in the plasma levels of the hormones prolactin, estradiol, and corticosterone at 5 mg/kg bw/day and above. No other effects were seen up to the highest dose tested of 15 mg/kg bw/day. Thirteen-week dietary studies in dogs reported vomiting, tremors and decreased bodyweight gain at doses of 75 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, no toxic effects were observed up to 100 mg/kg bw/day. In rats, there was decreased survival, decreased bodyweight gain, and evidence of anaemia, as well as early onset of mammary growths symptomatic of premature reproductive senescence, at 5 mg/kg bw/day and above. In dogs, there was decreased bodyweight gain, increases in liver enzymes and increased thyroid weight at 37.5 mg/kg bw/day. The lowest overall NOEL was 0.5 mg/kg bw/day in rats. This NOEL is the basis for the current ADI.

Carcinogenicity: In long-term rat studies, growths in mammary tissue were observed, however these changes were not considered relevant to humans. Therefore, based on 2-year studies in mice and rats, there is no evidence of carcinogenicity in humans from simazine.

Genotoxicity: Simazine is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in rats and rabbits did not produce any evidence for effects on reproductive parameters or foetal development.

Special studies on endocrine effects: Simazine was found to bind weakly to the estrogen receptor of rat uterine cells *in vivo*, but only at levels well in excess of the likely levels of human exposure.

Poisons Schedule: Simazine is considered not to require control by scheduling due to its low toxicity and is therefore included in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for simazine was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.5mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a health-based guideline value of 0.002 mg/L for simazine, incorporating an additional safety factor of 10 for possible non-genotoxic carcinogenicity (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Bengtson Nash SM, Goddard J, Müller JF (2006). Phytotoxicity of surface waters of the Thames and Brisbane River estuaries: a combined chemical analysis and bioassay approach for the comparison of two systems. *Biosensors and Bioelectronics,* 15:2086-2093.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1986). Guidelines for Canadian Drinking Water Quality – Technical documents: Simazine. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/simazine/]

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71:97-106.

NOTE: Important general information is contained in PART II, Chapter 6

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Tran ATK, Hyne RV, Doble P (2007). Determination of commonly used polar herbicides in agricultural drainage waters in Australia by HPLC. *Chemosphere,* 67:944-953.

WHO (World Health Organization) (2003) *Simazine in Drinking-water; Background document for development of WHO Guidelines for Drinking-water Quality*. WHO/SDE/WSH/03.04/42 WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Sodium

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (taste), the concentration of sodium in drinking water should not exceed 180 mg/L.

No health-based guideline value is proposed for sodium. Medical practitioners treating people with severe hypertension or congestive heart failure should be aware if the sodium concentration in the patient's drinking water exceeds 20 mg/L.

GENERAL DESCRIPTION

The sodium ion is widespread in water due to the high solubility of sodium salts and the abundance of mineral deposits. Near coastal areas, windborne sea spray can make an important contribution either by fallout onto land surfaces where it can drain to drinking water sources, or from washout by rain. Apart from saline intrusion and natural contamination, water treatment chemicals, domestic water softeners and sewage effluent can contribute to the sodium content of drinking water.

Sodium salts are used in the paper, glass, soap, pharmaceutical and general chemical industries, and for a variety of other purposes. Sodium is also used in the food industry and for culinary purposes. Considerable amounts are excreted by humans and it is a common constituent of domestic sewage.

Sodium, as sodium salts such as sodium chloride or sodium sulfate, has a taste threshold of about 135 mg/L. The taste becomes appreciable when the sodium concentration exceeds 180 mg/L.

In most countries the majority of water supplies contain less than 20 mg/L but concentrations of up to 250 mg/L have been reported.

Food is the major contributor to sodium intake. In Australia the average dietary sodium intake has been estimated at about 4 g/day. Low-sodium diets may restrict this to less than 2 g/day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, sodium concentrations vary from 3 mg/L to 300 mg/L, with a typical value of 50 mg/L. Concentrations can vary markedly with local conditions.

TREATMENT OF DRINKING WATER

Sodium salts cannot be easily removed from drinking water; however, any steps to reduce sodium concentrations are encouraged (such as the use of alternative salts in domestic water softeners). Processes such as reverse osmosis or distillation can be employed but are costly to operate.

MEASUREMENT

The sodium concentration in drinking water can be determined by flame atomic absorption spectroscopy, inductively coupled emission spectroscopy or flame emission spectroscopy (APHA Method 3500-Na Parts B, C or D 1992). The limits of determination are less than 0.1 mg/L.

HEALTH CONSIDERATIONS

Whether water is consumed directly or with food or beverages, virtually all of the sodium in it will be absorbed. Sodium is present in all body tissues and fluids and its concentration is maintained by the kidney; increases in the sodium concentration in plasma give rise to the sensation of thirst.

Sodium is essential to human life but there is no agreement on the minimum daily amount needed to maintain health. It has been estimated that a total daily intake of less than 200 mg/person is required to meet the needs of growing infants and children.

Excessive sodium intake, usually via diet, can severely aggravate chronic congestive heart failure.

While it is clear that reduced sodium intake can reduce the blood pressure of some individuals with hypertension, it is equally clear that this type of therapy is not effective in all cases. Health authorities are of the opinion, however, that reduced sodium intake is beneficial.

DERIVATION OF GUIDELINE

The guideline value for sodium in drinking water is based on the taste threshold for sodium in water of 180 mg/L.

While there is evidence linking excess sodium intake with cardiovascular disease, it must be recognised that sodium intake via the water supply makes only a modest contribution to total intake. Nevertheless, water authorities are strongly encouraged to keep sodium concentrations as low as possible.

People with severe hypertension or congestive heart failure may need to restrict their overall dietary intake of sodium further if the concentration in drinking water exceeds 20 mg/L. Medical practitioners treating people with these conditions should be aware of the sodium concentration in the patient's drinking water.

REFERENCES

APHA Method 3500-Na Part B (1992). Sodium: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Na Part C (1992). Sodium: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Na Part D (1992). Sodium: Flame emission photometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

Spirotetramat

(endorsed 2011)

GUIDELINE

Based on human health concerns, spirotetramat in drinking water should not exceed 0.2 mg/L.

RELATED CHEMICALS

Spirotetramat (CAS 203313-25-1) belongs to the tetramic acid/cyclic ketoenol class of chemicals. There are no other pesticides in this class (Tomlin 2009).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, spirotetramat would not be a health concern unless the concentration exceeded 0.2 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Spirotetramat is an insecticide for the control of aphids, whiteflies and mealybugs in citrus, grapes, pome fruit, stone fruit, tree nuts, hops, vegetables and potatoes.

While spirotetramat is approved as an active ingredient, there are currently no registered products containing spirotetramat in Australia. Spirotetramat is intended for professional use only, to be applied by spray, using open or enclosed cab tractor-mounted or drawn sprayer fitted with hydraulic nozzles.

Exposure sources: If there were registered products, the main source of public exposure to spirotetramat would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Future agricultural use of spirotetramat may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Monitoring studies for spirotetramat in raw and tap water are limited and few data appear to be available (USEPA 2008), Babczinski and Hellpointner 2008).

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of spirotetramat in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Spirotetramat in drinking water can be measured by high performance liquid chromatography (HPLC) with tandem mass spectrometry or HPLC with ultraviolet irradiation. The analytical method 00836 developed by Bayer CropScience has been validated (USEPA 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for spirotetramat is 0.05 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 5 mg/kg bw/day from a one-year study in dogs. This NOEL is based on decreased thyroid hormone triiodothyronine and thyroxine levels and thymus involution. The ADI incorporates a safety factor of 100, and was established in 2008.

The acute reference dose (ARfD) of 1 mg/kg bw/day for spirotetramat was established in 2008, based on a NOEL of 100 mg/kg bw/day from an acute neurotoxicity study in rats. The ARfD incorporates a safety factor of 100.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Spirotetramat is rapidly absorbed from the gastrointestinal tract. There is no evidence of bioaccumulation. Spirotetramat is completely metabolised in the rat and the majority is excreted via the urine and faeces within 24 hours. The primary metabolites are enols (66-100% of all metabolites) and ketohydroxy forms of spirotetramat.

Acute effects: Spirotetramat has low acute oral and dermal toxicity in animal studies. It exhibits skin sensitisation potential in animals and humans.

Short-term effects: Short-term dietary studies in dogs and rats reported effects on the thyroid and thymus gland in dogs and on the testes, lung and kidney in rats at dose levels above 5 mg/kg bw/day.

Long-term effects: Long-term dietary studies in rats and mice reported that the target organs in rats were the kidney in both sexes and the liver in females at dose levels of 169 mg/kg bw/day and above. There were no adverse effects reported in mice up to the highest dose tested. A one-year dietary study in dogs reported the thymus and thyroid as target organs of toxicity at dose levels of 20 mg/kg bw/day and above. The NOEL of 5 mg/kg bw/day in dogs is the basis for the ADI.

Carcinogenicity: Spirotetramat did not demonstrate any evidence of carcinogenicity in rats or mice.

Genotoxicity: Spirotetramat is not considered genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Reproduction studies in rats reported the presence of abnormal sperm cells, decreased sperm motility and decreased reproductive performance at 70 mg/kg bw/day. In developmental toxicity studies in rats and rabbits, there was an increased incidence of skeletal variations and malformations at the maternally toxic dose of 1000 mg/kg bw/day. Both reproductive and developmental effects occurred at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Spirotetramat is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.2 mg/L for spirotetramat was determined as follows:

$$0.2 \text{ mg/L} = \frac{5.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 5.0 mg/kg bw/day is the NOEL based on a long-term (1-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Babczinski P, Hellpointner E (2008). Environmental fate of spirotetramat (Movento®). *Bayer CropScience Journal,* 61:181-202.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Tomlin CD (ed) (2009). *The Pesticide Manual: a world compendium*, 15th Edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2008). Pesticide Fact Sheet: Spirotetramat. USEPA, Office of Prevention, Pesticides and Toxic Substances

NOTE: Important general information is contained in PART II, Chapter 6

Styrene (vinylbenzene)

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (odour), the concentration of styrene in drinking water should not exceed 0.004 mg/L.

Styrene would not be a health concern unless the concentration exceeded 0.03 mg/L.

GENERAL DESCRIPTION

Styrene may be present in drinking water as a result of contamination from industrial sources. It has occasionally been detected in water supplies in the United States and the Netherlands at concentrations of less than 0.001 mg/L.

The taste threshold of styrene in water at 40°C ranges from 0.02 mg/L to 2.6 mg/L, depending on individual sensitivities. At 60°C the odour threshold in water is 0.004 mg/L.

Styrene is used in the production of plastics and resins. It has been detected in food packaged in polystyrene containers. However, improvements in the use of polystyrene since 1980 have resulted in substantial decreases in the release of the monomer. The daily exposure to styrene has been estimated to be 0.04 mg per person, with smokers receiving a higher dose. Forest fires may contribute to atmospheric concentrations of styrene.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Styrene has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Styrene can be removed from drinking water by reaction with ozone to form aldehydes, ketones and benzoic acid. It can also be adsorbed onto granular activated carbon.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for analysis (USEPA Draft Method 503.1 1986). An inert gas is bubbled through the sample and styrene trapped on an adsorbent. The adsorbent is then heated and styrene analysed using gas chromatography with photoionisation detection. The limit of determination is less than 0.0001 mg/L.

HEALTH CONSIDERATIONS

Approximately 60–90% of styrene is absorbed following ingestion or inhalation. It is widely distributed in the body, with a preference for fatty tissues. It is metabolised by a number of tissues and organs to styrene-7,8-oxide.

An extensive review and summary of the human and animal toxicity data for styrene is available (IPCS 1983).

A number of studies have reported on occupational inhalation of styrene. High doses for long periods have resulted in irritation of the respiratory system and some neurotoxic effects on both central and peripheral nervous systems. Chromosomal aberrations in lymphocytes have been associated with high styrene exposures, but not with low concentrations, among workers in the glass fibre industry.

In a long-term study using rats, female body weights were depressed at high doses (250 mg/kg body weight per day). No other treatment-related effects were observed.

Most studies using rodents have not found any association between styrene intake and an increased incidence of tumours. Styrene is mutagenic in a variety of test microorganisms, but only after metabolic activation. It also induces gene mutations and chromosomal aberrations in mammalian cells. The mutagenic agent is probably styrene-7,8-oxide, the main metabolic by-product of styrene and a direct-acting mutagen. Two long-term gavage studies using rats have also reported that styrene-7,8-oxide significantly increased the incidence of fore-stomach tumours at a dose of 250 mg/kg body weight per day.

The International Agency for Research on Cancer has concluded that styrene-7,8-oxide is probably carcinogenic to humans (Group 2A, inadequate evidence in humans, sufficient evidence in experimental animals, and supporting mechanistic evidence) (IARC 1994).

DERIVATION OF GUIDELINE

The assessment of the toxicological data for styrene by the World Health Organization (WHO) has been used without review. The health-based guideline value of 0.03 mg/L was determined as follows:

$$0.03 \text{ mg/L} = \frac{7.7 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 7.7 mg/kg body weight per day is the no-effect level based on a 2-year drinking water study using rats (Beliles *et al.* 1985).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for carcinogenic and genotoxic effects).

This health-based value is greater than the odour threshold of 0.004 mg/L.

The WHO guideline value of 0.02 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

Beliles RP, Butala JH, Stack CR, Makris S (1985). Chronic toxicity and three-generation reproduction study of styrene monomer in the drinking water of rats. *Fundamental and Applied Toxicology*, 5:855–868.

IARC (International Agency for Research on Cancer) (1994). IARC Monographs on the Evaluation of World Health. Carcinogenic Risks to Humans: Some Industrial Chemicals. World Health Organisation, IARC, 60, Lyons.

IPCS (International Programme on Chemical Safety) (1983). Styrene. Environmental Health Criteria, 26. World Health Organization, IPCS.

USEPA Draft Method 503.1 (1986). Volatile organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Sulfate

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (taste), the concentration of sulfate in drinking water should not exceed 250 mg/L. Purgative effects may occur if the concentration exceeds 500 mg/L.

GENERAL DESCRIPTION

Sulfate occurs naturally in a number of minerals, and is used commercially in the manufacture of numerous products including chemicals, dyes, glass, paper, soaps, textiles, fungicides and insecticides. Sulfate, including sulfuric acid, is also used in mining, pulping, and the metal and plating industries. Barium sulfate is used as a lubricant in drilling rigs for groundwater supply.

In the water industry, aluminium sulfate (alum) is used as a flocculant in water treatment, and copper sulfate is used for the control of blue-green algae (cyanobacteria) in water storages.

The highest concentrations reported in drinking water overseas are from groundwater supplies where the presence of sulfate is due to natural leaching from rocks. Concentrations have been reported up to 2200 mg/L. In source waters, concentrations are typically less than 100 mg/L.

The taste threshold for sulfate is in the range 250–500 mg/L.

Under anoxic conditions, the reduction of sulfate to sulfide by sulfate-reducing bacteria can result in unpleasant taste and odour due to the release of hydrogen sulfide, and can increase corrosion in pipes.

Food is probably the major source of intake of sulfate. In areas where the concentration of sulfate in water is high, drinking water may constitute the principal source of intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, sulfate concentrations range from 1 mg/L to 240 mg/L, with a typical concentration of 20 mg/L. Sulfate concentrations can vary markedly in different parts of the country.

TREATMENT OF DRINKING WATER

Most sulfate salts are very soluble and cannot be removed from drinking water by conventional water treatment processes. Desalination methods such as reverse osmosis or distillation are required for sulfate removal.

MEASUREMENT

The sulfate concentration of drinking water can be determined by the methylthymol blue method (APHA 4500-SO_4^{2-} Part F 1992) or using ion chromatography (APHA Method 4500-SO_4^{2-} Part B 1992). Limits of determination are 0.1 mg/L and 1 mg/L respectively.

HEALTH CONSIDERATIONS

Sulfate is rapidly absorbed by the gastrointestinal tract but a number of factors, such as the accompanying cation, can influence the rate of absorption. Low doses are probably absorbed more effectively than high doses. Sulfate is found in all body tissue but is highest in the metabolically active areas of bone and tooth formation, and may be important in regulating bone development.

Sulfate is one of the least toxic anions. Ingestion of high doses can result in catharsis (loosening of the bowels) with dehydration as a possible side effect.

No harmful effects have been reported in studies with animals.

Sulfate can interfere with disinfection efficiency by scavenging residual chlorine. It can also increase corrosion of mild steel pipes.

DERIVATION OF GUIDELINE

The guideline value is based on the taste threshold of sulfate in drinking water of 250 mg/L.

REFERENCES

APHA Method 4500-SO_4^{2-} Part F (1992). Sulfate: Methylthymol blue method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 4500-SO_4^{2-} Part B (1989). Sulfate: Ion Chromatographic method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

Sulprofos

(endorsed 2011)

GUIDELINE

Based on human health concerns, sulprofos in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Sulprofos (CAS 35400-43-2) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes fenthion, parathion, profenofos, and ethoprofos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, sulprofos would not be a health concern unless the concentration exceeded 0.01 mg/L. Excursions above this level even for a short period are of concern, as sulprofos causes cholinesterase inhibition even after relatively short periods of exposure.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Sulprofos is an insecticide for the control of whiteflies and other small plant-sucking pests on cotton, tomato and pepper crops.

There are no registered products containing sulprofos in Australia, but de-registered compounds may still be detected in water. Previously registered products were intended for professional use and were available as concentrated solutions to be applied in diluted form using ground or aerial sprays directly onto cotton or tomato foliage.

Exposure sources: The main source of public exposure to sulprofos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of sulprofos in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of sulprofos in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of sulprofos in drinking water have been identified.

MEASUREMENT

No suitable techniques for the analysis of sulprofos in drinking water have been identified. However, it is expected that a suitable method using high performance liquid chromatography with tandem mass spectrometry could be developed if required.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for sulprofos is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.3 mg/kg bw/day from long-term (2-year) dietary studies in rats and rabbits. The NOEL is based on decreased cholinesterase activity in plasma and red blood cells at doses of 3 mg/kg bw/day (dogs) and 4 mg/kg bw/day (rats) in dietary studies conducted over 2 years. The ADI incorporates a safety factor of 100, and was first established in 1979.

The previous *Australian Drinking Water Guidelines* health value was also 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Sulprofos is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised via the organothiophosphate groups to form an organophosphate, and via sulfoxidation to form sulfoxides and sulfones. Excretion is mainly via urine and is complete within 72 hours.

Acute effects: Sulprofos has moderate acute oral and dermal toxicity. It is not a skin sensitiser. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes.

Short-term effects: A 4-week oral study in rats reported decreased plasma and brain cholinesterase activity at doses of 1 mg/kg bw/day and above. No other effects were reported in this study.

Three-month dietary studies were conducted in rats and dogs. In rats, there was plasma cholinesterase inhibition and decreased bodyweight gain at doses of 0.6 mg/kg bw/day and above. In dogs, plasma cholinesterase was inhibited at doses of 0.7 mg/kg bw/day and clinical signs of cholinesterase inhibition were seen at higher doses. The NOEL in both studies was 0.3 mg/kg bw/day.

Long-term effects: Two-year dietary studies were conducted in mice, rats and dogs. There was cholinesterase inhibition in plasma and erythrocytes at doses of 4 mg/kg bw/day (mice and rats) and 3 mg/kg bw/day (dogs). Brain cholinesterase inhibition occurred in all three species at higher doses. The lowest overall NOEL was 0.3 mg/kg bw/day (rat, dog), and this is the basis for the current ADI.

Carcinogenicity: Based on 2-year studies in mice and rats, there is no evidence of carcinogenicity for sulprofos.

Genotoxicity: Sulprofos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation study in rats and developmental studies in rats and rabbits did not identify any adverse effects on reproductive parameters or foetal development.

Neurotoxicity: A 3-week dietary studies in hens using doses up to 50 mg/kg bw did not identify any clinical or histological signs of neurotoxicity.

Poisons Schedule: Sulprofos is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for sulprofos was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.3 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.3 mg/kg bw/day is the NOEL based on long-term (2-year) studies in rats and dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Taste and Odour

(endorsed 2011)

GUIDELINE

The taste and odour of drinking water should not be offensive to most consumers

GENERAL DESCRIPTION

Taste and odour are two of the primary criteria consumers use to judge the quality and acceptability of drinking water. People's sense of taste and smell tends to vary, and so the acceptability of the same water can vary from person to person and from day to day for the same person. Similarly, one individual within a group may be more or less sensitive to a particular substance than the group as a whole. Whilst taste and odour present in water does not generally have a health impact, the presence of tastes and odours may raise consumer concern with regard to water quality.

SOURCES OF TASTE AND ODOUR

Taste and odour in drinking water can result from naturally occurring inorganic chemicals; from biological activity, either in the source, treatment process or distribution system; as a by-product of water treatment processes; or from chemical contamination at any point from source to tap.

Inorganic compounds are generally present in water in substantially higher concentrations than organic compounds. Taste thresholds for some commonly occurring inorganic ions are about 0.1 mg/L for manganese, 0.3 mg/L for iron, 3 mg/L for copper, 3 mg/L for zinc, 250 mg/L for chloride, and 250-500 mg/L for sulfate. Most of these ions have health guidelines at concentrations higher than their taste thresholds (except copper at 2 mg/L). In most cases the customer would reject the water for aesthetic reasons before it would be of health concern.

Contamination of source water from spills, discharges or leaks of organic compounds can result in unpleasant taste and odours. Diesel fuel, for example, has a taste and odour threshold of 0.0005 mg/L. Methyl tert-butyl ether (MTBE) is the most commonly used fuel oxygenate added to reduce atmospheric concentrations of carbon monoxide and other aromatics. MTBE has frequently been detected in samples of shallow groundwaters, particularly in the United States. It affects the taste/odour of water at concentrations below 0.030 mg/L (Young *et al.* 1996).

One of the most common odours in water is described as "earthy", "musty" or "woody". Compounds most often linked to these odours are geosmin and 2 methyl isoborneol (MIB), which have similar low odour threshold concentrations of 0.00001 mg/L (10 ng/L) (Young *et al.* 1996). Cyanobacteria that produce these compounds include taxa representing the genera *Anabaena, Aphanizomenon, Planktothrix, Oscillatoria* and *Phormidium* in either planktonic and benthic habitats. Actinomycetes grow preferably in terrestrial habitats such as exposed sediments and vegetative debris, and are considered to enter aquatic habitats mainly in run-off from the shoreline.

Production of odorous compounds has been reported for most of the major algal classes and other odours produced by particular algae have been described as sweet, aromatic, cucumber, flowery, geranium, nasturtium, violets, fishy, peaty, grassy, mouldy, and vegetable. These odours originate from a variety of odorous compounds produced by the algae including aldehydes, ketones, alkenes, alcohols, terpenes, sulfides, amines, hydrocarbons, fatty acids, esters, carbonyl and aromatics. Cell concentrations as low as 500 cells/mL for some cyanobacteria and for a range of other algae are sufficient to taint a water supply.

Disinfection chemicals can contribute taste or odour to water. The odour threshold for free chlorine varies with

pH, but is generally considered to be between 0.1 and 0.4 mg/L, whilst monochloramine and dichloramine odour thresholds are considered to be 0.5 mg/L and 0.15 mg/L respectively. A study by Piriou *et al.* (2004) has determined taste thresholds of 0.05 mg/L for free chlorine, 0.1 mg/L for monochloramine and 0.2 mg/L for chlorine dioxide using trained French panellists with flavour profile analysis. Untrained panellists were around 2-4 times less sensitive and the US consumer panel was 5-10 times less sensitive than the French consumer panel. This result can be linked to the different chlorination practices in the two countries (residuals are around 0.1-0.2 mg/L in France compared with 1.0-3.0 mg/L in the USA).

A number of organic compounds produced as by-products of disinfection, particularly chlorination, can cause tastes and odours. Some chlorinated phenols, for example, have an antiseptic smell and a very low taste and odour threshold, varying from 0.002 to 0.0001 mg/L, whilst some brominated phenols have a threshold as low as 0.0000005 mg/L (0.5 ng/L) (Mackey *et al.* 2004).

A range of chloroanisoles can result in earthy/musty odours (Young *et al.* 1996). For example 2,4,6-trichloroanisole (TCA) is produced from the action of biofilms in distribution systems on the disinfection by-product 2,4,6 trichlorophenol. TCA is detected at lower concentrations (typically <0.000001 mg/L [<1 ng/L]) than MIB or geosmin, but it is less frequently responsible for odour incidents.

Dimethyl di- and tri-sulfides (DMDS and DMTS) are responsible for septic/swampy odours. They have odour threshold concentrations at low ng/L concentrations. It has been suggested that these compounds may be produced by microorganisms in distribution systems (Franzmann *et al.* 2001, Heitz *et al.* 2000).

Taste and odour can also arise from impacts on the supplied water within the customer's property, such as contaminants in direct or indirect contact with water (e.g. contaminants from kettles, refrigerators, dishwashers or washing machine hoses). The compound 2,6-dibromophenol, identified as probably responsible for a "plastic" or "chemical" taste in water after it is boiled, has a taste threshold concentration of 0.0005 mg/L (Whitfield *et al.* 1992, Adams *et al.* 1999). Odours resembling kerosene and cat urine were found to be more intense and more diverse when chlorine dioxide (ClO_2) was used, and increased numbers of complaints about odours in domestic water supplies were associated with the presence of new carpets in customers' homes (Dietrich *et al.* 1992).

MEASUREMENT

Sensory analysis

Sensory methods provide a qualitative classification and a semi-quantitative determination of taste and odour intensity. Sensory techniques include flavour profile analysis and sensory gas chromatography.

A panel experienced in flavour profile analysis (Krasner 1995, McGuire 1995, Suffet *et al.* 1999) often represents the first step in coping efficiently with taste and odour episodes. The twenty-first edition of *Standard Methods for the Examination of Water and Wastewater* (APHA AWWA WEF Method 2170 - 2005) presents flavour profile analysis (FPA) as a technique for identifying taste and odour samples. FPA uses a group of four or five trained panellists to examine the sensory characteristics of samples. Flavour attributes are determined by tasting, odour attributes by sniffing the sample. Panellists must be able to detect and recognise various flavours present and quantify them according to standards. FPA requires well trained panellists and data interpreters, and reproducibility of results depends on the training and experience of panellists. These panels are useful for assessing complaints by consumers, potentially identifying the source of an adverse flavour; and for assessing the impact of a new or improved treatment process on taste and odour.

Sensory gas chromatography (GC) (Bruchet 1999, Suffet *et al.* 1999) is often used to complement chemical analysis when identifying odorous compounds. The effluent from the GC capillary column is directed to the nose of an operator through tubing and a sniffing funnel. This technique has been used for plastic odours detected from polyethylene pipes.

NOTE: Important general information is contained in PART II, Chapter 6

Chemical analysis

Chemical analysis during a taste and odour episode can identify the compound(s) responsible for the organoleptic characteristics of the sample and thus potentially ensure that the episode is not linked to a possible health threat. However, as the human nose is very sensitive, to obtain similar sensitivity from chemical analysis it is first necessary to concentrate the samples.

Closed loop stripping analysis (CLSA) (Bruchet 1999, Crozes *et al.* 1999, Khiari *et al.* 1999) and liquid-liquid extraction using methylene chloride (Khiari *et al.* 1999, Ventura *et al.* 1995) represent two methods of choice for the concentration of the most common odorous compounds. Solid phase micro extraction is an alternative technique to CLSA for the extraction of odorous compounds from water (Bao *et al.* 1999). Purge-and-trap and headspace methods can possibly be used as complementary sample preparation techniques for volatile compounds but these methods have limits of detection at or greater than 0.001 mg/L. Stir bar sorptive extraction (Twister extraction) is the most recent development in extraction techniques for taste and odour compounds (Benanou *et al.* 2004, Baltussen *et al.* 1999). A stir bar coated with polydimethyl siloxane is placed in the sample, which is stirred for 30-120 minutes. The compounds are extracted onto the stir bar surface through this procedure, and the bar is then placed into a thermal desorption unit, which is connected online to the gas chromatograph-mass spectrometer. The limit of detection for this method is reported to be <0.000001 mg/L (<1 ng/L) for both geosmin and MIB.

A gas chromatograph-mass spectrometer, for separation and detection of the off-flavours compounds, is a prerequisite for any laboratory wishing to identify taste and odour compounds.

TREATMENT OF DRINKING WATER

Inorganic compounds, which can cause taste and odour, can be removed by the use of appropriate treatment processes. For example iron and manganese can be removed during conventional treatment with pre-oxidation. High salinity water may require the use of reverse osmosis to make the water palatable.

Organic substances producing taste and odour are generally more common in source waters. Volatile compounds can sometimes be removed by aeration. However, the application of activated carbon, in powdered (PAC) or granular form (GAC), is often the most effective treatment for the removal of a range of odour compounds. In Australia, PAC is the chosen method for the treatment of cyanobacterial metabolites due to its ease of application and because it can be applied only when required (Newcombe 2006). However, this method is quite costly if continuously applied. In addition, high levels of PAC will increase the load on the sludge treatment facilities, and may result in additional wear and tear on mechanical equipment (pumps etc) resulting in higher maintenance costs. GAC has many other advantages over PAC, such as its long life, higher adsorptive capacity, the ease of process control, more efficient use of the carbon, and the ability to regenerate the carbon for reuse (Herzing *et al.* 1977). Unfortunately at present there are no GAC regeneration facilities in Australia, so the waste adsorbent must be disposed and replaced.

Oxidation can also reduce tastes and odours. The effectiveness of the process is dependent upon factors such as the type of oxidant used, the type of reaction (addition or substitution), the structure of the compound, contact time, and environmental factors (e.g. pH, temperature, the presence of interfering compounds). Chlorine, chloramine and chlorine dioxide are common oxidants/disinfectants used in drinking water treatment. They have been effective in treating a variety of tastes and odours. However, MIB and geosmin have been found to be virtually resistant to this form of oxidation due to their tertiary structures (Lalezary *et al.* 1986, Anselme *et al.* 1988, Glaze *et al.* 1990). Ozonation is more effective for MIB and geosmin; approximately 50% removal of these compounds has been recorded in the laboratory, and at pilot and full scale (Ho *et al.* 2002, Ho 2004). The combination of ozone and GAC (sometimes called biological activated carbon or BAC) can be very effective in removing a range of odour compounds.

When disinfection results in tastes and odours being formed within distribution systems, the removal of precursors such as natural organic matter during the treatment process, increased oxidation, or change of

oxidant can sometimes solve the problem.

Tastes and odours arising out of reaction with biofilms can be reduced by preventing or reducing biofilm growth. This is best accomplished by introducing treatment processes that reduce the food source for bacteria, the assimilable organic carbon, and hence reduce biofilm growth. The presence of a disinfectant residual will also reduce biofilm growth but care must be taken not to introduce disinfectant-related tastes and odours.

If materials used in distribution systems result in discernable tastes and odours, then replacement of the materials or *in situ* lining of the pipework is recommended. The use of non-return devices or back-flow prevention devices can eliminate taste and odour issues associated with the back-siphoning of water, including hoses attached to dishwashers and washing machines installed within close proximity to a draw-off point. The introduction of the Australian and New Zealand Standard AS/NZS 4020 (2005) to ensure materials used in contact with water comply with a range of tests, including the formation of taste under standard test conditions, should eventually eliminate taste and odour problems associated with materials in contact with drinking water. However the interaction of disinfected water with components such as plastics in customers' kettles or new plumbing can be more difficult to control.

HEALTH CONSIDERATIONS

Taste and/or odour in potable water may indicate pollution of the water, insufficient water treatment and/or inadequate maintenance of the distribution system. Odours of a biological origin can indicate increased biological activity, for example by algae. Some algae can produce toxins and the detection of these algae by odour provides a useful early warning of potential problems, although odour does not necessarily indicate the presence of toxins.

DERIVATION OF GUIDELINE

It is clearly unsatisfactory for a water authority to be supplying water that is objectionable in taste and odour to consumers. This will undermine consumer confidence and may lead to the use of water from sources that are aesthetically more acceptable, but potentially less safe. Such sources may include untreated private household supplies, bore water or water treated through poorly maintained domestic filters. It is also unrealistic to expect complete consumer satisfaction with aesthetic characteristics of a water supply; therefore an appropriate guideline should be that the taste and odour of drinking water not be offensive to most people. Due to the subjective nature and range of causes of taste and odour, it is not possible to set a quantitative guideline.

OPERATIONAL GUIDELINE FOR MIB/GEOSMIN

The major cause of taste and odour episodes in Australian water supplies is the presence of MIB and/or geosmin. Based on experience in water utilities, action based on concentrations of MIB/geosmin suggested below will help to minimise customer complaints.

At treatment plant inlet	Total MIB and/or geosmin >10 ng/L	Increase sampling to every 2 days at treatment plant inlet Start MIB/geosmin analysis on treatment plant outlet
At treatment plant outlet	Total MIB and/or geosmin >10 ng/L	Introduce powdered activated carbon dosing to treatment plant

Regular measurement and identification of algae should also be undertaken to complement MIB/geosmin analysis at the inlet to the water treatment plant. Depending on the species-specific relationship between cell numbers and MIB/geosmin concentrations, additional monitoring may be necessary at the treatment

NOTE: Important general information is contained in PART II, Chapter 6

plant inlet when algal organisms known to be producers of these compounds exceed approximately 1000 cells/mL.

GUIDELINES IN OTHER COUNTRIES

The 2004 World Health Organization (WHO) Guidelines require that taste and odour be acceptable to the consumer.

The 1998 European Economic Community Standard (Council Directive 98/83/EC), requires that taste and odour be acceptable to consumers and that there be no abnormal change.

The 2005 Canadian Guidelines stipulate that drinking water shall have an inoffensive taste and odour.

Since 1979 the United States Environmental Protection Agency has listed odour in the secondary drinking water contaminant standards and has listed a secondary maximum contaminant level for odour of 3 expressed as a Threshold Odour Number (TON). They also recommend a variety of reports for further information on identification and control of taste and odours.

REFERENCES

Adams JB, Lock SJ, Toward MR, Williams BM (1999). Bromophenol formation as a potential cause of 'disinfectant' taint in foods. *Food Chemistry*, 64(3):377-381.

Anselme C, Suffet IH, Mallevialle J (1988). Effects of ozonation on tastes and odors. *Journal of the American Water Works Association*, 80(10):45-51.

AS/NZS (2005). *Testing of products for use in contact with drinking water*, AS/NZS 4020:2005. Standards Australia, Standards New Zealand.

APHA AWWA WEF Method 2170 - Flavor Profile Analysis (2005). *Standard Methods for the Examination of Water and Wastewater*, 21st Edition. American Public Health Association, American Water Works Association, Water Environment Federation.

Baltussen E, Sandra P, David F, Cramers CJ (1999). Stir bar sorptive extraction (SBSE), a novel extraction technique for aqueous samples: Theory and principles. *Journal of Microcolumn Separations*, 11(12):471-478.

Bao M, Griffini O, Burrini D, Santianni D, Barbieri K, Mascini M (1999). Headspace solid-phase microextraction for the determination of trace levels of taste and odor compounds in water samples. *Analyst,* 124:459-466.

Benanou D, Acobas F, de Roubin MR (2004). Optimization of stir bar sorptive extraction applied to the determination of odorous compounds in drinking water. *Water Science & Technology*, 49(9):171–178.

Bruchet A (1999). Solved and unsolved cases of taste and odor episodes in the files of Inspector Cluzeau. *Water Science & Technology*, 40(6):15-21.

Crozes G, Hagstrom J, Suffet I, Young C (1999). Bench-scale evaluation of adsorptive processes for taste and odors control using rapid small-scale column tests and flavor profile analysis. *Water Science & Technology*, 40(6):39-44.

Dietrich AM, Orr MP, Gallagher DL, Hoehn RC (1992). Tastes and odors associated with chlorine dioxide. *Journal of the American Water Works Association*, 84(6):82-88.

Franzmann PD, Heitz A, Zappia LR, Wajon JE, Xanthis K (2001). The formation of malodorous dimethyl oligosulfides in treated groundwater: the role of biofilms and potential precursors. *Water Research*, 35(7):1730-1738.

NOTE: Important general information is contained in PART II, Chapter 6

Glaze WH, Schep R, Chauncey W, Ruth EC, Zarnoch JJ, Aieta EM, Tate CH, McGuire MJ (1990). Evaluating oxidants for the removal of model taste and odor compounds from a municipal water supply. *Journal of the American Water Works Association*, 82(5):79-84.

Heitz A, Kagi RI, Alexander R (2000). Polysulfide sulfur in pipewall biofilms: its role in the formation of swampy odour in distribution systems. *Water Science and Technology*, 41(4-5):271-278.

Herzing DR, Snoeyink VL, Wood NF (1977). Activated carbon adsorption of the odorous compounds 2-methylisoborneol and geosmin. *Journal of the American Water Works Association*, 69(4):223-228.

Ho L (2004). Removal of Cyanobacterial Metabolites from Drinking Water using Ozone and GAC. PhD Thesis. University of South Australia, Mawson Lakes Campus.

Ho L, Newcombe G, Croue J (2002). The reactivity of ozone with well characterised NOM fractions and its effect on the destruction of MIB and Geosmin. *Water Research* 36(3)511-518.

Khiari D, Bruchet A, Gittelman T, Matia L, Barrett S, Suffet I, Hund R (1999). Distribution-generated taste-and-odor phenomena. *Water Science & Technology*, 40(6)129-133.

Krasner S (1995). The use of reference materials in sensory analysis. *Water Science & Technology*, 31(11):265-272.

Lalezary S, Pirbazari M, McGuire MJ (1986). Oxidation of five earthy-musty taste and odor compounds. *Journal of the American Water Works Association*, 78(3):62-69.

Mackey ED, Baribeau H, Fonseca AC, Davis J, Brown J., Boulos L, Crozes GF, Piriou P, Rodrigues JM, Fouret M, Bruchet A, Hiltebrand DJ (2004). Public perception of tap water chlorinous flavour. AWWA Research Foundation, Denver, CO.

McGuire M (1995). Off-flavor as the consumers measure of drinking water safety. *Water Science & Technology*, 31(11):1-8.

Newcombe G (2006). Removal of natural organic material and algal metabolites using activated carbon. In: G Newcombe, D Dixon (eds), *Interface Science in Drinking Water Treatment: Theory and Applications*, Interface Science and Technology Series, 10, Elsevier, Amsterdam, the Netherlands.

Piriou P, Mackey ED, Suffet IH, Bruchet A (2004). Chlorinous flavour perception in drinking water. *Water Science & Technology*. 49(9):321-328.

Suffet I, Khiari D, Bruchet A (1999). The drinking water taste and odor wheel for the millennium: beyond geosmin and 2-methylisoborneol. *Water Science & Technology*, 40(6):1-13.

Sugiura N, Iwami N, Inamori Y, Nishimura O, Sudo R (1998). Significance of attached cyanobacteria relevant to the occurrence of musty odor in Lake Kasumigaura. *Water Research,* 32(12):3549-3554.

Ventura F, Matia L, Romero J, Boleda M, Marti I (1995). Taste and odor events in Barcelona's water supply. *Water Science & Technology*, 31(11):63-68.

Whitfield FB, Shaw KJ, Walker DI (1992). The source of 2,6-dibromophenol: cause of an iodoform taint in Australian prawns. *Water Science and Technology*, 25(22):131-138.

Young WF, Horth H, Crane R, Ogden T, Arnott M (1996). Taste and odour threshold concentrations of potential potable water contaminants. *Water Research*, 30(2):331-340.

NOTE: Important general information is contained in PART II, Chapter 6

Temephos

(endorsed 2011)

GUIDELINE

Based on human health concerns, temephos in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Temephos (CAS 3383-96-8) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, including fenthion, parathion, profenofos and ethoprophos (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, temephos would not be a health concern unless the concentration exceeded 0.4 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Temephos is an insecticide for the control of skin parasites on farm and non-farm animals, and for the control of mosquito and midge larvae in breeding areas.

There are registered products containing temephos in Australia. The products are intended for professional use only. Use patterns include hand-held sprays, shower sprays, dip solution, and shampoo for control of fleas on farm and non-farm animals, and as a liquid concentrate for application to surface water for control of mosquito and midge larvae in public health settings. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to temephos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The application of temephos to surface water in mosquito breeding areas may potentially lead to the contamination of drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of temephos in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of temephos in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Temephos can be measured in water by solvent extraction followed by high performance liquid chromatography (WHO 2008) or by on-line solid-phase extraction followed by thermospray liquid chromatography with mass spectrometry (Lacorte and Barcel 1995). The practical limit of quantitation is 0.04 μg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for temephos is 0.1 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a 4-week dietary study in humans. This NOEL is based on serum cholinesterase inhibition. The ADI incorporates a safety factor of 10, and was first established in 1988.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Temephos is only partially absorbed from the gastrointestinal tract, with wide, uniform tissue distribution in mammals. It is rapidly eliminated unchanged in the faeces or as temephos sulfoxide in urine. It has a low potential for bioaccumulation.

Acute effects: Temephos has low to moderate oral acute toxicity, and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: Short-term dietary studies in rats and dogs reported symptoms indicative of nervous system toxicity. In a 5-day dietary study in humans, temephos had no effects on blood cholinesterase activity at 256 mg/kg bw/day. In a 4-week dietary study in humans, temephos had no effect on cholinesterase activity at 1 mg/kg bw/day. In medium-term dietary studies in rats and dogs, effects included red blood cell cholinesterase inhibition and increased liver weight at dose levels above 0.1 mg/kg bw/day in rats and above 0.45 mg/kg bw/day in dogs.

Long-term effects: Long-term dietary studies in rats and dogs reported symptoms indicative of nervous system toxicity. No toxicity was noted up to 15 mg/kg bw/day, but cholinesterase was not measured in these studies.

Carcinogenicity: Based on long-term toxicity studies in rats, there is no evidence of carcinogenicity for temephos.

Genotoxicity: Temephos is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In reproduction studies in rats and developmental studies in rats and rabbits, there was no evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Temephos is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for temephos was determined as follows:

$$0.4 \text{ mg/L} = \frac{1.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 1.0 mg/kg bw/day is the NOEL based on a short-term (4-week) dietary study in humans.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from studies conducted in humans in order to take into account human variation.

Temephos is included in the WHO *Guidelines for Drinking Water Quality* list of pesticides excluded from guideline value derivation, as it is added to water supplies for control of mosquitoes and larvae (WHO 2006). The philosophy is that these pesticides play an important role in the control of disease, and guidelines that are unnecessarily stringent as to impede their use should be avoided if possible.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Lacorte S, Barcel D (1995). Determination of organophosphorus pesticides and their transformation products in river waters by automated on-line solid-phase extraction followed by thermospray liquid chromatography-mass spectrometry. *Journal of Chromatography A*, 712:103-112.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004) *Guidelines for Drinking-water Quality* 3rd Edition, WHO, Geneva, Swizterland.

WHO (World Health Organization((2008) *WHO Specifications and Evaluation for Public Health Pesticides – Temephos*. WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Temperature

(endorsed 1996)

GUIDELINE

No guideline is set due to the impracticality of controlling water temperature.

Drinking water temperatures above 20°C may result in an increase in the number of complaints.

GENERAL DESCRIPTION

Temperature is primarily an aesthetic criterion for drinking water. Generally, cool water is more palatable than warm or cold water.

In general, consumers will react to a change in water temperature. Complaints are most frequent when the temperature suddenly increases.

The turbidity and colour of filtered water may be indirectly affected by temperature, as low water temperatures tend to decrease the efficiency of water treatment processes by, for instance, affecting floc formation rates and sedimentation efficiency.

Chemical reaction rates increase with temperature, and this can lead to greater corrosion of pipes and fittings in closed systems. Scale formation in hard waters will also be greater at higher temperatures.

MEASUREMENT

Temperature measurements should be made with a good quality, mercury-filled Celsius thermometer (APHA Method 2550B 1992).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Water temperatures in major Australian reticulated supplies range from 10°C to 30°C. In some long, above-ground pipelines, water temperatures up to 45°C may be experienced.

CONTROL IN DRINKING WATER SUPPLIES

Control of water temperature in reticulated supplies is seldom practical or effective. Selective withdrawal from deep reservoirs can be used but this may introduce other water quality problems. Aeration can also be used. In some situations it may be possible to place pipes underground to reduce water temperature fluctuations, or to vary the times water remains in pipes and storage tanks.

HEALTH CONSIDERATIONS

The effectiveness of chlorine as a disinfectant is influenced by the temperature of the water being dosed. Generally higher temperatures result in more effective disinfection at a particular chlorine dose, but this may be counterbalanced by a more rapid loss of chlorine to the atmosphere (AWWA 1990).

Chlorine reacts with organic matter in water to produce undesirable chlorinated organic by-products, and higher temperatures increase the rate of these reactions.

Temperature can directly affect the growth and survival of microorganisms. In general the survival time of infectious bacteria and parasites is reduced as the temperature of the contaminated water increases.

Naegleria fowleri, which can cause amoebic meningitis, grows between 18°C and 46°C and is likely to occur in nondisinfected water supplies that reach 30°C seasonally. *Legionella pneumophila* (which causes Legionnaires' disease) and related bacteria are found in hot and cold water systems, with colonisation occurring in stagnant water at temperatures between 20°C and 45°C. Increased temperatures can also promote the growth of taste- and odour-producing organisms in lakes and impoundments, and in distribution systems.

GUIDELINES IN OTHER COUNTRIES

The European Economic Community Standards have a guideline value of 12°C and a maximum of 25°C.

The Canadian Guidelines have a recommended value of 15°C.

The 1984 World Health Organization (WHO) Guidelines do not include a value for temperature as control is usually impractical. The 1993 WHO Guidelines require that temperature should be acceptable to avoid consumer complaints.

REFERENCES

APHA Method 2550B (1992). Temperature: Laboratory and field methods. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

AWWA (American Water Works Association) (1990). *Water Quality and Treatment: A handbook of community water supplies*. AWWA, 4th edition, McGraw-Hill Inc.

NOTE: Important general information is contained in PART II, Chapter 6

Terbacil

(endorsed 2011)

GUIDELINE

Based on human health concerns, terbacil in drinking water should not exceed 0.2 mg/L.

RELATED CHEMICALS

Terbacil (CAS 5902-51-2) belongs to the uracil class of chemicals. Other pesticides in this class include bromacil and butafenacil (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, terbacil would not be a health concern unless the concentration exceeded 0.2 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Terbacil is a selective herbicide for the control of annual and perennial weeds in agricultural crops such as sugarcane, apples, peaches, citrus, and almond trees.

There are registered products that contain terbacil in Australia. The products are intended for professional use. Terbacil is available as concentrated solutions to be applied in diluted form using ground, aerial or hand-held sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to terbacil and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of terbacil may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of terbacil in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of terbacil by conventional drinking water treatment processes have been identified. However, advanced oxidation using ultraviolet radiation and hydrogen peroxide is effective under optimised conditions (Shemer *et al.* 2006, Elovitz *et al.* 2008).

MEASUREMENT

No suitable techniques for the analysis of terbacil in drinking water have been identified. However, it is expected that a suitable method using high performance liquid chromatography with tandem mass spectrometry could be developed if required.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for terbacil is 0.06 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 6.25 mg/kg bw/day from a long-term (2-year) dietary study in dogs. The NOEL is based on increased relative liver weight and elevated serum alkaline phosphatase at the highest dose tested of 60 mg/kg bw/day. The ADI incorporates a safety factor of 100, and was first established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Terbacil is readily and extensively absorbed via the gastrointestinal tract in mammals. It is not extensively metabolised, but the limited metabolic pathway proceeded by 6-methyluracil hydroxylation, sulfonation, and conjugation. Excretion was complete by 48 hours as metabolised compound in urine and minor amounts in faeces.

Acute effects: Terabacil has low acute oral and dermal toxicity. It is not a skin sensitiser in guinea-pig.

Short-term effects: A 3-month dietary studies in rats reported an increase in absolute and relative liver weights, associated with hepatocellular hypertrophy and vacuolation at doses of 250 mg/kg bw/day.

Long-term effects: Long-term dietary studies were conducted in mice, rats and dogs. In mice, there was decreased pituitary weight, and centrilobular hypertrophy at doses of 180 mg/kg bw/day. In rats, there were increased absolute and relative liver weights, associated with centrilobular hypertrophy and vacuolation, at doses of 125 mg/kg bw/day. In dogs, there was increased relative liver weight and elevated serum alkaline phosphatase, with no histological evidence of adverse effects, at the highest dose tested, 60 mg/kg bw/day. The next lowest dose and lowest overall NOEL was 6.25 mg/kg bw/day in this study, and this is the basis for the current ADI.

Carcinogenicity: Based on an 18-month study in mice, and a 2-year study in rats, there is no evidence of carcinogenicity for terbacil.

Genotoxicity: Terbacil is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 3-generation reproduction study in rats and a developmental study in rats did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Terbacil is considered not to require control by scheduling due to its low toxicity and is therefore in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.2 mg/L for terbacil was determined as follows:

$$0.2 \text{ mg/L} = \frac{6.25 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 6.25 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Elovitz MS, Shemer H, Peller JR, Vinodgopal K, Sivaganesan M, Linden KG (2008). Hydroxyl radical rate constants: comparing UV/H_2O_2 and pulse radiolysis for environmental pollutants. *Journal of Water Supply Research and Technology-Aqua,* 57(6):391-401.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Shemer H, Sharpless CM, Elovitz MS, Linden KG (2006). Relative rate constants of contaminant candidate list pesticides with hydroxyl radicals. *Environmental Science and Technology,* 40(14):4460-4466.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Terbufos

(endorsed 2011)

GUIDELINE

Based on human health concerns, terbufos in drinking water should not exceed 0.0009 mg/L.

RELATED CHEMICALS

Terbufos (CAS 13071-79-9) belongs to the organophosphate group of chemicals. There are many other pesticides in this class, which includes chlorpyrifos, dimethoate, ethoprophos and ethion (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population to terbufos is expected to be well below levels that may cause health concerns.

Terbufos is an acutely poisonous organophosphate pesticide. If it is detected in water as a result of spillage or misuse at levels above 0.0009mg/L, remedial action should be taken. Concentrations of terbufos greatly exceeding this guideline present an acute human health risk.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Terbufos is used as an insecticide and nematocide for the control of various above-ground insects, soil arthropods, and nematodes in agriculture, including food crops.

There are registered products containing terbufos in Australia. The products are granular formulations to be applied to the soil in agricultural settings**.** Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to terbufos and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of terbufos may potentially lead to contamination of source waters through processes such as run-off or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of terbufos in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of terbufos in drinking water have been identified.

MEASUREMENT

Terbufos can be measured in drinking water by solid phase extraction and capillary column gas chromatography–mass spectrometry (USEPA 2000). The practical limit of detection is 0.05 μg/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for terbufos is 0.0002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.0025 mg/kg bw/day from a short-term (6-month dietary) study. The NOEL is based on decreased serum cholinesterase activity in dogs. The ADI incorporates a safety factor of 10, and was established in 1992.

The previous ADI of 0.00003 mg/kg bw/day set in 1980 was based on the same study and endpoint. The use of a safety factor of 100 was reconsidered in 1992 following an evaluation by the WHO. The 1992 review affirmed the validity of cholinesterase inhibition as an endpoint of toxicity, but decreased the safety factor to 10.

The previous *Australian Drinking Water Guidelines* health value was 0.0005 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Terbufos is readily absorbed via the gastrointestinal tract and is widely distributed in tissues and blood. In rats, terbufos was slowly excreted as metabolites (within 7 days), mainly in the urine. The major metabolites were S-methylated metabolites, which are of similar or lower toxicity to terbufos.

Acute effects: Terbufos has high acute oral toxicity in rats and high acute dermal toxicity in rabbits. The skin sensitisation potential of terbufos is unknown. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes.

Short-term effects: Short-term dietary studies in rats and dogs reported symptoms indicative of nervous system toxicity. In 28-day dietary studies, decreased cholinesterase activity was reported at doses above 0.0125 mg/kg bw/day in rats and above 0.01 mg/kg bw/day in dogs. In a 3-month dietary study in rats and a 6-month dietary study in dogs, cholinesterase inhibition occurred at doses above 0.05 mg/kg bw/day in rats and 0.0025 mg/kg bw/day in dogs. The NOEL of 0.0025 mg/kg bw/day in dogs is the basis for the current ADI.

Long-term effects: Long-term dietary studies in rats and dogs reported symptoms indicative of nervous system toxicity. A 1-year dietary study in dogs reported inhibition of cholinesterase at the lowest dose tested, 0.015 mg/kg bw/day.

Carcinogenicity: Based on long-term dietary studies in mice or rats, there is no evidence of carcinogenicity for terbufos.

Genotoxicity: Terbufos is not considered to be genotoxic, based on *in vitro* or *in vivo* short-term studies.

Reproductive and developmental effects: Reproduction studies and developmental studies in rats reported no effects on reproductive parameters or foetal development other than that resulting from maternal toxicity.

Neurotoxicity: There was no clinical evidence of delayed or residual neurotoxicity or demyelination in 21-day toxicity studies in hens.

Poisons Schedule: Terbufos is included in Schedule 7 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons

Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.0009 mg/L for terbufos was determined as follows:

$$0.0009 \text{ mg/L} = \frac{0.0025 \text{ mg/kg body weight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 10}$$

where:

- 0.0025 mg/kg bw/day is the NOEL based on a medium-term (6-month) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 10 is the safety factor applied to the NOEL derived from animal studies. The safety factor of 10 was considered to provide an adequate margin of safety, as despite being derived from animal studies, the end-point (inhibition of plasma cholinesterase) is considered to be a very sensitive indicator.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency (2000). Method 526. Determination of selected semivolatile organic compounds in drinking water by solid phase extraction and capillary column gas chromatography/ mass spectrometry (GC/MS).

NOTE: Important general information is contained in PART II, Chapter 6

Terbuthylazine

(endorsed 2011)

GUIDELINE

Based on human health concerns, terbuthylazine in drinking water should not exceed 0.01 mg/L.

RELATED CHEMICALS

Terbuthylazine (CAS 5915-41-3) is in the triazine class of chemicals. Other pesticides in this class include ametryn, atrazine, simazine and terbutryn (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, terbuthylazine would not be a health concern unless the concentration exceeded 0.01 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Terbuthylazine is a herbicide used to control pre- and post-emergent weeds in a variety of agricultural crops and in forestry. It is also used as an industrial algicide in water cooling systems, ponds and fountains, and is used as an algicide in swimming pools.

There are currently no registered products containing terbuthylazine in Australia, but de-registered compounds may still be detected in water. Terbuthylazine has been used previously by professionals and applied by boom spray to crops as well as being used in swimming pools. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to terbuthylazine, if used, would be residues in food.

If used in the future in agriculture, its use may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Values for terbuthylazine in Australian drinking water were not found. Levels in surface and bore waters in Europe and North America ranged from less than 0.1 µg/L to approximately 2 µg/L. Levels in Australian waters would normally be expected to be within this range.

TREATMENT OF DRINKING WATER

Relatively high removal rates of terbuthylazine have been achieved using conventional flocculation, adsorption onto activated carbon and ozonoation (Ormad *et al*. 2008). If terbuthylazine is detected, jar testing with a matrix of multiple oxidants, adsorbents, and coagulants is recommended.

MEASUREMENT

Measurement of residue levels in water can be undertaken by use of solid phase extraction and high performance liquid chromatography with either ultraviolet detection or tandem mass spectrometry.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for terbuthylazine is 0.003 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.35mg/kg bw/day from a 24-month dietary study in rats showing a decrease in bodyweight gain and food consumption. The ADI incorporates a safety factor of 100, and was established in 2001.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Terbuthylazine is readily absorbed from the gastrointestinal tract (~ 90%) and distributed widely in tissues. It is extensively metabolised and excreted rapidly, mainly via the urine, within 7 days. There is no evidence of bioaccumulation.

Acute effects: Terbuthylazine has low acute oral and dermal toxicity. There is some evidence for skin sensitisation in guinea pigs.

Short-term effects: In dietary studies in rats, there was reduced bodyweight gain at 75 mg/kg bw/day in rats. There was no effect on organ weights or tissue histopathology at doses up to 5 mg/kg bw/day in rats.

Long-term effects: In long-term dietary studies in mice, rats and dogs, there was a decrease in bodyweight gain and food consumption in all species. There were no consistent changes in haematology, clinical chemistry or histopathological findings indicative of systemic toxicity. The lowest NOEL was 0.35 mg/kg/day in the 2-year rat study, based on reduced bodyweight gain and reduced food consumption. This NOEL is the basis for the ADI.

Carcinogenicity: Based on long-term studies in mice and rats, there is no evidence of carcinogenicity for terbuthylazine.

Genotoxicity: Terbuthylazine is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In a reproduction study in rats and developmental studies in rats and rabbits, terbuthylazine showed no evidence of effects on reproductive parameters or on foetal development.

Poisons Schedule: Terbuthylazine is included in Schedule 6 of the of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on the concentration and use. Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.01 mg/L for terbuthylazine was determined as follows:

$$0.01 \text{ mg/L} = \frac{0.35 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.35 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a guideline value of 0.007 mg/L for terbuthylazine (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere*, 71: 97–106.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Terbutryn

(endorsed 2011)

GUIDELINE

Based on human health concerns, terbutryn in drinking water should not exceed 0.4 mg/L.

RELATED CHEMICALS

Terbutryn (CAS 886-50-0) belongs to the triazene class of chemicals. There are many other pesticides in this class, which includes ametryn, propazine, prometon, and cyanazine (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.If present in drinking water as a result of a spillage or through misuse, terbutryn would not be a health concern unless the concentration exceeded 0.4 mg/L. Excursions above this level even for a short period are of concern, as the health-based guideline is based on short- to medium-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Terbutryn is a herbicide for the control of broad-leaf weeds in wheat, barley, triticale, peas, sugar cane and turf agricultural crops.

There are registered products that contain terbutryn in Australia. The products are intended for professional use. Terbutryn is available as concentrated solutions to be applied in diluted form using ground and aerial sprays. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to terbutryn and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of terbutryn may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No specific reports of terbutryn in Australian drinking waters have been identified. However, a study of four small river systems in Germany revealed maximum concentrations of up to 5.6 µg/L (Quednow and Puttmann 2007). Terbutryn has also been reported in ground water in the UK at concentrations up to 0.13 µg/L (Lapworth *et al.* 2006).

TREATMENT OF DRINKING WATER

Preoxidation of drinking water by chlorine is effective for the removal of terbutryn (Ormad *et al.* 2008). However, this approach will produce unidentified by-products, which will remain in the water. An alternative and equally effective approach is the preoxidation of terbutryn by ozone followed by activated carbon adsorption (Ormad *et al.* 2008).

MEASUREMENT

Numerous analytical methods are available for multiresidue analysis of triazene pesticides. For example, terbutryn may be measured in drinking waters by relatively common methods of solid phase extraction and gas chromatography–mass spectrometry, with a quantification limit of 30 ng/L (Ormad *et al.* 2008). More advanced methods, including gas chromatography coupled to high-resolution time-of-flight mass spectrometry, have also been reported (Hernandez *et al.* 2007). Terbutryn may also be measured in drinking waters along with other triazenes by ultra-performance liquid chromatography with tandem mass spectrometric detection. The limit of quantification for all analytes in this method was reported to be 5 ng/L (Drozdzynski and Folkman 2008).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for terbutryn is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 10 mg/kg bw/day from short-term dietary studies in rats (13-week) and dogs (6-months). The NOEL is based on clinical signs of toxicity in a 6-month dietary study in dogs, and evidence of liver and thyroid toxicity in a 16-week dietary study in rats. The ADI incorporates a safety factor of 100, and was established in 1986. An ADI for terbutryn was not set in Australia before 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.3 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Terbutryn is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, with the main metabolites in the form of polar metabolites and conjugates. It is excreted mainly in the urine and to a lesser extent in the faeces within 72 hours.

Acute effects: Terbutryn has low acute oral and dermal toxicity. It is a skin sensitiser in guinea pigs, although there are no reports of sensitisation in humans working with terbutryn.

Short-term effects: A 16-week dietary study in rats reported thyroid toxicity (follicular hypertrophy and follicular eosinophilic infiltration) and liver toxicity (increased serum alkaline phosphatase activity, cholesterol, alanine amino-transferase, and glucose) at doses of 50 mg/kg bw/day and above A 6-month dietary study in dogs reported clinical signs of toxicity (increased salivation, hyper-responsiveness to sound, and timid behaviour) and intestinal epithelial necrosis at doses of 25 mg/kg bw/day and above. The NOEL in both of these studies was 10 mg/kg bw/day, and this is the basis for the current ADI.

Long-term effects: In long-term dietary studies in mice, there was no evidence of toxicity up to doses of 150 mg/kg bw/day. In long-term dietary studies in rats, there was decreased bodyweight gain and food consumption, follicular hyperplasia, and liver adenomas at 150 mg/kg bw/day. The lowest NOEL was 15 mg/kg bw/day (rats) in these studies.

Carcinogenicity: There was evidence of carcinogenicity but only at dose levels well in excess of the likely level of human exposure.

Genotoxicity: Terbutryn is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A 3-generation reproduction study in rats, and developmental studies in rats and rabbits, did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Terbutryn is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.4 mg/L for terbutryn was determined as follows:

$$0.4 \text{ mg/L} = \frac{10 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 10 mg/kg bw/day is the NOEL based on a medium-term (13-week) dietary study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Drozdzynski D, Folkman W (2008). Analysis of triazine herbicides in water using solid phase extraction followed by ultra-performance liquid chromatography with tandem mass detection (UPLC-MS/MS). *Chemia Analityczna,* 53(3):391-400.

Hernandez F, Portoles T, Pitarch E, Lopez FJ (2007). Target and nontarget screening of organic micropollutants in water by solid-phase microextraction combined with gas chromatography/high-resolution time-of-flight mass spectrometry. *Analytical Chemistry,* 79(24):9494-9504.

Lapworth DJ, Gooddy DC, Stuart ME, Chilton PJ, Cachandt G, Knapp M, Bishop S (2006). Pesticides in groundwater: some observations on temporal and spatial trends. *Water and Environment Journal,* 20(2):55-64.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71(1):97-106.

Quednow K, Puttmann W (2007). Monitoring terbutryn pollution in small rivers of Hesse, Germany. *Journal of Environmental Monitoring,* 9(12):1337-1343.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Tetrachloroethene (also known as tetrachloroethylene or perchloroethylene)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of tetrachloroethene in drinking water should not exceed 0.05 mg/L.

GENERAL DESCRIPTION

Tetrachloroethene is used as a solvent in the dry-cleaning industry. It may be present in drinking water through contamination of water sources by spills or discharges. In the United Kingdom and the United States it has occasionally been detected in drinking water at concentrations below 0.001 mg/L. It has been found at higher concentration in contaminated groundwater.

The odour threshold in water is 0.3 mg/L.

Tetrachloroethene is widespread in the environment through use in dry-cleaning and as a metal-degreasing fluid. It has been reported in trace amounts in food, water, aquatic organisms and human tissue.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Tetrachloroethene has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

Tetrachloroethene can be removed from drinking water by adsorption onto granular activated carbon or by aeration.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of tetrachloroethene (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and tetrachloroethene extracted using methyl tert-butyl ether. The extract is then analysed using gas chromatography with an electron capture detector. The limit of determination is approximately 0.000004 mg/L (4 ng/L).

HEALTH CONSIDERATIONS

Tetrachloroethene is rapidly absorbed after ingestion or inhalation. It is eliminated primarily by the lungs. In the body it is slowly metabolised to trichloroacetic acid.

An extensive review and summary of the human and animal toxicity data for tetrachloroethene is available (IPCS 1984).

The most notable acute effect of short-term exposure is depression of the central nervous system. Short-term studies of up to 3 months using mice and rats reported weight loss, and found some evidence of liver and kidney toxicity at high doses (400 mg/kg body weight per day).

Inhalation exposure to impure tetrachloroethene at 100 ppm and above in air caused hepatocellular carcinomas in mice. Exposure at 200 ppm in air increased the incidence of leukaemia in rats.

Mutagenic activity was not observed in most tests with a number of strains of bacteria. No chromosome aberrations were observed using rat or mouse cells, or human lymphocytes.

The International Agency for Research on Cancer has concluded that tetrachloroethene is possibly carcinogenic to humans (Group 2B, inadequate human data but sufficient evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The guideline value of 0.05 mg/L for tetrachloroethene in drinking water was determined as follows:

$$0.05 \text{ mg/L} = \frac{14 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 14 mg/kg body weight per day is the no-effect level from a 90-day drinking water study using rats and mice (Buben and O'Flaherty 1985, Hayes *et al*. 1986).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 to account for possible carcinogenicity). An additional factor for the less than lifetime study was not applied as long-term carcinogenicity bioassays were available.

The World Health Organization guideline value of 0.04 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

Buben JA, O'Flaherty EJ (1985). Delineation of the role of metabolism in the heptatoxicity of trichloroethylene and perchloroethylene: A dose effect study. *Toxicology and Applied Pharmacology*, 78:105–122.

Hayes JR, Condie LW, Borzelleca JF (1986). The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. *Fundamental and Applied Toxicology*, 7:119–125.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1984). Tetrachloroethylene. Environmental Health Criteria, 31. World Health Organization, IPCS.

USEPA Draft Method 551 (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Thiobencarb

(endorsed 2011)

GUIDELINE

Based on human health concerns, thiobencarb in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Thiobencarb (CAS 28249-77-6) belongs to the thiocarbamate class of chemicals. There are many other pesticides in this class, including EPTC, pebulate, and molinate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, thiobencarb would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Thiobencarb is a selective herbicide for the control of grass weeds in rice crops.

There is at least one registered product containing thiobencarb in Australia. Thiobencarb products are intended for professional use, and are available as a concentrated solution to be diluted and applied to water via ground and aerial spray. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to thiobencarb and its metabolites is residues in food and drinking water. Residue levels in food produced according to good agricultural practice are generally low, and maximum residue limits are at the level of detection.

The agricultural use of thiobencarb involves direct application into irrigation water, which may then enter source waters for drinking water.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of thiobencarb in Australian drinking waters have been identified. However, thiobencarb has been reported in run-off from Australian rice fields (Quayle *et al.* 2006), indicating the potential for drinking water contamination in rice-growing regions.

TREATMENT OF DRINKING WATER

During chlorination of drinking water, thiobencarb has been shown to be quickly degraded, producing chlorobenzyl alcohol, chlorotoluene, chlorobenzyl chloride, chlorobenzoic acid and chlorobenzyl

aldehyde as chlorination by-products (Magara *et al.* 1994).

MEASUREMENT

No suitable techniques for the analysis of thiobencarb in drinking water have been identified. However, thiobencarb has commonly been measured in soils and run-off from rice growing operations using high performance liquid chromatography–tandem mass spectrometry, and such methods could be adapted if required.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for thiobencarb is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1.0 mg/kg bw/day from long-term dietary studies in dogs (1-year) and rats (2-year). The NOEL is based on decreased plasma cholinesterase activity, reduced bodyweight gain, and evidence of anaemia at doses of 5 mg/kg bw/day and above. The ADI incorporates a safety factor of 100, and was established in 1989.

The previous ADI of 0.007 mg/kg bw was set in 1977, based on a NOEL of 0.75 mg/kg bw/day. The basis for this NOEL is not available.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Thiobencarb is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised by oxidation and hydrolysis to the major metabolites, 4-chlorohippuric acid and 4-chlorobenzoic acid. Excretion was via urine and to lesser extent faeces, and was almost complete within 72 hours.

Acute effects: Thiobencarb has moderate acute oral toxicity and low acute dermal toxicity. It is not a skin sensitiser.

Short-term effects: A three-month oral study in rats reported increased liver weight and serum alkaline phospatase activity at dose levels of 33 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies in rats (2-year) and dogs (1-year) reported decreased plasma cholinesterase activity, reduced bodyweight gain, and evidence of anaemia at doses of 5 mg/kg bw/day and above. In a long-term dietary study in mice, liver discolouration was the only effect seen at high dose levels. The lowest overall NOEL was 1 mg/kg bw/day (in both rats and dogs). This NOEL is the basis for the current ADI.

Carcinogenicity: Based on 18-month studies in mice and a 2-year study in rats, there is no evidence of carcinogenicity for thiobencarb.

Genotoxicity: Thiobencarb is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats, and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Poisons Schedule: Thiobencarb is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for thiobencarb was determined as follows:

$$0.04 \text{ mg/L} = \frac{1.0 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1.0 mg/kg bw/day is the NOEL based on a long-term dietary studies in dogs (1 year) and rats (2 years).
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Magara Y, Aizawa T, Matumoto N, Souna F (1994). Degradation of pesticides by chlorination during water-purification. *Water Science and Technology,* 30(7):119-128.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Quayle WC, Oliver DP, Zrna S (2006). Field dissipation and environmental hazard assessment of clomazone, molinate, and thiobencarb in Australian rice culture. *Journal of Agricultural and Food Chemistry,* 54(19):7213-7220.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Thiometon

(endorsed 2011)

GUIDELINE

Based on human health concerns, thiometon in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Thiometon (CAS 640-15-3) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes chlorpyrifos, terbufos, azinphos methyl (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, chemical would not be a health concern unless the concentration exceeded 0.004 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Thiometon is an insecticide and acaricide used to control lice, mites and sawflies on a wide variety of agricultural crops.

There are currently no products containing thiometon registered for use in Australia, but de-registered compounds may still be detected in water. After a review, the toxicological database was considered inadequate to support continued registration of products containing thiometon.

Exposure sources: If used in the future, the main source of public exposure to thiometon and its metabolites would be residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of any thiometon products in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of thiometon in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of thiometon in drinking water have been identified.

MEASUREMENT

The National Measurement Institute reports that gas chromatography with mass spectrometry can achieve a limit of reporting of 0.0001 mg/L for thiometon.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for thiometon is 0.001 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.12 and 0.15 mg/kg bw/day from long-term (2-year) dietary studies in rats and dogs, respectively. The NOEL is based on decreased erythrocyte cholinesterase activity. The ADI incorporates a safety factor of 100, and was first established in 1989.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Thiometon is readily and extensively absorbed via the gastrointestinal tract in rats. It is extensively metabolised, primarily through sulfoxidation pathways to form sulfoxides and sulfones. These metabolites are rapidly excreted in urine as sulfates, glucoronides, and glutathione conjugates, almost completely within 24 hours.

Acute effects: Thiometon has moderate acute oral and dermal toxicity. Skin sensitisation studies are unavailable, but it has not been associated with sensitisation during occupational exposure. Clinical signs of acute poisoning were typical of cholinesterase inhibition and included hyperexcitability, salivation, bronchoconstriction, headache, vomiting and other behavioural changes.

Short-term effects: In 90-day dietary studies in rats and dogs, cholinesterase inhibition was seen at doses of 1.5 mg/kg bw/day (rats) and 0.6 mg/kg bw/day (dogs). No other effects were seen up to the highest doses tested, 4.5 mg/kg bw/day in rats and 1.2 mg/kg bw/day in dogs.

Long-term effects: In 1- and 2-year dietary studies in dogs and rats, respectively, erythrocyte cholinesterase inhibition was seen at doses of 0.3 mg/kg bw/day (dogs) and 1.5 mg/kg bw/day (rats) in 1- and 2-year dietary studies. Plasma cholinesterase inhibition was the only other effect seen up to the highest dose tested, 1.2 mg/kg bw/day, in dogs. In rats, increased serum alkaline phosphatase activity, and decreased serum cholesterol, protein, and glucose, and brain cholinesterase activity was seen at the highest dose tested, 14.4 mg/kg bw/day. The NOELs were 0.12 mg/kg bw/day (rats) and 0.15 mg/kg bw/day (dogs), and these are the basis for the current ADI of 0.001 mg/kg bw/day.

Carcinogenicity: Based on a 2-year study in rats, there is no evidence of carcinogenicity for thiometon.

Genotoxicity: Thiometon is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Three-generation reproduction studies in rats, and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: No delayed developmental toxicity was seen in a 21-day dietary study in chickens up to the highest dose tested, 4 mg/kg bw/day.

Poisons Schedule: Thiometon is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

NOTE: Important general information is contained in PART II, Chapter 6

The health-based guideline of 0.004 mg/L for thiometon was determined as follows:

$$0.004 \text{ mg/L} = \frac{0.12 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.12 mg/kg bw/day is the NOEL based on a long-term (2-year) dietary study in rats. This NOEL is closely supported by a NOEL of 0.15 mg/kg bw/day from a long-term (2-year) dietary study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

Thiram

(endorsed 2011)

GUIDELINE

Based on human health concerns, thiram in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Thiram (CAS 137-26-8) belongs to the dimethyldithiocarbamate class of chemicals. Another pesticide in this class is ziram (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, thiram would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Thiram is used as a fungicide for the control of disease in turf, ornamentals, vines and agricultural crops or as an antifouling agent on industrial equipment and boats.

There are registered products that contain thiram in Australia. The products are intended for professional and/or home garden use and are available as concentrated solutions. They are applied diluted as a seed dressing prior to sowing, as a drench to seedbeds, or as a spray using hand-held or ground boom equipment. They are also applied as concentrated paint using brushes, rollers or air spray equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to thiram and its metabolites are the use of home garden products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of thiram may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data on the occurrence of thiram in Australian waters could be found. Thiram adsorbs strongly to soils. In the USA, thiram is predicted at concentrations of approximately 4.3 and 0.84 µg/L in surface water and groundwater, respectively (USEPA 2004).

TREATMENT OF DRINKING WATER

No information on the efficiency of drinking water treatment processes to remove thiram could be found.

MEASUREMENT

Thiram in water can be analysed by a variety of methods (Sharma *et al.* 2003). Using high-performance liquid chromatography coupled with enrichment with a minicolumn allows determination of thiram to 0.5 µg/L (Suzuki *et al.* 1993).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for thiram is 0.004 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.4 mg/kg bw/day from a long-term (2-year) dietary study in dogs. The NOEL is based on neurological disturbances, anaemia and changes in the liver. The ADI incorporates a safety factor of 100, and was established in 1995.

The previous ADI for thiram was 0.001 mg/kg bw. It was amended in 1995 following the evaluation of new studies that addressed the inadequacies of the toxicological database.

The previous *Australian Drinking Water Guidelines* health value was 0.003 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Thiram is well absorbed via the gastrointestinal tract and is widely distributed in tissues and blood. It is extensively metabolised and excreted mainly in expired air and urine, with smaller amounts detected in the faeces. The majority of the dose is eliminated within 48 hours in rats.

Acute effects: Thiram has low to moderate acute oral toxicity and low acute dermal toxicity. It is a skin sensitizer in guinea pigs and is associated with allergic dermatitis in humans following occupational use.

Short-term effects: Short-term dietary studies were conducted in mice, rats and dogs. In both mice and rats, there was reduced food consumption and reduced bodyweight gain at 25 mg/kg bw/day and above. In rats, there was also evidence of anaemia and changes in clinical chemistry parameters indicative of liver/kidney damage. Histopathological changes in the stomach at 25 mg/kg bw/day and degenerative changes in the testes at 90 mg/kg bw/day were also observed. In dogs, there were reduced erythrocyte counts at 2.3 mg/kg bw/day as well as other haematological and clinical chemistry changes at higher dose levels.

Long-term effects: Long-term dietary studies have been conducted in mice, rats and dogs. In mice, there was reduced bodyweight gain, evidence of anaemia and irritation of the stomach at 57 mg/kg bw/day and above. In rats, there was pancreatic atrophy, bile duct hyperplasia, and extramedullary haematopoiesis in the liver and spleen at 1.5 mg/kg bw/day and above. At higher doses, there was evidence of neurotoxicity, anaemia, and benign neoplastic lesions in the liver and thyroid. In dogs, there were clinical chemistry changes indicative of liver effects at 2.6 mg/kg bw/day. Histopathological changes in the liver, as well as anaemia and clinical signs of neurotoxicity, were reported at 4 mg/kg bw/day. At the highest dose of 40 mg/kg bw/day, clonic convulsions, severe anaemia and ophthalmological effects were reported. The NOEL for neurological, anaemic and liver effects in the dog was 0.4 mg/kg bw/day, and is the basis for the current ADI.

Carcinogenicity: Long-term dietary studies in rats reported benign neoplastic lesions in the liver, thyroid and retina. These effects occurred at doses well in excess of the likely level of human exposure.

Genotoxicity: Thiram produced both positive and negative results in *in vitro* and *in vivo* short-term assays. The weight of evidence indicates that it is weakly genotoxic.

NOTE: Important general information is contained in PART II, Chapter 6

Reproductive and developmental effects: A two-generation reproduction study in rats and developmental toxicity studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: Short-term dietary studies in chicks and long-term dietary studies in rats and dogs reported symptoms indicative of nervous system toxicity, but only at dose levels well in excess of the likely level of human exposure.

Poisons Schedule: Thiram is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.007 mg/L for thiram was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.4 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 200}$$

where:

- 0.4 mg/kg bw/day is the NOEL based on a long-term (2-year) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 200 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation. With an additional safety factor of 2 to address the additional uncertainty in relation to the carcinogenicity of thiram.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Sharma VK, Aulakh JS, Malik AK (2003). Thiram: degradation, applications and analytical methods. *Journal of Environmental Monitoring,* 5:717-723.

Suzuki T, Yaguchi K, Kano I (1993). Screening methods for asulam, oxine-copper and thiram in water by high-performance liquid chromatography after enrichment with a minicolumn. *Journal of Chromatography A,* 643(1-2):173-179.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (2004). Reregistration eligibility decision (RED) for Thiram. EPA 738-R-04-012. USEPA.

NOTE: Important general information is contained in PART II, Chapter 6

Tin

(endorsed 2011)

GUIDELINE

No guideline value is considered necessary for tin in drinking water, as concentrations are likely to be considerably lower than the level that can cause health effects.

GENERAL DESCRIPTION

Tin is mainly used for plating. Tin coatings are used in the manufacture of food containers and in food processing equipment. Tin is also used in alloys such as solders, bronzes and pewters. Inorganic tin compounds are used as pigments in the ceramic and textile industries. Organic tin compounds are used as biocides (see Fact Sheet on organotins).

The concentration of tin in rivers, estuaries and oceans is generally less than 0.000005 mg/L (5 ng/L), but in some instances has been measured up to 0.002 mg/L. The use of organotin biocides can produce significantly higher concentrations in environmental waters. Levels of <0.042–0.3 mg/L were found in 37 different bottled mineral waters. A mean range of 0.001– 0.002 mg/L (maximum 0.030 mg/L) was found in a survey of water supplies in the United States; values greater than 0.002 mg/L are exceptional (WHO 2004, IPCS 2005).

Food, and particularly canned food, is the major source of human exposure to tin. Intake from this source can vary widely and estimates range from 0.1 mg per day up to 100 mg per day, with a median of 0.2 mg per day.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Australian drinking water supplies have not been routinely monitored for tin. Based on measurements made overseas, it is likely that concentrations would be extremely low.

TREATMENT OF DRINKING WATER

Treatment of drinking water to reduce the concentration of inorganic tin is unlikely to be required.

MEASUREMENT

The concentration of tin in drinking water can be determined using graphite furnace atomic absorption spectroscopy (APHA Method 3500-Sn part B 1992). The limit of determination is 0.01 mg/L.

HEALTH CONSIDERATIONS

Tin is thought to be an essential element in animals. It is not known whether it is essential for humans.

Tin, or tin salts, are poorly absorbed from the gastrointestinal tract. Most studies indicate that less than 5% is absorbed. Highest concentrations of tin occur in the bone, kidney and liver. Biological half-lives range from 1 to 4 months, and tin is excreted primarily via the kidneys and bile.

Extensive reviews and summaries of the human and animal toxicity data for tin are available (JECFA 2000, WHO 2004, IPCS 2005, ATSDR 2005).

There is no evidence of adverse effects in humans associated with long-term exposure to tin. The main effects, due to consumption of canned food with high tin concentrations (over 150 mg/kg), are gastric irritation resulting in vomiting, diarrhoea, fatigue and headache.

In animals, long-term ingestion studies over 2 years using rats and mice reported no significant adverse effects.

Inorganic tin in the form of stannous chloride was found not to be mutagenic in tests with bacteria; however, in mammalian cells *in vitro*, inorganic tin has induced DNA and chromosomal aberrations.

DERIVATION OF GUIDELINE

The low toxicity of tin and inorganic tin compounds is due largely to low absorption, low tissue accumulation and rapid excretion. A guideline value of approximately 0.7 mg/L could be derived from a 2-year feeding study with rats (WHO 1982), but this value is approximately three orders of magnitude higher than tin concentrations in drinking water. Therefore, the presence of tin in drinking water does not represent a hazard to human health and the establishment of a guideline value is not deemed necessary.

REFERENCES

APHA Method 3500-Sn Part B (1992). Tin: Atomic Absorption Spectrometric method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

ATSDR (Agency for Toxic Substances and Disease Registry) (2005). Toxicological Profile for Tin and Tin Compounds. ATSDR, US Department Of Health and Human Services Public Health Service. PB2006-100006

IPCS (International Programme on Chemical Safety) (2005). Concise International Chemical Assessment Document 65. Tin and Inorganic Tin Compounds. IPCS, World Health Organization Geneva, Switzerland.

JECFA (Joint Expert Committee on Food Additives) (2000). Who Food Additives Series 46:Tin (Addendum). JEFCA, World Health Organization.

WHO (World Health Organization) (2004). Inorganic Tin in Drinking-water: Background document for development of WHO *Guidelines for Drinking-water Quality*. WHO, Geneva, Switzerland. WHO/SDE/WSH/03.04/115.

NOTE: Important general information is contained in PART II, Chapter 6

Toltrazuril

(endorsed 2011)

GUIDELINE

Based on human health concerns, toltrazuril in drinking water should not exceed 0.004 mg/L.

RELATED CHEMICALS

Toltrazuril (CAS 69004-03-1) belongs to the triazinetrione class of chemicals. There are no other pesticides in this class (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, toltrazuril would not be a health concern unless the concentration exceeded 0.004 mg/L. Excursions above this level even for a relatively short period are of concern, as the health-based guideline is partially based on short-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Toltrazuril is a coccidiostat for the control of protozoa infection (coccidiosis) in poultry, young cattle and piglets.

There are registered products that contain toltrazuril in Australia. The products are intended for professional use and are available as concentrated formulations to be applied to drinking water and feed provided to animals. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to toltrazuril is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

The veterinary use of toltrazuril provides some potential for contamination of drinking water through the washing of equipment near dams, streams or watercourses.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on toltrazuril occurrence in Australian drinking water supplies were found. However, there is an identified risk that toltrazuril may reach groundwater after spreading of contaminated poultry manure on agricultural land. (EMEA 2008).

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of toltrazuril in drinking water, but it is expected that advanced treatment methodologies such as ozonation, reverse osmosis and advanced oxidation would be effective.

MEASUREMENT

Toltrazuril is a veterinary product and the majority of analytical methods have been developed to determine residue concentrations in animal tissues. A high performance liquid chromatography (HPLC) analytical method based on fluorescence detection has been used to determine toltrazuril concentrations in tissues and plasma (EMEA 2008). Determination of toltrazuril in eggs by HPLC with ultraviolet detection or HPLC with tandem mass spectrometric detection can achieve a limit of quantitation of 30 µg/kg and 1 µg/kg respectively (Mulder *et al.* 2004). No published reports on methods for analysis of toltrazuril in drinking water were found.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for toltrazuril is 0.01 mg per kg of bodyweight (mg/kg bw), based on a lowest-observed-effect level (LOEL) of 1 mg/kg bw/day from a 2-year dietary study in rats. The LOEL is based on the occurrence of pre-neoplastic uterine lesions. The ADI incorporates a safety factor of 100, and was first established in 1993.

An *Australian Drinking Water Guidelines* health value has not been previously established.

HEALTH CONSIDERATIONS

Metabolism: Toltrazuril is rapidly and moderately well absorbed via the gastrointestinal tract. It is extensively metabolised by sulfoxidation of the trifluoromethyl-thio group to form sulfoxide and sulfones. Excretion is in faeces and urine, and is almost complete within 7 days.

Acute effects: Toltrazuril has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 3-month dietary studies in rats and dogs, there was anaemia and organ weight changes (testes, liver and kidney) at 4.2 mg/kg bw/day in rats and dogs; decreased prostate weights at 13.5 mg/kg bw/day in dogs; and decreased bodyweight gain and effects on the liver at 16.6 mg/kg bw/day in rats.

Long-term effects: Long-term dietary studies were conducted in mice and rats. In rats, there was an increased incidence of pre-neoplastic nodular changes in the uterus at 1 mg/kg bw/day and decreased bodyweight gain and increased uterine fluid and distension of the uterus at 3 mg/kg bw/day. In mice, anaemia was seen at 41 mg/kg bw/day. No other effects were seen. The LOEL of 1 mg/kg bw/day is the basis for the current ADI.

Carcinogenicity: There was an increase in pre-neoplastic lesions in the rat uterus at the lowest dose tested of 1 mg/kg bw/day and above. This dose level is well in excess of the likely level of human exposure. Uterine adenomas were observed at the next highest dose tested of 3 mg/kg bw/day and above.

Genotoxicity: Toltrazuril is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats reported evidence of maternotoxicity (decreased bodyweight gain) and pup toxicity (increased pup mortality and decreased bodyweight gain) at 4 mg/kg bw/day. A developmental toxicity study in rabbits reported maternotoxicity, and increased foetal death and abortion rates at 3 mg/kg bw/day, but no evidence of teratogenicity.

Poisons Schedule: Toltrazuril is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

NOTE: Important general information is contained in PART II, Chapter 6

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.004 mg/L for toltrazuril was determined as follows:

$$0.004 \text{ mg/L} = \frac{1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 1 mg/kg bw/day is the LOEL based on a long-term (2-year) dietary study in rats, and is the NOEL from the reproduction study in rats and the developmental toxicity study in rabbits.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 1000 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation, with an additional safety factor of 10 to account for the uncertainty in the ADI, which is based on a LOEL for pre-neoplastic uterine lesions.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

EMEA (European Medicines Agency) (2008). Committee for Medicinal Products for Veterinary Use – Opinions on Veterinary Medicinal Products. Lomdon.

Mulder PPJ, Balzer-Rutgers P, Brinke EM, Bolck YJC, Berendsen BJA, Gerçek H, Schat B, JA v Rhijn (2004). Deposition and depletion of the coccidiostats toltrazuril and halofuginone in eggs. *Analytica Chimica Acta,* 29(1-2):331-337.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Toluene [CASRN 108-88-3]

(endorsed 2013)

GUIDELINE

Based on aesthetic considerations (taste and odour), the concentration of toluene in drinking water should not exceed 0.025 mg/L.

Based on health considerations the concentration of toluene should not exceed 0.8 mg/L.

GENERAL DESCRIPTION

Toluene is a colourless liquid, which occurs naturally as a component of crude oil and is present in petrol. It constitutes approximately 5-8% of unleaded gasoline by volume. It can enter water sources through atmospheric deposition, by leaching from synthetic coatings used to protect storage tanks, and by point-source pollution.

Toluene, also known as methylbenzene is produced in large quantities during petroleum refining and is a byproduct in the manufacture of styrene and coke-oven preparations. Toluene is also used as solvent in paints adhesives and nail polish. It also occurs in natural gas and emissions from volcanoes, forest fires, and cigarettes.

Toluene has a taste and odour threshold at 0.025mg/L.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Toluene has only rarely been identified in Australian drinking waters. Natural concentrations in most water sources are usually very low. Toluene can occur naturally in groundwater as a result of proximity to, or contact with, coal seams, petroleum and gas deposits, and shales. It may be mobilised by extraction activities (Lesage *et al*., 1997; Leusch and Bartkow, 2011; Volk *et al*, 2011). However, contamination can occur, usually via exposure to petrochemicals in surface waters or groundwaters. Known sources of groundwater contamination include leakage from sub-surface fuel storage tanks (do Rego & Netto, 2007). Emissions of fuel components from boating use is a known source of contamination of multiple-use lakes and reservoirs (Schmidt *et al*., 2004). Toluene was reported in 19% of samples from an extensive groundwater survey undertaken in Denmark with the highest concentration being 0.002 mg/L (Juhler & Felding, 2003). Toluene has been detected in well water at 0.005 to 0.1 mg/L in the USA (IPCS, 1985). Groundwater from contaminated wells in the USA contained up to 3.5 mg/L of toluene (ATSDR, 2000). Toluene has been reported at up to 0.001 mg/L in municipal drinking water in Croatia (Karaconji *et al*., 2006), up to 0.027 mg/L in Canada (average 0.002 mg/L; IPCS, 1985), up to 0.063 mg/L in municipal drinking water in Taiwan (Kuo *et al*., 1997) and is occasionally detected in drinking waters in the USA (Williams *et al*., 2004) up to 0.011 mg/L (IPCS, 1985).

TREATMENT OF DRINKING WATER

Volatile organic chemicals such as toluene are most commonly treated in drinking water by aeration stripping and/or adsorption to granular activated carbon (GAC). A conventional biologically active sand filter has been shown to be highly effective for the removal of toluene from contaminated water, under suitable conditions (Arvin *et al*., 2004). Effective bioremediation of highly contaminated groundwaters has also been demonstrated (Sedran *et al*., 2004; Zein *et al*., 2006).

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for the analysis of toluene (APHA, AWWA & WEF, 2012). An inert gas is bubbled through the sample and toluene is trapped on an adsorbent. The adsorbent is then heated and toluene analysed using gas chromatography with mass spectrometric (GC-MS) detection (Method 6200 B) or photoionisation (PI) detection (Method 6200 C) (APHA, AWWA & WEF, 2012). The method detection limit is 47 ng/L for GC-MS and 23 ng/L for GC-PI (APHA, AWWA & WEF, 2012).

HEALTH CONSIDERATIONS

In humans, toluene is readily absorbed from the gastrointestinal tract after ingestion, and is distributed preferentially in adipose tissue, then the kidneys, liver and brain. It is rapidly metabolised by the liver to benzyl alcohol, benzoic acid, and to a lesser extent, phenols.

Data on human health effects come mainly from inhalation studies. The predominant effects of acute exposure were impairment of the central nervous system and irritation of the mucous membranes, with fatigue and drowsiness being the most obvious symptoms (ATSDR, 2000).

Rats exposed to toluene vapour for 2 years exhibited decreased blood haematocrit values at high toluene concentrations (380 ppm in air). No data are available on long term oral toxicity; however, a 13 week gavage study using rats and mice reported increased liver weights at doses from 625 mg/kg body weight per day (NTP 1990).

Toluene generally did not exhibit genotoxic activity in tests on bacteria, yeast cells, and mammalian cells in vitro.

The International Agency for Research on Cancer has concluded that toluene is not classifiable as to its carcinogenicity in humans (Group 3, inadequate evidence in humans and in animals) (IARC, 1989).

DERIVATION OF GUIDELINE

The USEPA (2009) has set a drinking water guideline of 1.0 mg/L for toluene, while the WHO (2011) proposes a guideline of 0.7 mg/L.

The health-based guideline value of 0.8 mg/L for toluene in drinking water was determined as follows:

$$0.8 \text{ mg/L} = \frac{312 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000} \times \frac{5}{7}$$

where:

- 312 mg/kg body weight per day is the no effect level based on a 13-week oral study using rats (NTP 1990)
- 70 kg is the average weight of an adult
- 0.1 is the proportion of total daily intake attributable to the consumption of water
- 2 L/day is the average amount of water consumed by an adult
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 because a less than lifetime study was used)
- 5/7 is used to convert data based on a 5 day per week gavage study to a 7-day week equivalent.

This health-based guideline value exceeds the taste threshold of toluene in water of 0.025 mg/L.

The WHO guideline value of 0.7 mg/L is based on an adult body weight of 60 kg. The difference in guideline values is not significant.

NOTE: Important general information is contained in PART II, Chapter 6

REFERENCES

American Public Health Association (APHA), American Water Works Association (AWWA) and Water Environment Federation (WEF) (2012). Standard Methods for the Examination of Water and Wastewater, 22nd Edition. Eds. Rice EW, Baird RB, Eaton AD and Clesceri LS.

Arvin, E., Engelsen, P. and Sebber, U. (2004) Biodegradation of gasoline compounds (BTEX) in a water works sand filter. W*ater Science & Technology: Water Supply*, **4**(5-6), 29-33.

ATSDR (2000). Toxicological profile for toluene.US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, USA.

do Rego, E. C. P. and Netto, A. D. P. (2007) PAHs and BTEX in groundwater of gasoline stations from Rio de Janeiro City, Brazil. *B. Environ. Contam. Tox.*, **79**(6), 660-664.

IARC (International Agency for Research on Cancer) (1989) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: some organic solvents, resin monomers and related compounds, pigments and occupational exposures in paint manufacture and painting., Lyon.

IPCS (1985). Environmental Health Criteria 52: Toluene. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland.

Juhler, R. K. and Felding, G. (2003) Monitoring methyl tertiary butyl ether (MTBE) and other organic micropollutants in groundwater: Results from the Danish National Monitoring Program. *Water Air Soil Poll.*, **149**(1-4), 145-161.

Karaconji, B., Skender, L. and Karacic, V. (2006) Benzene, toluene, ethylbenzene, and isomeric xylenes in various water samples in Croatia. *B. Environ. Contam. Tox.*, **76**(3), 458-462.

Kuo, H. W., Chiang, T. F., Lo, L. I., Lai, J. S., Chan, C. C. and Wang, J. D. (1997) VOC concentration in Taiwan's household drinking water. *Sci. Total Environ.*, **208**(1-2), 41-47.

Lesage, S., Hao, X., and Kent, S. (1997) Distinguishing Natural Hydrocarbons from Anthropogenic Contamination in Ground Water. *Groundwater*, 35(1), 149-160

Leusch, F. and Bartkow, M. (2010) A short primer on benzene, toluene, ethylbenzene and xylenes (BTEX) in the environment and in hydraulic fracturing fluids http://www.ehp.qld.gov.au/management/coal-seam-gas/pdf/btex-report.pdf (accessed 19 March 2013)

NTP (National Toxicology Program) United States Department of Health and Human Services (1990) Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F1 mice. NTP Report No 371. NIH Publication No. 90 2826.

Schmidt, T. C., Haderlein, S. B., Pfister, R. and Forster, R. (2004) Occurrence and fate modeling of MTBE and BTEX compounds in a Swiss Lake used as drinking water supply. *Water Res.*, **38**(6), 1520-1529.

Sedran, M. A., Pruden, A., Wilson, G. J., Suidan, M. T. and Venosa, A. D. (2004) Biodegradation of methyl tert-butyl ether and BTEX at varying hydraulic retention times. *Water Environ. Res.*, **76**(1), 47-55.

Volk, H., Pinetown, C., Johnston,W. (2011) A desktop study of the occurrence of total petroleum hydrocarbon (TPH) and partially water-soluble organic compounds in Permian coals and associated coal seam groundwater. CSIRO Petroleum and Geothermal Research Portfolio Report EP-13-09-11-11 **2011** (Bentley, WA, Australia). Available at http://www.agl.com.au/Downloads/Literature%20Review% (accessed 19 March 2013)

Williams, P. R. D., Benton, L. and Sheehan, P. J. (2004) The risk of MTBE relative to other VOCs in public drinking water in California. *Risk Anal.*, **24**(3), 621-634.

NOTE: Important general information is contained in PART II, Chapter 6

Zein, M. M., Suidan, M. T. and Venosa, A. D. (2006) Bioremediation of groundwater contaminated with gasoline hydrocarbons and oxygenates using a membrane-based reactor. *Environ. Sci. Technol.*, **40**(6), 1997-2003.

Total dissolved solids

(endorsed 2011)

GUIDELINE

No specific health guideline value is provided for total dissolved solids (TDS), as there are no health effects directly attributable to TDS. However for good palatability total dissolved solids in drinking water should not exceed 600 mg/L.

GENERAL DESCRIPTION

Total dissolved solids (TDS) consist of inorganic salts and small amounts of organic matter that are dissolved in water. Clay particles, colloidal iron and manganese oxides and silica, fine enough to pass through a 0.45 micron filter membrane can also contribute to total dissolved solids.

Total dissolved solids comprise: sodium, potassium, calcium, magnesium, chloride, sulfate, bicarbonate, carbonate, silica, organic matter, fluoride, iron, manganese, nitrate, nitrite and phosphates.

The palatability of drinking water can be rated according to TDS concentrations and a breakdown is provided below, based on World Health Organization guidelines (WHO 2004):

TDS (mg/L)	Palatability
0 – 600	good
600 – 900	fair
900 – 1200	poor
> 1200	unacceptable (unpalatable)

Precisely what level of TDS an individual water supply system decides to accept is a function of community acceptance, available water resources, and the cost and practicality of effecting any change to natural TDS levels.

High TDS values may be associated with excessive scaling in pipes, fittings and household appliances. Water with very high or very low TDS may also be corrosive.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian cities, TDS values can range from below 100 mg/L to more than 750 mg/L; regional supplies can have TDS values up to 1000 mg/L and some rural and remote communities may have TDS in excess of 1000 mg/L, owing mainly to groundwater characteristics.

MEASUREMENT

Three methods can be used to determine to determine TDS. The most accurate entails a complete analysis of the sample and summation of the concentration of all the anions and cations. The most common and least expensive method is to convert electrical conductivity measurements to TDS values by multiplication by a factor that varies with the type of water (APHA Method 2510A 1992). Gravimetric measurement (i.e. by evaporation and weighing) can also be used (APHA Method 2540C 1992). As a rough guide, electrical conductivity, measured in micro Siemens per cm ($\mu S.cm^{-1}$, also known as EC, or electroconductivity units), is multiplied by 0.64 to estimate TDS. The relationship, however, is dependant

on both chemistry and temperature, and factors of between 0.50 and 0.64 are used across Australia. The factor is also likely to vary between raw and treated water. Inferring a TDS value therefore has to be based on local circumstances.

TREATMENT OF DRINKING WATER

It is difficult to remove dissolved solids from drinking water. Suitable technologies include reverse osmosis, ion exchange, and distillation, but all of these require considerable energy input and can be expensive to operate. Lime softening may also be effective where high TDS is mainly due to hardness.

HEALTH CONSIDERATIONS

No health effects have been associated specifically with high TDS concentrations. The health effects of individual components of TDS are discussed separately in the discussions on inorganic chemicals (Section 6.3.1 and relevant Fact Sheets). Indirectly, high TDS water, being less palatable than that with a low TDS, might discourage consumers from drinking tap water, leading to use of potentially less healthy water (from alternative sources, natural or manufactured) and/or other less healthy drinks.

GUIDELINES IN OTHER COUNTRIES

The 2004 World Health Organization guidelines do not specify a value for TDS but do describe values according to palatability.

The European Communities (1998) Directive 98/83/EC nominates a conductivity value of 2500 $\mu S.cm^{-1}$ at 20ºC and, in a footnote, requires that the water not be aggressive. An online converter function (Lenntech 2010) converts that value to 1600 mg/L of TDS (the standard for EC is intended to be at 25ºC).

The United States Environmental Protection Agency nominates a TDS level of 500 mg/L in its list of National Secondary Drinking Water Regulations (USEPA 2009) and explains the category as follows:

> ***National Secondary Drinking Water Regulations (NSDWRs or secondary standards) are non-enforceable guidelines regulating contaminants that may cause cosmetic effects (such as skin or tooth discoloration) or aesthetic effects (such as taste, odor, or color) in drinking water. EPA recommends secondary standards to water systems but does not require systems to comply. However, states may choose to adopt them as enforceable standards.***

The Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment (2008) provides Guidelines for Canadian Drinking Water Quality, and a TDS level of 500 mg/L is nominated as an "aesthetic objective".

REFERENCES

APHA Method 2510A (1992). Conductivity: Laboratory method. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

APHA Method 2540C (1992). Total Dissolved Solids Dried at 180ºC. *Standard Methods for the Examination of Water and Wastewater*, 18th edition. American Public Health Association, Washington.

Drinking Water Federal-Provincial-Territorial Committee on Drinking Water of the Federal-Provincial-Territorial Committee on Health and the Environment (2008). Guidelines for Canadian Drinking Water Quality http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/sum_guide-res_recom/chemical-chimiques-eng.php#tbl4 (accessed 17 May 2009).

European Communities (1998). Council Directive 98/83/EC of 3 November 1998 on the quality of water intended for human consumption. http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:1998:330:0032:0054:EN:PDF (accessed 17 May 2009).

Lenntech (2010). Online converter-calculator http://www.lenntech.com/unit-conversion-calculator/tds_engels.htm (accessed 17 May 2009).

USEPA (United States Environmental Protection Agency) (2009). National Secondary Drinking Water Regulations http://www.epa.gov/safewater/contaminants/index.html#sec (accessed 17 May 2009).

WHO (World Health Organization) (2004) Guidelines for Drinking Water Quality http://www.who.int/water_sanitation_health/dwq/GDWQ2004web.pdf (accessed 17 May 2009).

NOTE: Important general information is contained in PART II, Chapter 6

Triadimefon

(endorsed 2011)

GUIDELINE

Based on human health concerns, triadimefon in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Triadimefon (CAS 43121-43-3) belongs to the triazole class of chemicals. Another pesticide in this class is amitrole (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, triadimefon would not be a health concern unless the concentration exceeded 0.09 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Triadimefon is a fungicide for the control of powdery mildews, rusts and other fungal diseases in turf and agricultural crops.

There are registered products that contain triadimefon in Australia. The products are intended for professional use and are available as wettable powders or concentrated solutions, to be applied as a diluted or concentrated spray using ground rig or aerial application, or added to fertilizer and applied using truck mounted equipment. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to triadimefon and its metabolites is residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of triadimefon may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of triadimefon in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

No specific data on the treatment of triadimefon in drinking water have been identified.

NOTE: Important general information is contained in PART II, Chapter 6

MEASUREMENT

Triadimefon can be measured in water using high performance liquid chromatography with tandem mass spectrometry (Schermerhorn *et al*. 2005). The limit of quantitation for this technique is 0.5 ng/L.

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for triadimefon is 0.03 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.5 mg/kg bw/day from a long-term rat dietary study. The NOEL is based on haematological effects. The ADI incorporates a safety factor of 100, and was established in 1987.

The previous *Australian Drinking Water Guidelines* health value was 0.002 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Triadimefon is readily and extensively absorbed via the gastrointestinal tract of mice and rats. It undergoes some metabolism in rats, and 80% is excreted in the urine and faeces in 7 days. Triadimenol was the major metabolite recovered.

Acute effects: Triadimefon has low to moderate acute oral toxicity and low dermal toxicity. It is not a skin sensitiser.

Short-term effects: A 1-month oral study in rats reported increased relative and absolute liver weights with no associated liver pathology at dose levels of 10 mg/kg bw/day and above.

Medium-term studies conducted in rats and dogs reported no toxicological effects up to doses of 100 mg/kg bw/day in rats, and hepatic enzyme induction at doses of 15 mg/kg bw/day and above in dogs. Pathological effects in the liver were not observed.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs reported liver toxicity and effects on haematological parameters to be the main toxicological effects. In mice, there was an increase in erythrocyte counts, haemoglobin and haematocrit concentrations, liver weights, and hyperplastic liver nodules (indicative of liver toxicity) at 90 mg/kg bw/day. In rats, decreased body weight gain and reduced erythrocyte counts and haemoglobin levels were observed at doses of 25 mg/kg bw/day and above. The NOEL of 2.5 mg/kg bw/day from this study forms the basis for the current ADI.

A 2-year dietary study in dogs reported reduced bodyweight gain and microsomal enzyme induction in the liver at doses of 25 mg/kg bw/day and above, with no pathological changes.

Carcinogenicity: Based on 2-year studies in mice, rats and dogs there is no evidence of carcinogenicity for triadimefon.

Genotoxicity: Only *in vitro* short-term studies were available, and these show no evidence of genotoxicity. No *in vivo* studies were conducted.

Reproductive and developmental effects: In 2- and 3-generation reproduction studies in rats, there was evidence of maternotoxicity and effects on reproductive parameters, but only at doses well in excess of the likely level of human exposure. Developmental studies in rats and rabbits did not produce any evidence of effects on foetal development.

Neurotoxicity: Neurological studies conducted in mice and rats reported no adverse effects up to doses of 3 mg/kg bw/day.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Triadimefon is included in Schedule 5 and 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010), depending on its concentration and use. Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.09 mg/L for triadimefon was determined as follows:

$$0.09 \text{ mg/L} = \frac{2.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.5 mg/kg bw/day is the NOEL based on a long-term (2-year) study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Schermerhorn PG, Golden PE, Krynitsky AJ, Leimkuehler WM (2005). Determination of 22 triazole compounds including parent fungicides and metabolites in apples, peaches, flour, and water by liquid chromatography/tandem mass spectrometry. *Journal of AOAC International,* 88(5):1491-1502.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

NOTE: Important general information is contained in PART II, Chapter 6

Trichlorfon

(endorsed 2011)

GUIDELINE

Based on human health concerns, trichlorfon in drinking water should not exceed 0.007 mg/L.

RELATED CHEMICALS

Trichlorfon (CAS 52-68-6) belongs to the organophosphate class of chemicals. There are many other pesticides in this class, which includes dichlorvos, diazinon, and phorate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, trichlorfon would not be a health concern unless the concentration exceeded 0.007 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Trichlorfon is used as an insecticide for the control of insect pests in home garden lawns, sports fields and agricultural crops, and as an acaricide for the control of parasites in livestock, and household and commercial aquariums.

There are registered products that contain trichlorfon in Australia. The products are intended for professional and home garden use. They are available as concentrated solutions to be diluted and applied by aerial and ground sprays to crops as an insecticide. When used as an acaricide, trichlorfon is supplied as pellets for addition to aquarium water, and as pellets or paste for oral drenching of livestock. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The potential sources of public exposure to trichlorfon and its metabolites are the use of home garden and aquarium products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of chemical may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater. When trichlorfon is added to water, it degrades to dichlorvos.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No reports of trichlorfon in Australian drinking waters have been identified.

TREATMENT OF DRINKING WATER

There is insufficient information on the treatment of trichlorfon in drinking water, but it is expected that advanced treatment methodologies such as ozonation and advanced oxidation would be effective.

MEASUREMENT

Several methods have been reported for the analysis of trichlorfon in water, including high performance liquid chromatography with ultraviolet detection, with a limit of detection (LOD) of 2.0 µg/L (Zhu *et al.* 2008); liquid chromatography with mass spectrometry-mass spectrometry, LOD 10 µg/L (Geiss *et al.* 2006); liquid chromatography with ionspray mass spectrometry, LOD 0.2 µg/L (Molina *et al.* 1996); and gas chromatography with mass spectrometry, LOD 0.73 µg/L (Molto *et al.* 1991).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for trichlorfon is 0.002 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.2 mg/kg bw/day from a long-term (10-year) dietary study in monkeys. The NOEL is based on inhibition of cholinesterase. The ADI incorporates a safety factor of 100, and was established in 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.005 mg/L (NHMRC and NRMMC, 2004).

HEALTH CONSIDERATIONS

Metabolism: Triclorfon is extensively and rapidly absorbed from the gastrointestinal tract and distributed widely in the body. Under normal alkaline physiological conditions, it is converted to dichlorvos by a non-enzymatic process. Following metabolism, excretion is rapid, primarily via the urine, with a half-life of about 80 minutes.

Acute effects: Triclorfon has moderate acute oral toxicity and low acute dermal toxicity. It is a skin sensitiser in guinea pigs. Clinical symptoms of toxicity were typical of cholinesterase inhibition and included tremors, prostration, coma, piloerection, ataxia, and salivation.

Short-term effects: In short-term oral studies (12-26 weeks) in rats, dogs and monkeys, the most significant effect was on the nervous system, with cholinesterase depression reported at 10 mg/kg bw/day in rats and 1.25 mg/kg bw/day in dogs. A 12-week oral study in humans reported clinical signs of toxicity and cholinesterase depression at 0.2 mg/kg bw/day.

Long-term effects: In long-term dietary studies in rats, dogs and monkeys, the most significant effect was on the nervous system, with monkeys the most sensitive to cholinesterase depression, at 1 mg/kg bw/day. The NOEL was 0.2 mg/kg bw/day, and this is the basis of the ADI.

Carcinogenicity: Based on long-term studies in rats, there is no evidence of carcinogenicity for trichlorfon.

Genotoxicity: While positive in *in vitro* assays, the weight of evidence indicates that trichlorfon is not genotoxic *in vivo*.

Reproductive and developmental effects: Reproduction studies in rats and developmental studies in mice and rats did not show any evidence of effects on reproductive parameters or foetal development.

Neurotoxicity: There was no evidence of delayed neuropathy in chickens. Short-term neurotoxicity studies in rats, hens and monkeys, conducted with oral doses up to 200 mg/kg bw/day, confirmed that trichlorfon has an effect on the nervous system, with effects such as increased locomotor activity and decreased learning ability and nerve conduction velocity, impaired nerve conduction, demyelination of nerves and axonal degeneration observed at high doses.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Trichlorfon is included in Schedule 6 and in Schedule 4 (as metrifonate, for human therapeutic use) of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline value of 0.007 mg/L for trichlorfon was determined as follows:

$$0.007 \text{ mg/L} = \frac{0.2 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.2 mg/kg bw/day is the NOEL based on a long-term (10-year) dietary study in monkeys.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is a safety factor derived from a long-term study in monkeys. The safety factor of 100 incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variations..

The World Health Organization has not established a health-based guideline value for trichlorfon and it is excluded from the list of agricultural chemicals guideline value derivation because it is "unlikely to occur in drinking water" (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Geiss S, Gebert S (2006). Extraction of highly polar organophosphorus pesticides from water. *Acta Hydrochimica et Hydrobiologica*, 34:464-473.

Molina C, Grasso P, Benfenati E, Barcelo D (1996). Automated sample preparation with extraction columns followed by liquid chromatography-ionspray mass spectrometry. Interferences, determination and degradation of polar organophosphorus pesticides in water samples. *Journal of Chromatography A*, 737:47-58.

Molto JC, Pico Y, Font G, Manes J (1991). Determination of triazines and organophosphorus pesticides in waters amples using solid-phase extraction, *Journal of Chromatography*, 555:137-145.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Zhu H-Z, Liu W, Mao J-W, Yang MM (2008). Cloud point extraction and determination of trace trichlorfon by high performance liquid chromatography with ultraviolet-detection based on its catalytic effect on benzidine oxidising. *Analytica Chimica Acta*, 614:58-62.

NOTE: Important general information is contained in PART II, Chapter 6

Trichlorobenzenes
1,2,3-trichlorobenzene (1,2,3-TCB)
1,2,4-trichlorobenzene (1,2,4-TCB)
1,3,5-trichlorobenzene (1,3,5-TCB)

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (taste and odour), the concentration of trichlorobenzenes in drinking water, either individually or in total, should not exceed 0.005 mg/L.

Trichlorobenzenes would not be a health concern unless the concentration exceeded 0.03 mg/L.

GENERAL DESCRIPTION

Trichlorobenzenes are present in the environment mainly as a result of a variety of industrial processes. They have only occasionally been found in drinking water supplies overseas, and rarely above 0.001 mg/L. Food and air are the primary routes of exposure.

Taste and odour thresholds vary from 0.005 mg/L to 0.03 mg/L, depending on individual sensitivities and water temperature.

Industrial-grade TCB is more than 90% 1,2,4-TCB with the remainder 1,2,3-TCB. The compound has a wide variety of uses. It is used as a solvent for high-melting products, an electrical coolant, a lubricant and an insecticide, and in polyester dyeing and termiticide preparations.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

TCBs have not been found in Australian drinking waters. They are included here to provide guidance in the unlikely event of contamination, and because they have been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

The concentration of TCBs in drinking water can be reduced by adsorption onto granular activated carbon.

MEASUREMENT

TCBs can be analysed using a solvent extraction procedure (USEPA Method 612 1984). The TCBs are extracted using dichloromethane and analysed using gas chromatography with electron capture detection. The limit of determination for 1,2,4-TCB is 0.00005 mg/L (50 ng/L). The purge and trap method can also be used (USEPA Draft Method 502.1 1986).

HEALTH CONSIDERATIONS

The TCBs are readily absorbed from the gastrointestinal tract and distributed in fat, skin and the liver. In rats and rabbits the TCBs are metabolised into trichlorophenols and mercapturic acids.

An extensive review and summary of the human and animal toxicity data for chlorobenzenes is available (IPCS 1991).

There are very few studies on the effects of human exposure. TCBs have caused marked irritation of the

mucous membranes following inhalation over short periods of exposure. No data are available on the effects of long-term exposure.

Animal studies are of short-term duration. A 13-week study using rats reported that toxic effects of the three isomers were similar: low doses produced no adverse effects, but higher doses (77 mg/kg body weight per day) caused changes to the liver and thyroid.

No increase in the incidence of tumours was observed in longer-term animal studies. TCBs did not exhibit mutagenic activity in tests with bacteria.

DERIVATION OF GUIDELINE

As the three TCBs have similar toxic effects, the guideline value can be based on the total concentration of all the TCBs rather than on the individual compounds. The health-based guideline value for total TCBs in drinking water was determined as follows:

$$0.03 \text{ mg/L} = \frac{7.7 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 7.7 mg/kg body weight per day is the no-effect level from a 13-week dietary study using rats (Côté *et al*. 1988).
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for a less than lifetime study).

This health-based guideline value exceeds the taste and odour threshold of 0.005 mg/L.

The World Health Organization guideline value of 0.02 mg/L was based on an adult body weight of 60 kg. The difference in guideline values is not significant.

REFERENCES

Côté M, Chu I, Villeneuve DC, Secours VE, Valli VE (1988). Trichlorobenzenes: results of a 13 week feeding study in the rat. *Drug and Chemical Toxicology*, 11:11–28.

IPCS (International Programme on Chemical Safety) (1991). Chlorobenzenes other than hexachlorobenzene. Environmental Health Criteria, 128. World Health Organization, IPCS.

USEPA Method 612 (1984). Guidelines establishing test procedures for the analysis of pollutants under the Clean Water Act. Federal Register, 40, CFR Part 136, 43234–43442.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

1,1,1-Trichloroethane

(endorsed 1996)

GUIDELINE

Data are inadequate to set a guideline value for 1,1,1-trichloroethane in drinking water.

GENERAL DESCRIPTION

1,1,1-Trichloroethane may be present in drinking water as a result of contamination from industrial discharges and spills. In the United States, 1,1,1-trichloroethane has occasionally been found in water supplies at concentrations ranging from 0.0002 mg/L to 0.02 mg/L.

It is widely used as a cleaning solvent, and is used to clean electrical equipment, motors, electronic components, printed circuit boards, photographic film, and various metal and plastic parts. It is also used as a lubricant in metal-cutting oils and as a component in inks, correction fluid and drain cleaners.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

1,1,1-Trichloroethane has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

1,1,1-Trichloroethane can be removed from drinking water by adsorption onto granular activated carbon, by aeration and by boiling. If aeration is used for removal, consideration should be given to effects associated with inhalation.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of 1,1,1-trichloroethane (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and 1,1,1-trichloroethane extracted using methyl tert-butyl ether. The extract is then analysed using gas chromatography with an electron capture detector. The limit of determination is approximately 0.000008 mg/L (8 ng/L).

HEALTH CONSIDERATIONS

1,1,1-Trichloroethane is absorbed rapidly and efficiently from the human gastrointestinal tract and the lungs. It is metabolised to a very limited extent (probably less than 6%) by both humans and animals.

An extensive review and summary of the human and animal toxicity data for 1,1,1-trichloroethane is available (IPCS 1992).

Inhalation of high concentrations of 1,1,1-trichloroethane has proved fatal, causing acute congestion of the lungs, fluid build-up and fatty deposits in the liver.

In animals, long-term studies have reported diminished body-weight gains at high doses (above 350 mg/kg body weight) but data were insufficient to determine no-effect levels. Liver tumours were observed in mice, but not in rats, fed 1,1,1-trichloroethane for 2 years; however, the study reported a high number of accidental deaths in both the control and study groups, and the results may not be significant.

Mutagenic activity has been reported in tests with some strains of bacteria, but not others.

The International Agency for Research on Cancer has concluded that 1,1,1-trichloroethane is not classifiable as to its carcinogenicity in humans (Group 3, no adequate data in humans, inadequate evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The World Health Organization health-based guideline value of 2 mg/L was based on a short-term inhalation study. The data were not considered to be sufficient to set an Australian guideline.

REFERENCE

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1992). 1,1,1-Trichloroethane. Environmental Health Criteria, 136. World Health Organization, IPCS.

USEPA Draft Method 551 (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (ESML), Cincinnati, Ohio.

Trichloroethylene (TCE)

(endorsed 1996)

GUIDELINE

Data are inadequate to set a guideline value for trichloroethylene in drinking water.

GENERAL DESCRIPTION

TCE may be present in drinking water as a result of direct contamination of water sources, or from atmospheric contamination of rainfall. In the United States, TCE has been detected in the water supplies of about 20% of cities tested, with mean concentrations of 0.02 mg/L or less.

TCE is used in cleaning fluids, as an industrial solvent and as a degreaser for metal components. The most significant route of exposure to humans is inhalation, particularly from use as a cleaning fluid.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

TCE has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

TCE can be removed from drinking water by aeration, or by adsorption onto granular activated carbon.

MEASUREMENT

A solvent extraction procedure is suitable for the analysis of TCE (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and TCE extracted using methyl tert-butyl ether. The extract is then analysed using gas chromatography with an electron capture detector. The limit of determination is approximately 0.000002 mg/L (2 ng/L).

HEALTH CONSIDERATIONS

TCE is readily absorbed by all routes of exposure and distributed to all tissues. It is metabolised to reactive epoxides and the trichloro derivatives of acetaldehyde, ethanol and acetic acid.

An extensive review and summary of the human and animal toxicity data for trichloroethylene is available (IPCS 1985).

In humans, TCE is a known central nervous system depressant and has been used as a general anaesthetic. Liver damage has been reported in people occupationally exposed to high concentrations.

There is some evidence that TCE induces liver and lung tumours in various strains of mice. In an inhalation study, TCE produced a dose-related increase in malignant lymphomas in female mice exposed to 100 ppm or above in air. TCE is a weakly acting mutagen in bacteria and yeast.

The International Agency for Research on Cancer has concluded that TCE is not classifiable as to its carcinogenicity in humans (Group 3, inadequate evidence in humans and limited evidence in animals) (IARC 1987).

DERIVATION OF GUIDELINE

The World Health Organization health-based guideline value of 0.07 mg/L was based on a 6-week feeding study using mice which identified a low-effect level but not a no-effect level. No long-term studies are available to establish a no-effect level. The data were not considered to be sufficient to set an Australian guideline.

REFERENCES

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

IPCS (International Programme on Chemical Safety) (1985). Trichloroethylene. Environmental Health Criteria, 50. World Health Organization, IPCS.

USEPA Draft Method 551 (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Triclopyr

(endorsed 2011)

GUIDELINE

Based on human health concerns, triclopyr in drinking water should not exceed 0.02 mg/L.

RELATED CHEMICALS

Triclopyr (CAS 55335-06-3) belongs to the pyridinecarboxylic acid class of chemicals. Other pesticides in this class include chlopyralid, fluroxypyr and picloram (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, triclopyr would not be a health concern unless the concentration exceeded 0.02 mg/L. Excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Triclopyr is a post-emergence herbicide for the control of woody and broad-leaf weeds in commercial and industrial areas, native pastures, right of ways, forests, fence lines and in the home garden.

There are registered products that contain triclopyr, its triethylamine salt or butoxyethyl ester in Australia. The products are intended for professional and/or home garden use. Products are available as a concentrated solution to be applied directly to weeds as a dilute spray by hand-held, mistblower, boom and aerial application methods. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main source of public exposure to triclopyr and its metabolites is the use of home garden products. A further possible source of exposure is residues in food from livestock grazing in treated areas. Residue levels in food produced according to good agricultural practice are generally low.

Commercial use of triclopyr may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

REPORTED VALUES IN AUSTRALIAN WATERS

No data on occurrence of triclopyr in Australian waters could be found. In the USA, the United States Environmental Protection Agency predicts that triclopyr will not reach high concentrations in groundwater, and concludes that it is not a concern in drinking water that is derived from groundwater. Triclopyr is also not expected to be present in significant concentrations in surface water due to its quick degradation (USEPA 1998).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No information could be found on the efficiency of drinking water treatment processes in removing triclopyr.

MEASUREMENT

Triclopyr can be measured by routine gas chromatography with mass spectrometry analysis, with a limit of reporting of 0.01 µg/L (Queensland Health 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for triclopyr is 0.005 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 0.5 mg/kg bw/day from a long-term dietary study in dogs. The NOEL is based on histopathological changes in the kidney. The ADI incorporates a safety factor of 100 and was established in 1986.

The previous *Australian Drinking Water Guidelines* health value was 0.01 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Triclopyr is readily and extensively absorbed via the gastrointestinal tract in animals and humans. It is not extensively metabolised, and the majority of the dose is excreted unchanged in the urine within 72 hours.

Acute effects: Triclopyr has moderate acute oral toxicity and low acute dermal toxicity. It is a skin sensitiser.

Short-term effects: Short-term dietary studies conducted in mice, rats and dogs reported the kidney to be the most sensitive target organ. Studies in mice and rats reported changes in relative kidney weights together with histopathological changes in the kidney at doses of 20 mg/kg bw/day and above. At 70 mg/kg bw/day in rats, there was an increase in relative liver weights and decreased bodyweight gain, as well as some evidence of necrosis of hepatocytes. In dogs, there was evidence of decreased renal function at doses of 2.5 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies conducted in mice, rats and dogs reported the liver and kidneys to be the main target organs. A 2-year study in mice reported decreased bodyweight gain, and clinical chemistry changes indicative of kidney and liver damage, together with histopathological effects in the bladder, liver and kidneys, at doses of 25.5 mg/kg bw/day and above.

A 2-year study in rats reported increased absolute and relative kidney weights and histopathological effects in the kidney at doses of 12 mg/kg bw/day and above.

A 1-year dog study reported evidence of decreased renal function together with histopathological changes in the kidney at doses of 2.5 mg/kg bw/day and above. The NOEL from this study was 0.5 mg/kg bw/day and this is the basis of the current ADI.

Carcinogenicity: Based on long-term studies in mice, rats and dogs, there is no evidence of carcinogenicity for triclopyr.

Genotoxicity: Triclopyr is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: Two and three-generation reproductive studies in rats and developmental studies in rats and rabbits did not produce any evidence of effects on reproductive parameters or foetal development, other than maternal toxicity at high dose levels that were well in excess of the likely level of human exposure.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Triclopyr is included in Schedule 6 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.02 mg/L for triclopyr was determined as follows:

$$0.02 \text{ mg/L} = \frac{0.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 0.5 mg/kg bw/day is the NOEL based on a long-term (1-year) study in dogs.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Queensland Health (2007). Organochlorine, organophosphorous and synthetic pyrethroid pesticide, urea and triazine herbicides and PCBs in water. QHFSS SOP 16315.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1998). Reregistration eligibility decision (RED) for triclopyr. EPA 738-R-98-011. USEPA.

Trifluralin

(endorsed 2011)

GUIDELINE

Based on human health concerns, trifluralin in drinking water should not exceed 0.09 mg/L.

RELATED CHEMICALS

Trifluralin (trifluraline)(CAS 1582-09-8) belongs to the dinitroaniline class of chemicals. Other pesticides in this class include oryzalin and pendimethalin (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, trifluralin would not be a health concern unless the concentration exceeded 0.09 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Trifluralin is a pre-emergent herbicide for the control of ryegrass, wireweed, and yellow burr weeds in cereal, legume, herb, and vegetable crops, and for the control of garden weeds in domestic settings.

There are registered products containing trifluralin in Australia. The products are intended for professional and home garden use. Use patterns include application to soil by ground boom spray in agricultural settings and by hand-held spray for home gardens. Data on currently registered products are available from the Australian Pesticides and Veterinary Medicines Authority.

Exposure sources: The main sources of public exposure to trifluralin and its metabolites are the use of home garden weedkiller products, and residues in food. Residue levels in food produced according to good agricultural practice are generally low.

Agricultural use of trifluralin may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Trifluralin has low solubility in water and generally is not detected in surface water. It is unlikely to leach into groundwater supplies, given its low solubility and strong soil adsorption (Health Canada 1989). Trifluralin has been detected very occasionally in drinking water supplies in Australia at concentrations well below the health value. It was reported above 1 µg/L in 0.2% of the samples taken in the Murray-Darling Basin (NSW 1998/99 data) (NSW Department of Pirmary Industries 2005).

In the USA, trifluralin was found in 172 of 2047 surface water samples and in 1 of 507 groundwater samples analysed, but was not found in 229 drinking-water supplies analysed in Italy (WHO 1996). It was detected at low concentrations (ng/L) in raw drinking water samples and in 1 of 91 groundwater samples (41 µg/L) in Canada (Health Canada 1989).

TREATMENT OF DRINKING WATER

Trifluralin is efficiently removed (100% effectiveness) by conventional treatment using alum, sedimentation and filtration. In addition, it may be removed from drinking water by reverse osmosis, granular activated carbon and air stripping (Health Canada 1989, Ormad *et al.* 2008).

MEASUREMENT

Trifluralin can be analysed in water by solvent extraction or by solid phase extraction. Quantification is performed by gas chromatography with electron capture detection (GC-ECD); gas chromatography with mass spectrometry (GC-MS) or liquid chromatography (LC) with ultraviolet detection. The limit of detection (LOD) is 0.01µg/L (Van Hoof *et al.* 2001, Carabias-Martinez *et al.* 2003). Direct injection liquid chromatography–mass spectrometry or online solid phase microextraction liquid chromatography with mass spectrometry can achieve a lower LOD. The GC-ECD-GCMS United States Environmental Protection Agency method 3510/8080 can achieve a LOD of 0.1 µg/L (Queensland Government 2007).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for trifluralin is 0.02 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 2.5 mg/kg bw/day from a 2-generation study in rats. The NOEL is based on decreased foetal and parental weights. The ADI incorporates a safety factor of 100, and was established in 1991.

The previous *Australian Drinking Water Guidelines* health value was 0.05 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Trifluralin is poorly absorbed via the gastrointestinal tract and the majority is excreted unchanged in the faeces. Absorbed trifluralin is eliminated as metabolites in urine within three days. The primary metabolites are dealkylated and hydrogenated compounds.

Acute effects: Trifluralin has low acute oral and dermal toxicity. It has some potential for skin sensitisation.

Short-term effects: In a 3-month dietary study in rats born from mothers pre-treated with oral doses of trifluralin, bodyweights were decreased and relative liver weights were increased at 100 mg/kg bw/day. In a 4-month dietary study in rats, effects on the kidney were seen at the lowest dose, 20 mg/kg bw/day and above.

Long-term effects: Long-term dietary studies in rats reported liver weight changes and tissue irritation in the kidney at 33.5 mg/kg bw/day.

Carcinogenicity: Kidney tumours were noted in rats at high dose levels as a result of persistent tissue irritation. This effect was not considered relevant to humans at the normal low levels of exposure.

Genotoxicity: Trifluralin is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: In three reproduction studies in rats (2-3 generations), there was reduced fertility and reduced neonatal viability at dose levels above 2.5 mg/kg bw/day. In developmental studies in rats and rabbits, there was no evidence of effects on foetal development.

NOTE: Important general information is contained in PART II, Chapter 6

The NOEL of 2.5 mg/kg bw/day is the basis for the ADI.

Poisons Schedule: Trifluralin is considered not to require control by scheduling due to its low toxicity and is therefore in Appendix B of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.09 mg/L for trifluralin was determined as follows:

$$0.09 \text{ mg/L} = \frac{2.5 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 2.5 mg/kg bw/day is the NOEL based on a 2-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the conservative assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

The World Health Organization has a Guideline value of 0.02 mg/L for trifluralin (WHO 2004).

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Carabias-Martinez R, Garcia-Hermida C, Rodriguez-Gonzalo E, Soriano-Bravo FE, Hernandez-Mendez J (2003). Determination of herbicides, including thermally labile phenylureas, by solid-phase microextraction and gas chromatography-mass spectrometry. *Journal of Chromatography A,* 1002(1-2):1-12.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Health Canada (1989) Guidelines for Canadian Drinking Water Quality – Technical Documents: Trifluralin. [http://www.hc-sc.gc.ca/ewh-semt/pubs/water-eau/trifluralin/index-eng.php]

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

NSW Department of Pirmary Industries (2005). Impacts of urban and rural development. [http://www.dpi.nsw.gov.au/fisheries/habitat/threats/urban]

Ormad MP, Miguel N, Claver A, Matesanz JM, Ovelleiro JL (2008). Pesticides removal in the process of drinking water production. *Chemosphere,* 71(1): 97-106.

Queensland Government (2007). Water Monitoring Data Collection Standards, WMO010, Natural Resources and Water.

NOTE: Important general information is contained in PART II, Chapter 6

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th Edition, British Crop Production Council, UK.

Van Hoof F, Van Wiele P, Bruchet A, Schmitz I, Bobeldiji I, Sacher F, Ventura F, Marti I, Morecos Do Monte MH, Sa Da Costa M (2001). Multiresidue determination of pesticides in drinking and related waters by gas chromatography/mass spectrometry after solid-phase extraction: interlaboratory study. *Journal of AOAC International,* 84(5):1420-9.

WHO (World Health Organization) (1996). Trifluralin in Drinking-water. WHO, Geneva, Switzerland.

WHO (World Health Organization) (2004). *Guidelines for Drinking-water Quality*. 3rd Edition, WHO, Geneva, Switzerland.

NOTE: Important general information is contained in PART II, Chapter 6

Trihalomethanes (THMs)
trichloromethane (chloroform)
bromodichloromethane
dibromochloromethane
tribromomethane (bromoform)

(endorsed 1996)

GUIDELINE

Based on health considerations, the concentration of trihalomethanes, either individually or in total, in drinking water should not exceed 0.25 mg/L.

Trihalomethane concentrations fluctuating occasionally (for a day or two annually) up to 1 mg/L are unlikely to pose a significant health risk.

Action to reduce THMs is encouraged, but must not compromise disinfection, as nondisinfected water poses significantly greater risk than THMs.

GENERAL DESCRIPTION

In Australia, trihalomethanes are present in drinking water principally as the result of disinfection using chlorination or, to a much lesser extent, chloramination. Chlorine, which produces hypochlorous acid when added to water, can react with naturally occurring organic material, such as humic and fulvic acids, to produce trihalomethanes. The brominated trihalomethanes are produced by the oxidation of bromide present in water to form hypobromous acid, which can then react with organic matter in a similar way.

High trihalomethane concentrations may indicate the presence of other chlorination by-products.

Chloroform is produced commercially and is an important solvent. It is used in the manufacture of refrigerants, and as an ingredient in pharmaceutical and cosmetic preparations. Brominated trihalomethanes are also produced industrially, but less commonly than chloroform.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies concentrations of total trihalomethanes range up to 0.6 mg/L.

LIMITING PRESENCE IN DRINKING WATER

The concentration of trihalomethanes can be minimised by removing precursors, by removing trihalomethanes after formation, or by using alternative disinfectants. Precursors can be removed by activated carbon, by coagulation followed with filtration, or by oxidation with ozone or potassium permanganate. Once produced, trihalomethanes can be removed with air stripping or adsorption onto granular activated carbon. Alternative disinfection agents to chlorine, such as chloramines, ozone and chlorine dioxide, can substantially reduce trihalomethane concentrations, but may produce other by-products.

MEASUREMENT

There are a number of methods available for the analysis of trihalomethanes, including head-space analysis, solvent extraction, purge and trap, and direct collection on resins. The solvent extraction procedure is relatively simple to use (USEPA Draft Method 551 1990). Sodium chloride is added to the sample and the trihalomethanes extracted using methyl tert-butyl ether. The extracts are then analysed

using gas chromatography with electron capture detection. Limits of determination are 0.00002 mg/L (20 ng/L) or less.

HEALTH CONSIDERATIONS

The trihalomethanes are rapidly and efficiently absorbed following ingestion. They are metabolised primarily to carbon dioxide and/or carbon monoxide, and rapidly exhaled. They are fat soluble, and accumulate in tissues with the highest lipid content (such as adipose tissue, brain, kidney and blood).

In animals, the trihalomethanes are central nervous system depressants and liver and kidney toxicants. Chloroform and bromoform are also known to cause central nervous system depression in humans.

Some epidemiological studies have reported associations between the ingestion of chlorinated drinking water (which typically contains THMs) and increased cancer mortality rates. The International Agency for Research on Cancer has concluded that the available data for chlorinated water provide inadequate evidence of carcinogenicity in humans (Group 3, inadequate evidence in humans and limited evidence in animals) (IARC 1991).

Long-term carcinogenicity bioassays with animals have shown that trihalomethanes can produce tumours in rats and mice, but only at doses that are toxic to the animals. Chloroform increased the incidence of liver tumours in mice when administered in food at doses from 25 mg/kg body weight per day, but not in drinking water at the same doses, and has induced kidney tumours in male rats at doses from 263 mg/kg body weight per day. Dibromochloromethane, given by gavage 5 days per week, clearly induced liver tumours in female mice at 100 mg/kg body weight per day, and possibly in male mice, but not in rats. Bromodichloromethane, given by gavage 5 days per week, induced kidney tumours in rats at 100 mg/kg body weight, and in male mice at 50 mg/kg body weight; a rare tumour of the large intestine in male rats at doses of 50 and 100 mg/kg body weight; and liver tumours in female mice at 75 and 150 mg/kg body weight. Bromoform induced a small increase in relatively rare tumours of the large intestine in rats at a gavage dose of 200 mg/kg body weight per day, but not in mice.

Results of studies on the genotoxicity of trihalomethanes in bacteria have been inconsistent, with most reporting negative results. Trihalomethanes have, however, induced chromosomal aberrations in human lymphocyte cells *in vitro*, and in mouse bone-marrow cells *in vivo*.

Available studies indicate that THMs can produce maternal and foetal toxicity at high doses, but not teratogenicity.

The International Agency for Research on Cancer has concluded that chloroform and bromodichloromethane are possibly carcinogenic to humans (Group 2B, inadequate evidence in humans but sufficient evidence in animals); and that bromoform and dibromochloromethane are not classifiable as to their carcinogenicity to humans (Group 3, inadequate evidence in humans and limited evidence in animals)(IARC 1991).

DERIVATION OF GUIDELINE

The guideline value for trihalomethanes in drinking water was determined as follows:

$$0.25 \text{ mg/L} = \frac{7 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 7 mg/kg body weight per day is the no-effect level based on a 90-day study using rats (Chu *et al.* 1982). The use of this value was recommended by the NHMRC Standing Committee on Toxicity following a review of the available toxicity data for THMs.
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water. A higher value was not used because exposure to chloroform from other sources may be significant.
- 2 L/day is the average amount of water consumed by an adult.
- 100 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations and 10 for intraspecies variations). An additional safety factor for the less than lifetime study was not applied as long-term studies have reported the same effects, and no other forms of toxicity were observed. In addition, the changes observed to the liver at higher doses were mild in nature and disappeared when exposure stopped. The use of this safety factor was recommended by the NHMRC Standing Committee on Toxicity.

Separate guideline values were not derived for each compound as the no-effect levels were similar (ranging from 6.5 to 7.8 mg/kg body weight per day), and the compounds are metabolised in the body in similar ways. The guideline value should therefore apply to the concentration of each compound, or the sum of any combination of individual THM concentrations.

The World Health Organization (WHO) has derived separate guideline values for each compound, but in doing so recognises that the compounds have similar toxicological action.

The WHO guideline values for chloroform (0.2 mg/L) and bromodichloromethane (0.06 mg/L) were based on calculations that estimated additional lifetime risks of one fatal cancer per 100,000 people. The use of this approach is questionable because there is evidence that tumours do not occur at low concentrations.

The WHO guideline values for bromoform (0.1 mg/L) and dibromochloromethane (0.1 mg/L) were based on different studies and safety factors from those recommended by the NHMRC Standing Committee on Toxicity, although toxicological effects were similar.

It is recommended that future reviews of the guidelines consider the various THMs individually, as data are emerging that suggest the different THMs have different toxic effects. Data were not sufficient at the time of this review to justify individual assessments

In view of the safety factors used in the derivation of the guideline value, it is unlikely that short-term consumption of water containing significantly higher concentrations of trihalomethanes would pose a health risk.

REFERENCES

Chu I, Villeneuve DC, Secours VE, Becking GC, Valli VE (1982). Trihalomethanes: II Reversibility of toxicological changes produced by chloroform, bromodichloro-methane, chlorodibromomethane and bromoform in rats. *Journal of Environmental Science and Health*, B17:225–240.

IARC (International Agency for Research on Cancer) (1991). IARC monographs on the evaluation of carcinogenic risks to humans: Chlorinated drinking water; chlorination by-products; some other halogenated compounds; cobalt and cobalt compounds. World Health Organization, IARC, 52, Lyons.

USEPA Draft Method 551 (1990). Determination of chlorination disinfection by-products and chlorinated solvents in drinking water by liquid–liquid extraction and gas chromatography with electron capture detection. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

NOTE: Important general information is contained in PART II, Chapter 6

Turbidity

(endorsed 2011)

GUIDELINE

Chlorine-resistant pathogen reduction: Where filtration alone is used as the water treatment process to address identified risks from Cryptosporidium and Giardia, it is essential that filtration is optimised and consequently the target for the turbidity of water leaving individual filters should be less than 0.2 NTU, and should not exceed 0.5 NTU at any time

Disinfection: A turbidity of less than 1 NTU is desirable at the time of disinfection with chlorine unless a higher value can be validated in a specific context.

Aesthetic: Based on aesthetic considerations, the turbidity should not exceed 5 NTU at the consumer's tap.

GENERAL DESCRIPTION

Turbidity is a measure of the light-scattering property of water caused by the presence of fine suspended matter such as clay, silt, plankton and other microscopic organisms. The degree of scattering depends on the amount, size and composition of the suspended matter. At low levels, turbidity can only be detected by instruments, but at higher levels the water has a "muddy" or "milky" appearance clearly visible to the naked eye. As a guide, water with a turbidity of 5 Nephelometric Turbidity Units (NTU) appears slightly muddy or milky in a glass, while at >60 NTU, it is not possible to see through the water. "Crystal-clear" water usually has a turbidity of less than 1 NTU.

There are three distinct aspects to turbidity to be considered within the catchment-to-consumer risk management framework:

- the use of turbidity as a measure to provide assurance of the optimal operation of filter performance, where filtration is used to address identified risks associated with chlorine-resistant pathogens in the source water;
- the impact of turbidity on the efficiency of disinfection processes;
- the effect that turbidity has on the aesthetics of the treated water.

MEASUREMENT

For laboratory-based analyses, the ration-recording nephelometroc turbidity meter is the preferred method for turbidity meanreument, as it can compensate for the effect of dissolved colour. Results are expressed in NTU and are calibrated against a prepared formazin standard (APHA 2130B, 2005). The detection limit is about 0.1 NTU.

When using turbidity for accurate monitoring of filter performance (i.e. where filtration is the only water treatment process to remove chlorine-resistant pathogens), it is recommended that on-line, continuously reading turbidity meters be installed on the outlet of each individual filter in addition to any on-line turbidity meter that is installed on the combined filter outlet. It is prudent to have the turbidity meter outputs linked into plant SCADA and/or alarm systems, to ensure that immediate action is taken in response to the detection of filtered water turbidity above the set target. This intensity or operational monitoring is strongly recommended to ensure that any performance issues related to individual filters are detected and addressed proactively (USEPA 2004, Mosse 2009). Particle counting facilities are used for the same purpose of filter optimisation but the results are too dependent on the actual equipment used and their mode of operation to provide general guidance in the same context as for tubidity.

While real-time monitoring of the turbidity trends generated from the on-line instruments is crucial in determining the instantaneous performance of the plant, and therefore the safety of the water, longer-term monitoring is beneficial to demonstrate the need for continuous improvement and maintenance activities such as filter inspections, optimised backwash and other process procedures.

TREATMENT OF DRINKING WATER

Pathogen reduction

Chlorine-based disinfection is only effective against bacterial and most viral, pathogens. At the doses typically applied in water treatment, chlorine is not effective against the protozoan pathogen *Cryptosporidium* and only has a limited effect on *Giardia* in the absence of large filtered water storages to provide adequate contact time for effective disinfection. *Cryptosporidium* oocysts are quite small (4-6 µm) and will pass readily through a conventional media filter in the absence of effective coagulation and flocculation. Filtration combined with effective coagulation, flocculation and clarification can be used as a barrier for *Cryptosporidium* and other protozoan pathogens. In many cases, coagulation-assisted clarification and filtration may be the only existing treatment barrier to protozoan pathogens.

In the absence of reliable real-time pathogen detection methodologies, continuous turbidity monitoring is considered the best available surrogate for assessing filter performance.

Many studies have investigated the relationship between pre-treatment turbidity, turbidity reduction (or particle removal) via filtration, and pathogen reduction. It has been demonstrated in pilot scale trials that a change in filter effluent turbidity from 1.0 through 0.5 to 0.3 NTU would not significantly improve the reliability of pathogen control. However, by setting filter effluent turbidity goals below 0.2 NTU, significant improvements in microbial quality could be obtained (Xagoraraki *et al.* 2004). The USEPA identified that turbidity limits of 0.15 NTU from individual filters with an upper limit of 0.3 NTU provided a substantial improvement in removal of *Cryptosporidium* compared to its previous limits of 0.3 NTU, with an upper limit of 1 NTU (USEPA 2006).

Targets for filtered water turbidity should be based on the pathogen risks in the raw water; for example, surface run-off from a catchment with significant sewage inputs or dairy farms would have tighter turbidity targets than a catchment without such impacts. Therefore, when setting turbidity targets for filtered water, raw water quality and treatment capabilities need to be aligned to manage any potential health risks. The United States Environmental Protection *Agency Long Term 2 Enhanced Surface Water Treatment Rule* (USEPA 2006) and the *Drinking-water Standards for New Zealand* (NZ-MOH 2008) directly relate raw water quality to the setting of filtered water turbidity targets.

Where a given water supply system risk assessment identifies a significant risk associated with protozoan pathogens, and a high level of operational monitoring of turbidity and any associated adjustment or maintenance of coagulation, flocculation, clarification and filtration processes or facilities are not considered practical, then alternative processes (e.g. ultraviolet radiation disinfection) may need to be applied to ensure the identified risk is adequately addressed.

Catchment management and source protection can be good enough to obviate the need for water treatment to remove and/or inactive protozoan pathogens. Exclusion of contamination from humans and domesticated animals in run-off from catchments and source areas generally leads to only minimal risk from protozoan pathogens in the Australian context, and specific treatment to remove protozoa is not required. In many cases, however, catchments and sources are not sufficiently managed and protected to ensure safe drinking water without additional treatment.

NOTE: Important general information is contained in PART II, Chapter 6

Where water is harvested from partly protected catchments and sources with a relatively low level of contamination, protozoan pathogens can be removed adequately by conventional treatment alone. Conventional treatment involves the addition of coagulants, removal of solids using clarifiers such as sedimentation, solids contact or dissolved air floatation, and removal of the remaining solids in clarified water in media filters, followed by chlorine-based disinfection. Such treatment is widely used and technically capable of reducing turbidity to below 0.2 NTU but requires close operator attention and continuous monitoring as discussed above.

Where water is harvested from sources with significant risks of contamination with protozoan pathogens, filtration to 0.2 NTU alone may not reduce the risk from protozoan pathogens to acceptable levels. Other treatment, such as membrane filtration, or disinfection by ultraviolet radiation or ozonation, may be needed.

In most cases, the turbidity of the filtered water during ripening periods after filter backwash, may exceed 0.3 NTU. It is considered best practice to limit these short spikes in turbidity to no longer than 15 minutes. Spikes above 0.3 NTU represent periods of increased risk, and appropriate risk management practices should be employed, such as rejecting ripening water to waste or optimising filter backwash processes.

Turbidity added after treatment can arise from the use of lime to raise the final pH of the water. This turbidity is unlikely to have an associated pathogen risk.

Disinfection

High turbidity has been shown to shield microorganisms from the action of disinfectants (Katz 1986). Low turbidity, however, is no guarantee that water is free from pathogenic microorganisms.

If the turbidity in a water supply exceeds 1 NTU, adequate disinfection may be more difficult to maintain, but may nevertheless be achievable.

Where water that is to be disinfected has not been previously filtered, it is desirable that the turbidity be less than 1 NTU at the time of disinfection, subject to the type of disinfectant being used. For example, disinfection using ultraviolet light is likely to remain effective at turbidities above 1 NTU, providing transmission is maintained, whereas the effectiveness of chlorine-based disinfectant can be affected above 1 NTU.

If water of a higher turbidity is to be disinfected, then validation work should be undertaken to demonstrate that disinfection of water under such conditions is effective.

Disinfection is discussed in more detail in Information Sheet 1 Disinfection of drinking water.

Aesthetics

Turbidity has an impact on the aesthetic acceptability of water. Many consumers relate the appearance of water to its safety, and turbid or coloured water is interpreted as being unsafe to drink. Turbidity must therefore be maintained as low as possible to the point of supply to customers.

Passage of water through a distribution system can also lead to an increase in turbidity, generally as a result of the resuspension of fine sediments settled over a long period of time, or from the breakdown of pipe materials or biofilms lining the walls of the pipes. While the associated health risk is generally minimal, it may be significant in poorly maintained systems, as some biofilms are known to harbour living microorganisms. Therefore turbidity in the distribution system can be also used as an indicator of good distribution management practices.

NOTE: Important general information is contained in PART II, Chapter 6

HEALTH CONSIDERATIONS

Consumption of highly turbid waters is not necessarily a health hazard, but may constitute a health risk if the suspended particles harbour pathogenic microorganisms capable of causing disease in humans, or if the particles have adsorbed toxic organic or inorganic compounds.

For a treatment system designed for chlorine-resistant pathogen reduction via filtration only, detection of increases in the turbidity of filtered water above 0.5 NTU should trigger investigative action. Major filtration failures should referred to the relevant health authority or regulator to assess the potential health risk.

Turbidity can have a significant impact on the microbiological quality of drinking water. High turbidity interferes with both the detection and the disinfection of pathogens, by adsorbing them into the particulate matter and thus shielding them. Some turbidity may also promote bacterial growth if they provide a source of nutrients.

It is important to recognise the sources of suspended or particulate matter in water, and the potential associated risks to human health. Particulate matter from multi-use surface catchments often contains human pathogens. The poor management of turbid water events is a significant factor in many waterborne disease outbreaks (Hrudey and Hrudey 2004).

REFERENCES

APHA Method 2130B (2005). Turbidity: nephelometric method. *Standard Methods for the Examination of Water and Wastewater*, 21st edition. American Public Health Association, Washington.

Hrudey SE, Hrudey EJ (2004). *Safe Drinking Water – Lessons from recent outbreaks in affluent countries.* IWA Publishing, London.

Katz EL (1986). The stability of turbidity in raw water and its relationship to chlorine demand. *Journal of theAmerican Water Works Association*, 78:72–75.

Mosse P (2009). Longterm filter monitoring and safe drinking water – data from two Australian water utilities. *Water Works – Officical Journal of the Water Industry Operators Association*, June 2009, Water Industry Operators Association, Shepparton, p8-11.

NZ-MOH (New Zealand Ministry of Health) (2008). *Drinking-water Standards for New Zealand* 92005, revised 2008)

USEPA (2004). *Long Term 1 Enhanced Surface Water Treatment Rule – Turbidity Provisions – Technical Guidance Manual.*

USEPA. 2006. *National Primary Drinking Water Regulations: Long Term 2 Enhanced Surface Water Treatment Rule: Final Rule.*

Drinking-water Standards for New Zealand 2005 (revised 2008) (2008). Ministry of Health.

Xagoraraki I, Harrington GW, Assavasilavasukul P, Standridge JH (2004). Removal of emerging pathogens and pathogen indicators. *AWWA Journ*al, 96(5):102-113

NOTE: Important general information is contained in PART II, Chapter 6

Uranium

(endorsed 2011)

GUIDELINE

Based on health considerations, the concentration of uranium in drinking water should not exceed 0.017 mg/L.

GENERAL DESCRIPTION

Uranium may be present in the environment as a result of leaching from soils, rocks and natural deposits, release in mill tailings, combustion of coal and other fuels, and use of phosphate fertilisers (which can contain as much as 150 mg/kg uranium). Naturally occurring uranium is a mixture of three radionuclides, U-238, U-234, and U-235. U-238 and U-234 decay predominantly by alpha particle emission, whereas U-235 emits both gamma rays and alpha particles. Natural uranium consists almost entirely of the U-238 isotope, the other isotopes being less than 1% abundant. Uranium is used primarily as a fuel in nuclear power plants.

Studies overseas have reported uranium concentrations in drinking water of generally less than 0.001 mg/L; however, concentrations as high as 0.7 mg/L have been reported in some private water supplies in Canada.

Food is the major source of uranium intake and highest concentrations are found in shellfish. Dietary intake of uranium through food is estimated between 0.001 and 0.004 mg/day (WHO 2004). Intake through drinking water is normally low; however, drinking water can contribute the majority of daily intake in circumstances in which uranium is present at higher concentrations (WHO 2004).

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No data are available on concentrations of uranium in Australian drinking water.

TREATMENT OF DRINKING WATER

Conventional treatment processes are not effective in removing uranium from water supplies. Some laboratory or pilot scale studies have found that coagulation using ferric sulfate at optimal pH dosages can achieve 80–95% removal of uranium, whereas at least 99% removal can be achieved using lime softening, anion exchange resin or reverse osmosis processes (WHO 2004).

MEASUREMENT

The concentration of uranium in water can be determined using solid fluorimetry with laser excitation (Blanchard *et al*. 1985), or inductively coupled plasma mass spectrometry (Boomer *et al*. 1987). The limit of determination is about 0.0001 mg/L.

The isotopes of uranium can be determined by radiochemical techniques using high resolution alpha spectrometry to measure their activity (EML 1990, USEPA 1980). The limit of determination is about 0.005 Bq/L (equivalent to approximately 0.0004 mg/L uranium).

HEALTH CONSIDERATIONS

The toxicity of uranium has been reviewed by the World Health Organization (WHO 2004), the Swedish National Food Administration (Svensson *et al.* 2005), the United Kingdom Committee on Toxicity (COT 2006), and Health Canada (2001).

Average absorption of dietary uranium by the gastrointestinal tract is 1-2%, but may be as low as 0.1% or as high as 5-6% depending on the solubility of the uranium compound ingested. Uranium rapidly appears in the bloodstream and is primarily associated with red blood cells. Uranyl compounds readily combine with proteins and nucleotides to form stable complexes. Clearance of uranium from the blood is rapid but it accumulates in the kidney and bone, with little in the liver. Once equilibrium in the skeleton has occurred, uranium is excreted in the urine and faeces. The half life of uranium in rat and rabbit kidney is of the order of 5–15 days, but in bone it is 100–300 days (Health Canada 2001).

In humans and experimental animals, the main toxic effect of short-term exposure to high concentrations of uranium is inflammation of the kidney. Little information is available on the effects of long-term exposure to low concentrations. Epidemiological studies report increases in urinary markers of possible kidney proximal tubule damage at drinking water concentrations between 0.1 and 1 mg/L, but not at lower concentrations (Moss *et al.* 1983, Mao *et al.* 1995, Zamora *et al.* 1998, Kurttio *et al.* 2002, Kurttio *et al.* 2006, Seldén *et al.* 2009, Magdo *et al.* 2007).

A tolerable daily intake (TDI) of 0.0006 mg per kg bodyweight (µg/kg bw) has been derived by WHO (2004) and Health Canada (2001). This is based on a lowest-observed-adverse-effect level (LOAEL) of between 0.06 (males) and 0.09 (females) mg/kg bw/day in a 91-day rat drinking water study (Gilman *et al.* 1998) and application of an uncertainty factor of 100 (10 for interspecies extrapolation and 10 for intraspecies variation). The critical effect was degenerative kidney lesions, noted by the authors as not being severe. Although these represented a clear adverse effect, they were not dose-related, and in addition, because the effects were minimal, it is considered the dose at which they occurred may be close to the no-observed-adverse-effect level (NOAEL) (WHO 2004, COT 2006, Health Canada 2001). Thus an uncertainty factor to extrapolate from a LOAEL to a NOAEL was not applied in the derivation of the TDI.

No data are available on chemically induced mutagenic effects in relation to uranium.

Studies have shown high specific activity uranium isotopes to be carcinogenic in animals, causing malignant tumours in mice and bone sarcomas in rats. Similar studies using natural uranium (uranium-238) have not shown similar effects, possibly due to the lower radiation doses involved. Epidemiological data are inadequate to show whether exposure to uranium in drinking water will lead to an increased risk of cancer.

DERIVATION OF GUIDELINE

i) From chemical toxicity data:

The guideline value for uranium in drinking water of 0.017 mg/L was set from chemical toxicity data as follows:

$$0.017 \text{ mg/L} = \frac{0.0006 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.8}{2 \text{ L/day}}$$

NOTE: Important general information is contained in PART II, Chapter 6

where:

- 0.0006 mg/kg bodyweight/day is the TDI (WHO 2004, Health Canada 2001).
- 70 kg is taken as the average weight of an adult.
- 0.8 is a proportionality factor based on a conservative assumption that 80% of total daily intake may be attributable to the consumption of water (WHO 2004).
- 2 L/day is the estimated maximum amount of water consumed by an adult.

ii) From radiological data:

$$3.0 \text{ Bq/L} = \frac{0.1 \text{ mSv /year}}{730 \text{ L/year} \times 4.5 \times 10^{-5} \text{ mSv/Bq}}$$

where:

- 0.1 mSv/year is the committed effective dose limit for an individual radionuclide. This is set at approximately a twentieth of the average background radiation dose from all sources (UNSCEAR 2000)
- 730 L/year is the estimated maximum amount of water consumed by an adult (2 L/day x 365 days).
- 4.5×10^{-5} mSv/Bq is the committed effective dose received per unit intake of uranium-238 activity (Bq) (ICRP 1996).

iii) Comparing the chemical and radiological data:

A ^{238}U activity concentration of 3.0 Bq/L is equivalent to a chemical concentration of natural uranium of 0.24 mg/L. This is considerably greater than the guideline of 0.015 mg/L derived from the chemical toxicity data. The guideline value derived from chemical toxicity data is therefore also protective of radiological effects.

REFERENCES

Blanchard RL, Hahne RMA, Kahn B, McCurdy D, Mellor RA, Moore WS, Sedlet J, Whittaker E (1985). Radiological sampling and analytical methods for national primary drinking water regulations. *Health Physics*, 48:587–600.

Boomer DW, Powell MJ (1987). Determination of uranium in environmental samples using inductively coupled plasma mass spectrometry. *Analytical Chemistry*, 59:2810–2813.

COT (UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment) (2006). COT Statement on uranium levels in water used to reconstitute infant formula. COT statement 2006/07, May 2006.

EML (1990). EML Procedures Manual, 27th edition. Environmental Measurements Laboratory, Department of Energy, New York, United States.

Gilman AP, Villeuve DC, Secours VE, Yagminas AP, Tracy BL, Quinn JM, Valli VE, Willes RJ, Moss MA (1998). Uranyl Nitrate: 28-Day and 91-Day Toxicity Studies in the Sprague-Dawley Rat. *Toxicological Sciences*, 41(1):117-128.

Health Canada (2001). Guidelines for Canadian Drinking Water Quality: Supporting Documantation. Uranium.

ICRP (International Commission on Radiation Protection) (1996). *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 5 Compilation of Ingestion and Inhalation Dose Coefficients*. ICRP Publication 72, Pergamon Press, Oxford.

NOTE: Important general information is contained in PART II, Chapter 6

Kurrtio P, Harmoinen A, Saha H, Salonen L, Karpas Z, Komulainen H, Auvinen A (2006). Kidney toxicity of ingested uranium from drinking water. *American Journal of Kidney Diseases,* 47(6):972-982.

Kurttio P, Auvinen A, Salonen L, Saha H, Pekkanen J, Makelainen I, Vaisanen SB, Penttila IM, Komulainen H (2002). Renal effects of uranium in drinking water. *Environmental Health Perspectives,* 110(4):337-342.

Magdo HS, Forman J, Graber N, Newman B, Klein K, Satlin L, Amler RW, Winston JA, Landrigan PJ (2007). Grand rounds: nephrotoxicity in a young child exposed to uranium from contaminated well water. *Environmental Health Perspectives,* 115: 1237-1241.

Mao Y, Desmeules M, Schaubel D, Berube D, Dyck R, Brule D, Thomas B (1995). Inorganic components of drinking water and microalbuminuria. *Environmental Research,* 71(2):135-140.

Moss MA, McCurdy RF, Dooley KC, Givner ML, Dymond LC, Slayter JM, Courneya MM (1983). Uranium in drinking water – report on clinical studies in Nova Scotia. In: Brown SS, Savory J (eds), *Chemical Toxicology and Clinical Chemistry of Metals*, Academic Press, London, pp 149-152.

Seldén AI, Lundholm C, Edlund B, Högdahl C, Ek B-M, Bergström BE, Ohlson C-G. Nephrotoxicity of uranium in drinking water from private drilled wells. *Environmental Research*. In press, corrected proof.

Svensson K, Darnerud PO, Skerfving S (2005). A risk assessment of uranium in drinking water, National Food Administration of Sweden, Sweden.

UNSCEAR (United Nations Scientific Committee on the Effects of Atomic Radiation) (2000). 2000 Report Vol. I. Sources, effects and risks of ionising radiation. Report to the General Assembly, with Scientific Annexes.

USEPA United States Environmental Protection Agency (1980). Prescribed procedures for measurement of radioactivity in drinking water. USEPA, Environmental Monitoring and Support Laboratory, EPA-600/4-80-032, Cincinnati, United States.

WHO (World Health Organization) (2004). Uranium in drinking-water: Background document for development of WHO Guidelines for Drinking-water Quality. WHO.

Zamora ML, Tracy BL, Zielinski JM, Meyerhof DP, Moss MA (1998). Chronic ingestion of uranium in drinking water: A study of kidney bioeffects in humans. *Toxicological Sciences*, 43(1):68-77.

NOTE: Important general information is contained in PART II, Chapter 6

Vernolate

(endorsed 2011)

GUIDELINE

Based on human health concerns, vernolate in drinking water should not exceed 0.04 mg/L.

RELATED CHEMICALS

Vernolate (CAS 1929-77-7) belongs to the thiocarbamate class of chemicals. Other pesticides in this class include EPTC, molinate, and pebulate (Tomlin 2006).

HUMAN RISK STATEMENT

With good water quality management practices, the exposure of the general population is expected to be well below levels that may cause health concerns.

If present in drinking water as a result of a spillage or through misuse, vernolate would not be a health concern unless the concentration exceeded 0.04 mg/L. Minor excursions above this level would need to occur over a significant period to be a health concern, as the health-based guideline is based on medium- to long-term effects.

With good water quality management practices, pesticides should not be detected in source waters used for drinking water supplies. Persistent detection of pesticides may indicate inappropriate use or accidental spillage, and investigation is required in line with established procedures in the risk management plan for the particular water source.

GENERAL DESCRIPTION

Uses: Vernolate is a selective herbicide for the control of grasses and broad-leaf weeds in food-producing agricultural crops including soybeans, peanuts and potatoes.

There are currently no registered products that contain vernolate in Australia, but de-registered compounds may still be detected in water. Previously, products containing vernolate were intended for professional use and were available as concentrated solutions to be applied directly to soils in diluted form using ground, aerial or hand-held sprays.

Exposure sources: If used in the future, the main source of public exposure to vernolate and its metabolites would be residues in food. Residue levels in food produced according to good agricultural practice are generally be low.

Agricultural use of any vernolate in the future may potentially lead to contamination of source waters through processes such as run-off, spray drift or entry into groundwater.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

No published reports on vernolate occurrence in Australian drinking water supplies were found.

TREATMENT OF DRINKING WATER

No specific data on the treatment of vernolate in drinking water have been identified.

MEASUREMENT

United States Environmental Protection Agency (USEPA) method 525.2 for the determination of organic compounds in drinking water by liquid-solid extraction and capillary column gas chromatography with mass spectrometry (GC-MS) can achieve a limit of quantitation (LOQ) of 0.047 µg/L to 0.14 µg/L for vernolate (USEPA 1995). Vernolate can be extracted from water by liquid-liquid extraction with dichloromethane and analysed by GC/MS in selected ion monitoring mode, with a LOQ of 0.5 µg/L. USEPA method 634 for the determination of thiocarbamate pesticides in industrial and municipal wastewaters by gas chromatography is also approved for the analysis of vernolate in water (USEPA 1993). USEPA method 507 for the determination of nitrogen and phosphorus containing pesticides in water by gas chromatography with a nitrogen-phosphorus detector can achieve a LOQ of 0.13 µg/L (Munch 1995). Solid-phase microextraction (SPME), followed by gas-liquid chromatography (GC) employing a nitrogen-phosphorus detector can achieve a LOQ of 0.1 µg/L, and SPME-GC employing mass spectrometry can achieve a LOQ of 0.02 µg/L (Choudhury *et al.* 1996).

HISTORY OF THE HEALTH VALUES

The current acceptable daily intake (ADI) for vernolate is 0.01 mg per kg of bodyweight (mg/kg bw), based on a no-observed-effect level (NOEL) of 1 mg/kg bw/day from a reproduction study in rats. The NOEL is based on decreased bodyweight gain and food intake. The ADI incorporates a safety factor of 100 and it was first established in 1989.

The previous *Australian Drinking Water Guidelines* health value was 0.03 mg/L (NHMRC and NRMMC 2004).

HEALTH CONSIDERATIONS

Metabolism: Vernolate is rapidly absorbed via the gastrointestinal tract in rats. Metabolism is extensive, and proceeds through sulfoxidation. Excretion is primarily through urine in the form of conjugates and is complete by 7 days.

Acute effects: Vernolate has low acute oral and dermal toxicity. It is not a skin sensitiser.

Short-term effects: In 14-week dietary studies in rats and dogs, no effects were seen up to the highest doses tested, namely, 45 mg/kg bw/day in rats and 32 mg/kg bw/day in dogs.

Long-term effects: Long-term dietary studies were conducted in mice and rats. In mice, the only effects were changes in relative weights of kidney and liver at the highest dose tested, 100 mg/kg bw/day, and these were of doubtful toxicological significance. In rats, there was an increase in macrophage infiltration in lungs, and decreased bodyweight gain at the highest dose tested, 13 mg/kg bw/day. The lowest overall NOEL was 3.3 mg/kg bw/day in rats.

Carcinogenicity: Based on 2-year dietary studies in mice and rats, there is no evidence of carcinogenicity for vernolate.

Genotoxicity: Vernolate is not considered to be genotoxic, based on *in vitro* and *in vivo* short-term studies.

Reproductive and developmental effects: A 2-generation reproduction study in rats and developmental studies in mice and rabbits found no evidence of effects on reproductive parameters or foetal development. In the rat reproduction study, there was a decrease in parental food intake and an associated decrease in bodyweight gain at doses of 5 mg/kg bw/day and above in first generation parent animals. The NOEL of 1 mg/kg bw/day is the basis for the current ADI.

Neurotoxicity: A 35-day neurotoxicity study in hens found no evidence of delayed neurotoxicity at oral doses of 10 mg/kg bw.

NOTE: Important general information is contained in PART II, Chapter 6

Poisons Schedule: Vernolate is included in Schedule 5 of the *Standard for the Uniform Scheduling of Medicines and Poisons No.1, 2010* (the Poisons Standard)(DoHA 2010). Current versions of the Poisons Standard should be consulted for further information.

DERIVATION OF THE HEALTH-BASED GUIDELINE

The health-based guideline of 0.04 mg/L for vernolate was determined as follows:

$$0.04 \text{ mg/L} = \frac{1 \text{ mg/kg bodyweight/day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 100}$$

where:

- 1 mg/kg bw/day is the NOEL based on a 2-generation reproduction study in rats.
- 70 kg is taken as the average weight of an adult.
- 0.1 is a proportionality factor based on the assumption that 10% of the ADI will arise from the consumption of drinking water.
- 2 L/day is the estimated maximum amount of water consumed by an adult.
- 100 is the safety factor applied to the NOEL derived from animal studies. This safety factor incorporates a factor of 10 for interspecies extrapolation and 10 for intraspecies variation.

REFERENCES

NOTE: The toxicological information used in developing this fact sheet is from reports and data held by the Department of Health and Ageing, Office of Chemical Safety and Environmental Health.

Choudhury TK, Gerhardt KO, Mawhinney TP (1996). Solid-phase microextraction of nitrogen- and phosphorus-containing pesticides from water and gas chromatographic analysis. *Environmental Science and Technology,* 30(11):3259–3265.

DoHA (2010) ***The Poisons Standard; Schedule 1-Standard for the Uniform Scheduling of Medicines and Poisons***, Department of Health and Ageing, Commonwealth of Australia, Canberra (http://www.comlaw.gov.au/Details/F2010L02386/Download).

Munch JW (1995). *Method 507 Determination of Nitrogen and Phosphorus Containing Pesticides in Water by Gas Chromatography with a Nitrogen Phosphorus Detector*. National Exposure Research Laboratory, Office of Research and Development, United States Environmental Protection Agency, Cincinnati, Ohio.

NHMRC (National Health and Medical Research Council), NRMMC (Natural Resources Management Ministerial Council) (2004). *Australian Drinking Water Guidelines*. National Water Quality Management Strategy, Paper 6. NHMRC and NRMMC.

Tomlin CD (ed) (2006). *The Pesticide Manual: a world compendium*, 14th edition, British Crop Production Council, UK.

USEPA (United States Environmental Protection Agency) (1993). *Method 634. Methods for the Determination of Nonconventional Pesticides in Municipal and Industrial Wastewater.* USEPA United States Environmental Protection Agency, Washington DC,

USEPA (United States Environmental Protection Agency) (1995). *Method 525.2 Determination of Organic Compounds in Drinking Water by Liquid-Solid Extraction and capillarity Columns Gas Chromatography/ Mass Spectrometry Revision 2.0*. Environmental Monitoring Systems Laboratory Office of Research and Development, USEPA, Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

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Vinyl chloride

(endorsed 1996)

GUIDELINE

No safe concentration for vinyl chloride in drinking water can be confidently set. However, for practical purposes, the concentration should be less than 0.0003 mg/L, which is the limit of determination.

GENERAL DESCRIPTION

Vinyl chloride is used industrially in the production of poly vinyl chloride (PVC), which has wide application in the plastics, rubber, paper and glass industries.

Vinyl chloride may be present in drinking water through pollution of water sources by chemical spills. Water bottled and stored for long periods in PVC containers may contain very low concentrations of vinyl chloride. It has occasionally been detected in drinking water supplies that use PVC pipes in the United States and Germany, with a maximum reported concentration of 0.01 mg/L. In Australia there are stringent requirements on the maximum permissible residual vinyl chloride concentrations in PVC pipes and fittings.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Vinyl chloride has not been found in Australian drinking waters. It is included here to provide guidance in the unlikely event of contamination, and because it has been detected occasionally in drinking water supplies overseas.

TREATMENT OF DRINKING WATER

There are no published reports on methods for the removal of vinyl chloride from drinking water.

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for the analysis of vinyl chloride (USEPA Draft Method 502.1 1986). An inert gas is bubbled through the sample and vinyl chloride trapped on an adsorbent. The adsorbent is then heated and vinyl chloride analysed using gas chromatography with electron capture detection. The limit of determination is 0.0003 mg/L.

HEALTH CONSIDERATIONS

Vinyl chloride is readily absorbed following ingestion. It is metabolised to chloroethylene oxide, which can rearrange spontaneously to chloroacetaldehyde. Both substances are highly reactive and mutagenic.

In humans, vinyl chloride is a narcotic agent, and occupational exposure to high doses causes a number of symptoms including Raynaud's phenomenon, a painful disorder of the hands. This is not a concern for environmental exposure.

Vinyl chloride is a well-documented human carcinogen, with inhalation of high concentrations causing tumours in the liver, particularly angiosarcoma. Tumours in the brain and lung and malignancies of the lymphatic and haematopoietic tissues have also been reported.

No data are available on oral exposure in humans.

Vinyl chloride is also carcinogenic to animals. When administered by inhalation at doses above 100 ppm in air, it induced tumours of the liver and of some other organs in rats, mice and hamsters. Oral administration resulted in dose-related tumours of the liver at a dose of 14 mg/kg body weight per day. Some tumours were also observed in other organs, including the lungs and mammary glands.

Vinyl chloride has exhibited mutagenic activity in a variety of tests on bacteria and mammalian cells.

The International Agency for Research on Cancer has concluded that vinyl chloride is carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans) (IARC 1987).

DERIVATION OF GUIDELINE

Vinyl chloride is a genotoxic human carcinogen, and there is no safe or acceptable concentration for vinyl chloride in drinking water. The guideline of less than 0.0003 mg/L is based on a consideration of health effects in relation to the limit of determination.

i) The excess risk of lifetime consumption of drinking water with a vinyl chloride concentration of 0.0005 mg/L was conservatively estimated by the World Health Organization (WHO), using a linear multistage model, at one additional cancer per million people.

ii) A value of 0.0005 mg/L can also be derived as follows:

$$0.0005 \text{ mg/L} = \frac{0.13 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000}$$

where:

- 0.13 mg/kg body weight per day is the no-effect level from lifetime studies using rats (Feron *et al.* 1981, Til *et al.* 1991). Tumours were reported at higher doses.
- 70 kg is the average weight of an adult.
- 0.1 is the proportion of total daily intake attributable to the consumption of water.
- 2 L/day is the average amount of water consumed by an adult.
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for evidence of carcinogenicity).

The limit of determination is slightly less than the values derived from health considerations, and provides an adequate degree of protection. This is consistent with the general approach adopted for genotoxic human carcinogens (see Section 6.3.4).

The WHO guideline value of 0.005 mg/L was based on a calculation that estimated an additional lifetime risk of one fatal cancer per 100,000 people.

REFERENCES

Feron VJ, Hendriksen CFM, Speek AJ, Til HP, Spit BJ (1981). Lifespan oral toxicity study of vinyl chloride in rats. *Food and Cosmetics Toxicology*, 19:317–333.

IARC (International Agency for Research on Cancer) (1987). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Overall Evaluations of Carcinogenicity. An updating of IARC monographs volumes 1 to 42. World Health Organization, IARC, Supplement 7.

Til HP, Feron VJ, Immel HR (1991). Lifetime (149-week) oral carcinogenicity study of vinyl chloride in rats. *Food and Chemical Toxicology*, 29:713–718.

USEPA Draft Method 502.1 (1986). Volatile halogenated organic compounds in water by purge and trap gas chromatography. United States Environmental Protection Agency, Environmental Monitoring and Support Laboratory (EMSL), Cincinnati, Ohio.

NOTE: Important general information is contained in PART II, Chapter 6

Xylenes [CASRN 1330-20-7]

(endorsed 2013)

GUIDELINE

Based on aesthetic considerations (taste and odour), the concentration of xylenes in drinking water should not exceed 0.02 mg/L.

Based on health considerations the concentration of xylenes should not exceed 0.6 mg/L.

GENERAL DESCRIPTION

The term 'xylenes' encompasses three isomers of dimethylbenzene. The isomers are distinguished by the designations ortho- (o-) [CASRN 95-47-6], meta- (m-) [CASRN 108-38-3], and para- (p-) [CASRN 106-42-3], which specify to which carbon atoms of the benzene ring the two methyl groups are attached. o-Xylene is also known as 1,2-dimethylbenzene, m-xylene is also known as 1,3-dimethylbenzene, and p-xylene is also known as 1,4-dimethylbenzene. The mixture is a slightly greasy, colourless liquid commonly encountered as a solvent.

Xylenes occurs naturally in petroleum and coal tar and represent about 0.5–1% of crude oil, depending on the source (hence xylenes are found in small amounts in petrol and aviation fuels). Xylenes can also be formed naturally during forest fires (ATSDR, 2007). It is mainly produced from reformate, but is also obtained from coal carbonisation derived from coke ovens.

Xylenes have a taste and odour threshold of 0.02 mg/L.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

Xylenes have only rarely been identified in Australian drinking waters. Natural concentrations in most water sources are usually very low. Xylenes can occur naturally in groundwater as a result of proximity to, or contact with, coal seams, petroleum and gas deposits, and shales. It may be mobilised by extraction activities (Lesage *et al*., 1997; Leusch and Bartkow, 2011; Volk *et al*, 2011). However, contamination can occur, usually via exposure to petrochemicals in surface waters or groundwater. Known sources of groundwater contamination include leakage from sub-surface fuel storage tanks (do Rego & Netto, 2007) and nearby hydrocarbon deposits (IPCS, 1997). Emissions of fuel components from boating use is a known source of contamination of multiple-use lakes and reservoirs (Schmidt et al., 2004). Xylenes were reported in 3% of samples from an extensive groundwater survey undertaken in Denmark with the highest concentration being 0.000 03 mg/L (Juhler & Felding, 2003). Concentrations in groundwater in the USA were generally <0.001 mg/L, but were as high as 1 mg/L in contaminated areas (IPCS, 1997; ATSDR, 2007). Xylenes have been reported at up to 0.000 5 mg/L in municipal drinking water in Croatia (Karaconji *et al*., 2006), and are occasionally detected in drinking waters in the USA (Williams et al., 2004), up to a maximum of 0.012 mg/L (IPCS, 1997).

TREATMENT OF DRINKING WATER

Volatile organic chemicals such as xylenes are most commonly treated in drinking water by aeration stripping and/or adsorption to granular activated carbon (GAC). A conventional biologically active sand filter has been shown to be highly effective for the removal of xylenes from contaminated water, under suitable conditions (Arvin *et al*., 2004). Effective bioremediation of highly contaminated groundwaters has also been demonstrated (Sedran *et al*., 2004; Zein *et al*., 2006).

MEASUREMENT

A purge and trap gas chromatographic procedure can be used for the analysis of xylenes (APHA, AWWA & WEF, 2012). An inert gas is bubbled through the sample and xylenes are trapped on an adsorbent. The adsorbent is then heated and xylenes analysed using gas chromatography with mass spectrometric (GC-MS) detection (Method 6200 B) or photoionisation (PI) detection (Method 6200 C) (APHA, AWWA & WEF, 2012). The method detection limit is 38 ng/L for GC-MS and 24 ng/L for GC-PI (APHA, AWWA & WEF, 2012).

HEALTH CONSIDERATIONS

Xylenes are readily absorbed after inhalation and metabolised almost completely to methyl benzoic acid. They can cross the placenta. No data are available on human absorption after ingestion, or on health effects of oral exposure in humans.

A 2-year gavage study using rats and mice reported decreased growth at high doses (500 mg/kg body weight per day) but no xylene-related lesions (NTP 1986). There was no evidence of carcinogenicity in oral and skin administration studies using rats and mice, and xylenes were not mutagenic in tests using bacteria and mammalian cells.

The International Agency for Research on Cancer has concluded that xylenes are not classifiable as to their carcinogenicity in humans (Group 3, inadequate evidence in humans and in animals) (IARC 1989).

DERIVATION OF GUIDELINE

The USEPA (2009) has set a drinking water guideline of 10 mg/L for total xylenes, while the WHO (2011) proposes a guideline of 0.5 mg/L.

The health-based guideline value for xylenes in drinking water was determined as follows:

$$0.6 \text{ mg/L} = \frac{250 \text{ mg/kg body weight per day} \times 70 \text{ kg} \times 0.1}{2 \text{ L/day} \times 1000} \times \frac{5}{7}$$

where:

- 250 mg/kg body weight per day is the no effect level based on a 2-year gavage study using rats (NTP 1986).
- 70 kg is the average weight of an adult
- 0.1 is the proportion of total daily intake attributable to the consumption of water
- 2 L/day is the average amount of water consumed by an adult
- 1000 is the safety factor in using the results of an animal study as a basis for human exposure (10 for interspecies variations, 10 for intraspecies variations and 10 for the limited toxicological end point)
- 5/7 is used to convert data based on a 5 day per week feeding study to a 7-day week equivalent.

The WHO guideline value of 0.5 mg/L is based on an adult body weight of 60 kg. The difference in guideline values is not significant.

The health-based guideline value exceeds the taste and odour threshold of xylenes in water of 0.02 mg/L.

REFERENCES

American Public Health Association (APHA), American Water Works Association (AWWA) and Water Environment Federation (WEF) (2012). Standard Methods for the Examination of Water and Wastewater, 22nd Edition. Eds. Rice EW, Baird RB, Eaton AD and Clesceri LS.

Arvin, E., Engelsen, P. and Sebber, U. (2004) Biodegradation of gasoline compounds (BTEX) in a water works sand filter. *Water Science & Technology: Water Supply*, **4**(5-6), 29-33.

ATSDR (2007). Toxicological profile for xylene. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, USA.

do Rego, E. C. P. and Netto, A. D. P. (2007) PAHs and BTEX in groundwater of gasoline stations from Rio de Janeiro City, Brazil. *B. Environ. Contam. Tox.*, **79**(6), 660-664.

IARC (International Agency for Research on Cancer) (1989) IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: some organic solvents, resin monomers and related compounds, pigments and occupational exposures in paint manufacture and painting., Lyon.

IPCS (1997) Environmental Health Criteria 190: Xylenes. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland.

Juhler, R. K. and Felding, G. (2003) Monitoring methyl tertiary butyl ether (MTBE) and other organic micropollutants in groundwater: Results from the Danish National Monitoring Program. *Water Air Soil Poll.*, **149**(1-4), 145-161.

Karaconji, B., Skender, L. and Karacic, V. (2006) Benzene, toluene, ethylbenzene, and isomeric xylenes in various water samples in Croatia. *B. Environ. Contam. Tox.*, **76**(3), 458-462.

Lesage, S., Hao, X., and Kent, S. (1997) Distinguishing Natural Hydrocarbons from Anthropogenic Contamination in Ground Water. *Groundwater*, 35(1), 149-160

Leusch, F. and Bartkow, M. (2010) A short primer on benzene, toluene, ethylbenzene and xylenes (BTEX) in the environment and in hydraulic fracturing fluids http://www.ehp.qld.gov.au/management/coal-seam-gas/pdf/btex-report.pdf (accessed 19 March 2013)

NTP (National Toxicology Program) United States Department of Health and Human Services (1986) Toxicology and carcinogenesis gavage studies of xylenes (mixed) in F344/N rats and B6C3F1 mice. NTP Report No. 327.

Schmidt, T. C., Haderlein, S. B., Pfister, R. and Forster, R. (2004) Occurrence and fate modeling of MTBE and BTEX compounds in a Swiss Lake used as drinking water supply. *Water Res.*, **38**(6), 1520-1529.

Sedran, M. A., Pruden, A., Wilson, G. J., Suidan, M. T. and Venosa, A. D. (2004) Biodegradation of methyl tert-butyl ether and BTEX at varying hydraulic retention times. *Water Environ. Res.*, **76**(1), 47-55.

USEPA (2009) National Primary Drinking Water Regulations. United States Environmental Protection Agency, Washington, DC, USA.

WHO (2011) Guidelines for Drinking Water Quality, 4th Edition. World Health Organisation, Geneva, Switzerland.

Williams, P. R. D., Benton, L. and Sheehan, P. J. (2004) The risk of MTBE relative to other VOCs in public drinking water in California. *Risk Anal.*, **24**(3), 621-634.

Zein, M. M., Suidan, M. T. and Venosa, A. D. (2006) Bioremediation of groundwater contaminated with gasoline hydrocarbons and oxygenates using a membrane-based reactor. *Environ. Sci. Technol.*, **40**(6), 1997-2003.

NOTE: Important general information is contained in PART II, Chapter 6

NOTE: Important general information is contained in PART II, Chapter 6

Zinc

(endorsed 1996)

GUIDELINE

Based on aesthetic considerations (taste), the concentration of zinc in drinking water should be less than 3 mg/L.

No health-based guideline value is proposed for zinc.

GENERAL DESCRIPTION

Zinc is widely distributed and occurs in small amounts in almost all rocks, commonly as the sulfide.

It is used as a coating to prevent corrosion of iron and steel products, and in the manufacture of brass. Zinc oxide is an important component in the manufacture of paint and rubber products, including tyres.

In surface and ground waters, the concentration of zinc from natural leaching is usually less than 0.01 mg/L. Tap water can contain much higher concentrations as a result of corrosion of zinc-coated pipes and fittings. Zinc concentrations in galvanised iron rainwater tanks are typically 2 mg/L to 4 mg/L but have been reported as high as 11 mg/L.

Taste problems can occur if the zinc concentration in drinking water exceeds 3 mg/L. Water with a zinc concentration above 5 mg/L tends to be opalescent, develops a greasy film when boiled, and has an undesirable dry 'metallic' taste.

Zinc is present in plant and animal tissues, and food is the major source of zinc intake. Drinking water usually makes a negligible contribution to total intake.

TYPICAL VALUES IN AUSTRALIAN DRINKING WATER

In major Australian reticulated supplies, the concentration of zinc ranges up to 0.26 mg/L, with a typical concentration of 0.05 mg/L.

TREATMENT OF DRINKING WATER

Zinc concentrations in drinking water can be reduced by alum coagulation at pH 6.5–7 (30% removal) or by lime softening at pH 9.5 to pH 10 (60% removal).

MEASUREMENT

The concentration of zinc in drinking water can be determined by flame atomic absorption spectroscopy or inductively coupled plasma emission spectroscopy (APHA Method 3500-Zn Parts B or C 1992). The limits of determination are approximately 0.02 mg/L.

HEALTH CONSIDERATIONS

Zinc is an essential element for humans. The recommended intake for adults is 12 mg per day. Nutritional zinc deficiency results in retarded growth, anorexia, mental lethargy, skin changes and night blindness.

Approximately 20–30% of dietary zinc is absorbed by the gastrointestinal tract. Highest concentrations are found in the liver, kidney, bone, retina, prostate and muscle.

In humans, consumption of very high amounts of zinc can result in nausea, vomiting, diarrhoea and abdominal cramps. The major effects of long-term exposure to zinc are copper deficiency, anaemia and gastric erosion.

In animal studies, zinc has been reported to reduce the toxic effects of nickel and cadmium. High doses over long periods may, however, be toxic to nerve cells of mammals.

There is no evidence that occupational exposure to zinc increases the risk of cancer.

Zinc has been shown to induce chromosomal aberrations in mammalian cells, but is inactive in bacterial mutation tests.

DERIVATION OF GUIDELINE

The guideline value for zinc in drinking water has been based on the taste threshold of 3 mg/L. Higher zinc concentrations can impart an undesirable taste and a cloudy appearance. Zinc concentrations over 0.5 mg/L may indicate corrosion problems.

REFERENCES

APHA Method 3500-Zn Part B (1992). Zinc: Atomic Absorption Spectrometric method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

APHA Method 3500-Zn Part C (1992). Zinc: Inductively Coupled Plasma method. Standard Methods for the Examination of Water and Wastewater, 18th edition. American Public Health Association, Washington.

NOTE: Important general information is contained in PART II, Chapter 6

DRINKING WATER TREATMENT CHEMICALS

Aluminium chlorohydrate

(endorsed 2005)

Aluminium chlorohydrate is used as a primary coagulant in the treatment of drinking water. It is effective over a range of pH values and forms strong floc. It is particularly effective in some low alkalinity waters.

GENERAL DESCRIPTION

Aluminium chlorohydrate, $Al_2(OH)_5Cl$ (also known as ACH, polyaluminium chlorohydrate or aluminium chlorhydroxide), solution is a clear colourless, odourless liquid. It has a specific gravity of 1.32–1.35 at 25°C, a pH of 3.5–4.5, and is completely soluble in water.

ACH is of the polyaluminium chloride family, with a high aluminium oxide content and high basicity. It is supplied with an aluminium content of 12.2 to 12.7% (23–24% as equivalent alumina) and a basicity of 83–84%. The chemical coagulates over a wide pH range (pH 6–9) and does not usually require alkalinity adjustment.

The formula $Al_2(OH)_5Cl$ is simply a representation of the proportions of aluminium, hydroxide and chloride in the solution and it does not imply the predominant aluminium species is dimeric (see below). A generic formula for the ACH species may be given as $Al_n(OH)_mCl_{(3n-m)}$ where the m/n ration exceeds 1.05.

ACH can be stored in fibreglass-reinforced plastic, polyethylene, polypropylene or phenol formaldehyde, but can be corrosive to metals.

CHEMISTRY

ACH is manufactured from aluminium metal, which is reacted with either hydrochloric acid or aluminium chloride solution under controlled conditions.

ACH solution is a complex, dynamic mixture of positively charged polynuclear aluminium species, with no single species predominating and with molecular weights exceeding 1000. When applied to water, these species interact with and destabilises negatively charged colloidal matter, such as inorganic particles and the high molecular weight organic compounds that largely constitute natural organic matter. The polynuclear species also hydrolyse to form dense flocs of aluminium hydroxides that further act to entrap particles and remove some organic. An example of one of the many polynuclear species that may be present in ACH solution is the so called Al-13 ion that has the formula $[AlO_4.Al_{12}(OH)_{24}(H_2O)_{12}]^{13+}$.

The hydrolysis of ACH produces far less acid than the hydrolysis of aluminium sulfate owing to the very high degree of hydroxylation of the aluminium. As a result, ACH requires little of no pH correction with alkali when applied to water and results in only marginal increase in the concentration of dissolved salt.

The hydrolysis reaction proceeds as follows:

$$Al_2(OH)_5.Cl + H_2O \Leftrightarrow 2Al(OH)_3 + H^+ + Cl^-$$

As the hydrolysis reactions proceed, mononuclear hydroxide products can form polynuclear species. The reactions are complex and the species formed are quite variable. Examples of the species formed are:

- mononuclear: Al OH^{2+}, $Al(OH)_2^+$, $Al(OH)_3$ (solid precipitate), $Al(OH)_4^-$
- polynuclear: $Al_8(OH)_{20}^{4+}$, $Al_{13}O_4(OH)_{24}^{7+}$.

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, ACH is used as a primary coagulant. It is effective in cold temperatures and is particularly suited for use in low alkalinity raw water. It is commonly used for coagulation before membrane filtration, because this appears to reduce membrane fouling and prolong the life of the filter. The concentration of coagulant used depends on the properties of the raw water, including factors such as turbidity, dissolved organic carbon, temperature and alkalinity.

Typical ACH doses (with 23% Al_2O_3 content) are 3–100 mg/L. The actual concentration required should be determined by laboratory trials; higher doses may be required with particularly dirty water.

CONTAMINANTS

The contaminants that may be present in ACH are:

- antimony
- arsenic
- barium
- beryllium
- cadmium
- chromium
- copper
- fluoride
- iron
- lead
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- thallium
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ACH should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Most of the aluminium ions resulting from the use of ACH as a coagulant are removed by conventional water treatment processes. Residual chloride is usually at low levels that do not adversely affect drinking water quality.

STATUS

ACH was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Fitzgerald JJ and Rosenberg AH (1999). Chemistry of aluminium chlorohydrate and activated aluminium chlorohydrates. In *Cosmetic Science and Technology Series, 20. Antiperspirants and deodorants*, second edition, Laden K (ed). Marcel Dekker Inc, 83–136.

Rosenberg AH, Hodges RD and Harper TL (1995). *Chemical characterisation of polyaluminium chlorides and TOC removal*. American Water Works Association Water Quality Technology Conference.

Ruehl KE (1998). Effective coagulation for variable source water: a coagulant comparison by bench and full sale evaluations. American Water Works Association Water Quality Technology Conference.

NOTE: Important general information is contained in PART II, Chapter 8

Aluminium sulfate (alum)

(endorsed 2005)

Aluminium sulfate (alum) is a general purpose coagulant that is used in water treatment to remove turbidity, natural organic matter (NOM) (including colour), microorganisms and many inorganic chemicals. Removal of NOM reduces the formation of disinfection by-products, because it removes the organic precursors of the by-products.

GENERAL DESCRIPTION

For use in water treatment, aluminium sulfate (alum) is generally supplied as a bulk liquid, but it can also be supplied in granular form. The concentration of the supplied liquid solution varies, and users should establish the concentration with the supplier. Typically, alum solutions contain 7.5–8.4% Al_2O_3 w/w (i.e. 43–50% w/w $Al_2(SO4)_3{\cdot}14H_2O$), and have a specific gravity of 1.28–1.34 at 20°C. Solutions at the upper end of the available strengths may become unstable at low temperatures.

Alum is also available as a crystalline solid with varying degrees of hydration (14–18 H_2O). It has a pH of 1.2–3.0 and can be stored in rubber-lined containers or in fibreglass, stainless steel (type 316) or plastic.

CHEMISTRY

Alum is produced by the reaction of sulfuric acid with an aluminium-rich ore such as refined bauxite.

In water, the aluminium ion reacts with natural alkalinity (hydroxyl or bicarbonate) or added alkalinity (lime, caustic soda or soda ash) to form aluminium hydroxide species. The hydrolysis proceeds as follows:

$$Al_2(SO_4)_3.6H_2O \Leftrightarrow 2Al(OH)_3 + 6H^+ + 3SO_4^{2-}$$

As the hydrolysis reactions proceed, mononuclear products can form polynuclear species. The reactions are complex and the species formed are quite variable. Examples of the species formed are:

- mononuclear: $Al\ OH^{2+}$, $Al(OH)_2^+$, $Al(OH)_3$ (solid precipitate), $Al(OH)_4^-$
- polynuclear: $Al_8(OH)_{20}^{4+}$, $Al_{13}O_4(OH)_{24}^{7+}$

The generally positively charged Al species are available to interact with negatively charged colloidal matter in water. Such matter includes inorganic turbidity particles and the high molecular weight fraction of organic compounds present in NOM. The interaction destabilises the repulsive forces between the negatively charged particles, allowing them to collide and agglomerate to form microfloc (a process referred to as adsorption–destabilisation).

At higher concentrations of alum, metal hydroxides precipitate and can enmesh any colloidal particles in a process known as 'sweep coagulation', which renders water suitable for clarification. Alum has an optimum pH for coagulation of 5.5–7.5, with the lower end of the range (pH 5.5–6.2) being used for organics removal and enhanced coagulation (see below), and the higher end (pH 6.5–7.5) being used for sweep coagulation. Adsorption–destabilisation to form small floc, which can be removed by contact and direct filtration, typically occurs in the pH range 6–7.

'Enhanced coagulation' refers to coagulation at low pH with high doses of alum, and is used to remove NOM. The pH and alum dose need to be optimised, to maximise the removal of dissolved organic carbon (DOC).

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

The dose of alum used depends on the properties of the raw water, including (but not limited to) the turbidity, DOC, temperature and alkalinity. Waters of low turbidity often need higher doses of alum to bring about coagulation than more turbid waters. Indeed, waters of low turbidity and high colour are the most difficult to treat.

Typical alum doses (expressed as mg/L $Al_2(SO_4)_3 \cdot 14H_2O$) range from 5 to 200 mg/L and may even be as high as 500 mg/L if the water is particularly dirty.

The dose rate for alum is expressed in different units throughout Australia, and it is important to take this into account when comparing rates.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. In Australia, alum is produced by reacting aluminium trihydroxide or refined bauxite with sulfuric acid, and most of the impurities in the alum are derived from these raw materials.

The following chemical contaminants may be present in alum (NRC 1982):

- antimony
- arsenic
- barium
- beryllium
- cadmium
- chromium
- copper
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- thallium
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, alum should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Aluminium residuals after filtration can cause floc to form in the distribution system, which can cause customer complaints. To minimise residual levels of aluminium, alum should be used at pH and dosage conditions that exceed the solubility of aluminium. At 25°C, aluminium is least soluble at a pH near 6. At colder temperatures, the pH of minimum solubility increases. For example, at 4°C, aluminium is least soluble at pH 6.5–7. Hence, if water is treated at pH 6 throughout the year, levels of residual dissolved aluminium will be higher in winter. Poor dosage selection or inadequate mixing also leads to elevated aluminium residuals.STATUS

Aluminium sulfate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The 2003 revision did not change the status of this chemical for the treatment of drinking water.

NOTE: Important general information is contained in PART II, Chapter 8

REFERENCES

Amirtharajah A and Mills KM (1982). Rapid Mix Design for Mechanisms of Alum Coagulation. *Journal of the American Water and Wastewater Association* 74(4):210–216.

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B403-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Letterman RD, Amirtharajah A and O'Melia CR (1999). Coagulation and Flocculation. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 6.1–6.66.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Ammonia

(endorsed 2005)

Ammonia, NH_3, is added to drinking water to react with chlorine to form chloramine disinfectants. Chloramination is not as powerful as chlorination but provides a longer lasting residual in the water distribution system.

GENERAL DESCRIPTION

Ammonia, NH_3, is a colourless gas or liquid, with a sharp, intensely irritating odour. It is lighter than air and easily liquefied by pressure. Ammonia has a boiling point of –33.5°C, a freezing point of –77.7°C, and a specific gravity of 0.8 as a liquid. Ammonia gas is combustible and is very soluble in water. When hydrated, ammonia can attack copper, zinc and alloys containing these metals. Ammonia can be supplied as a compressed liquid (anhydrous ammonia), dissolved in water (aqueous ammonia) or as solutions of ammonium salts (e.g. ammonium sulfate).

Gaseous ammonia is compatible with some steels, stainless steel (type 316), neoprene and monel. Aqueous ammonia can be stored in iron, steel, stainless steel, fibreglass-reinforced plastic or rubber-lined vessels.

CHEMISTRY

Ammonia is prepared commercially in vast quantities, mostly using the Haber process to combine nitrogen directly with hydrogen. It can also be made using the cyanamide process, and is produced as a by-product of the destructive distillation of coal. Most of the ammonia produced is used to make fertilisers.

The reactions between ammonia and chlorine are complex, but the simplified equations shown below are often used. The chloramines produced are monochloramine (NH_2Cl — equation 1), dichloramine ($NHCl_2$ — equation 2) and trichloramine or nitrogen trichloride (NCl_3 — equation 3).

$$NH_3^+ + HOCl \rightarrow NH_2Cl + H_2O \quad (1)$$

$$NH_2Cl + HOCl \rightarrow NHCl_2 + H_2O \quad (2)$$

$$NHCl_2 + HOCl \rightarrow NCl_3 + H_2O \quad (3)$$

Other products are also formed, such as nitrogen (N_2) and nitrate (NO_3^-).

The sum of the concentrations of the three chloramine species is referred to as 'combined chlorine' and is often expressed as Cl_2, in the units of mg/L. The sum of the combined chlorine concentration and the free chlorine concentration (i.e. hypochlorous acid and hypochlorite ion) is referred to as 'total chlorine'. The relative amounts of the three species of chloramine formed depend on the ratio of chlorine to ammonia, the pH and the temperature. Monochloramines are preferred because they do not cause the taste and odour problems that can arise with dichloramines and trichloramines. Users should refer to available data on how pH and the ratio of chlorine to ammonia affect the distribution of chloramines (see discussion in the following section), and should be aware of the breakpoint phenomenon (whereby chlorine applied in sufficient doses will oxidise ammonia and eliminate chloramines, forming a free chlorine residual).

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking-water treatment, ammonia is added with chlorine (at a fixed ratio of ammonia to chlorine) to produce chloramine disinfectants. Chloramines react with bacteria and oxidisable material more slowly than free chlorine, but last longer than free chlorine. Depending on the order and process used trihalomethanes (THMs) may form. Chloramines thus tend to be used as a secondary disinfectant to provide a disinfectant residual in the distribution system, but may also be used as a primary disinfectant if an appropriate contact time is allowed. Chloramines are particularly suited to providing disinfectant residuals in long distribution systems, where it is difficult to maintain a residual using chlorine.

To produce monochloramine, the pH should be between 8 and 9, and the chlorine to ammonia ratio should be between 3:1 and 4:1. A ratio above 4:1 may produce chlorinous odours. Ammonia may be added before or after chlorine. In primary disinfection, chlorine is usually added first, because it kills bacteria, viruses and spores much more efficiently than does monochloramine, provided that sufficient contact time is allowed for disinfection before the ammonia is added. Ammonia and chlorine can be added together, provided that contact time is sufficient to ensure disinfection.

Chloramines present in water are harmful to people on kidney dialysis and to animal species in aquaria; therefore, it is important for water utilities using chloramination to inform consumers at risk.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. Ammonia is generally supplied at 99.9 % purity or better, but the product may include a very small amount of oil (hydrocarbons), heavy metals and water.

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ammonia should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*. Free ammonia liberated in the distribution system may contribute to nitrification problems or biological growth.

Chloramines may form some halogenated organic by-products. THMs may also be produced, but to a much lesser extent than with chlorination. More information on chloramines can be obtained from the Chloramine Fact Sheet in the *Australian Drinking Water Guidelines*.

STATUS

Aqueous ammonia was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington, DC.

Connell GF (1996). *The Chlorination/Chloramination Handbook*. Water Disinfection Series, American Water Works Association. Denver, Colorado.

Hass CN (1999). Disinfection. In *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed). American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 14.1–14.60.

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Ammonium sulfate

(endorsed 2005)

Ammonium sulfate is used as a source of ammonia to react with chlorine in drinking water treatment, to form chloramines. Chloramination is not as powerful as chlorination but provides a longer lasting residual in the water distribution system.

GENERAL DESCRIPTION

Ammonium sulfate, $(NH_4)2SO_4$, is an off-white crystal which is soluble in water (up to a concentration of 10 g/L). It has a specific gravity of 1.77 (at 20°C), and is available in several grades as 60–100% effective product.

Ammonium sulfate can be stored in rubber-lined vessels or in containers made from stainless steel (type 316), neoprene, monel, fibreglass-reinforced plastic, polyethylene or polyvinyl chloride. If the ammonium sulfate is dry, cast iron can also be used.

CHEMISTRY

Ammonium sulfate is by-product of the manufacture of caprolactam (a nylon-base material), coal gas and coke. It can also be prepared by the reaction of ammonia with sulfuric acid. It dissolves in water to form ammonium hydroxide (NH_4OH — equation 1), which then releases ammonia gas (NH_3 — equation 2):

$$(NH_4)_2 + 2H_2O \rightarrow 2NH_4OH + H_2SO_4 \qquad (1)$$

$$NH_4OH \Leftrightarrow NH_3 + H_2O \qquad (2)$$

Solutions of ammonium salts or aqueous ammonia (ammonia dissolved in water) have an alkaline pH. The actual pH depends on the concentration and the temperature. It is important to vent facilities storing ammonium salt solutions, because of the formation of ammonia gas. Ammonium salt solutions and aqueous ammonia have the same characteristics; therefore, the same care should be taken during handling.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Ammonium sulfate is used as a source of ammonia for disinfection (see ammonia fact sheet for further details).

The amount of ammonium sulfate to be added can be determined by multiplying the required ammonia level by the molecular ratio of 7.77 (i.e. $(NH_4)2SO_4 = 7.77 \times NH_3$). The ammonia fact sheet includes information on levels needed for chloramination.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. Ammonium sulfate may contain moisture and insoluble material as well as the following chemical contaminants (JECFA, NRC 1982):

- aluminium
- arsenic
- chloride
- lead
- pyridine
- selenium
- iron
- nickel

NOTE: Important general information is contained in PART II, Chapter 8

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ammonium sulfate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Excessive dosage can lead to biological growth in distribution system (see ammonia fact sheet for further details).

STATUS

Ammonium sulfate was originally endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B302-00. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Connell GF (1996). *The Chlorination/ Chloramination Handbook*. Water Disinfection Series, American Water Works Association, Denver, Colorado.

Hass CN (1999). Disinfection. In *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed). American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 14.1–14.60.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jefca/database/cover.htm>

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Calcium hydroxide

(endorsed 2005)

Calcium hydroxide (hydrated lime) is used to raise pH and adjust alkalinity for coagulation optimisation, corrosion control and water softening. It can also be used to dewater sludge.

GENERAL DESCRIPTION

Calcium hydroxide, $Ca(OH)_2$ (also known as lime or hydrated lime), adds hydroxide ions to water, thereby increasing its pH and alkalinity. It is a soft, white, crystalline powder.

The hydrated lime available commercially is a powder that contains mainly calcium hydroxide, or a mixture of calcium hydroxide and magnesium hydroxide. Pure hydrated lime has a specific gravity of 2.3–2.4. The bulk density of commercial lime varies from 450 to 560 kg/m^3, and it usually contains 80–96% calcium hydroxide. Its solubility at 20°C is 0.165% (or 0.165 g/100 g of saturated solution). Hydrated lime can be stored in rubber-lined containers or in fibreglass-reinforced plastic, polyethylene, polyvinyl chloride, cast iron or steel.

CHEMISTRY

Calcium hydroxide is obtained by hydrating quicklime with sufficient water to satisfy its chemical affinity for water. Quicklime is the product of the calcination of limestone, and consists mainly of the oxides of calcium (CaO) and magnesium (MgO). Calcium hydroxide is added to water to provide hydroxide ions to raise pH and alkalinity, and to neutralise free carbon dioxide or carbonic acid. It reacts with carbon dioxide to form calcium bicarbonate.

$$Ca(OH)_2 + 2CO_2 \rightarrow Ca(HCO_3)_2$$

To remove carbonate hardness, hydroxide ions are used to raise the pH of water. This causes precipitation, as bicarbonate ions are converted to the carbonate (pH > 10), precipitating calcium carbonate.

$$H_2CO_3 + Ca(OH)_2 \rightarrow CaCO_3(s) + 2H_2O$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In production of drinking water, calcium hydroxide is used:

- at the start of the water treatment process, to adjust pH and boost alkalinity, to assist coagulation
- at the end of the treatment process, to adjust final pH and alkalinity, and to minimise corrosion
- to soften hard waters by raising the pH, and thus precipitating calcium carbonate
- with carbon dioxide, to increase soft water's resistance to pH changes during distribution and to decrease its corrosivity
- to reduce the moisture content of sludge — if the concentration of calcium hydroxide is sufficiently high it will collapse the sludge structure, helping to reduce the water content of the sludge.

Lime is usually made up as a solution or as a slurry of up to 10% concentration; a slurry with a concentration of 1–5% is most commonly employed.

Typical lime concentrations used in drinking water treatment depend on the quality of the water to be treated and the purpose of the treatment (water softening, pH adjustment, alkalinity increase). Lime concentrations can vary from 5 to 500 mg/L, and the appropriate concentration should be determined by laboratory trials.

NOTE: Important general information is contained in PART II, Chapter 8

Poor mixing, poor pipe design, lime scaling and impurities often lead to blockages in lime dosing systems. To overcome such problems, the design of the system should minimise areas of solids accumulation, and the dosing system should be flushed each time it is turned off with water, chlorinated water or weak acid. Regular cleaning of the batch and dosing tanks using a solution of weak acid is also recommended.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in calcium hydroxide, depending on the source of the raw materials (JECFA, KIWA 1994, NRC 1982):

- aluminium
- arsenic
- barium
- cadmium
- chromium
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- selenium
- silica
- silver

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, calcium hydroxide should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Adding lime to water can significantly raise the turbidity. It can also increase the concentrations of iron, aluminium and manganese. Thus, it is often best to add lime at the start of the water treatment process, so that any impurities added with the lime can be removed during the treatment process.

The sludge resulting from water softening consists mainly of calcium carbonate, or a mixture of calcium carbonate and magnesium hydroxide. This sludge is generally dense, stable and inert; dries well; has a solids content of about 5% from the clarifier (although it can range from 2 to 30%); and has a pH greater than 10.5.

STATUS

Calcium hydroxide was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B202-02. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). APHA Method 2340B, C, Hardness. In: Standard Methods for the Examination of Water and Wastewater, 20th edition., American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). Compendium of Food Additive Specifications. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jefca/database/cover.htm>

KIWA (1994) Guideline quality of materials and chemicals for drinking water supplies. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

National Lime Association (1992). Chemical Lime Facts, 6th edition. National Lime Association, Washington, DC.

National Lime Association (1995). Lime: Handling, Application and Storage, 7th edition. National Lime Association, Arlington, Virginia.

NRC (National Research Council) (1982). Water Chemicals Codex. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, National Research Council.

NOTE: Important general information is contained in PART II, Chapter 8

Calcium hypochlorite

(endorsed 2005)

Calcium hypochlorite is a drinking water disinfectant used only for small systems.

GENERAL DESCRIPTION

Calcium hypochlorite, $Ca(OCl)_2$, is a white crystalline solid. It has a specific gravity of 2.35, decomposes in water and alcohol, is not hygroscopic and is practically clear in a water solution. The chemical is a highly active oxidiser and is relatively stable. The oxidising capability of 1 g calcium hypochlorite (65% strength) is equivalent to the oxidising capability of 0.65 g chlorine gas.

Calcium hypochlorite is available commercially as a dry solid, with a strength of up to 74% available chlorine. In this form, it loses about 0.013% of its strength per day under normal storage conditions, although the rate can be higher if the chemical is in contact with water or is exposed to the atmosphere. It is also available in a tablet form for use in automatic feed equipment at low-flow treatment plants or for dosing of in-system reservoirs.

Appropriate handling materials for calcium hypochlorite include glass, ceramics, fibreglass-reinforced plastic, polyethylene, polyvinyl chloride. Rubber-lined containers can also be used.

CHEMISTRY

Calcium hypochlorite is formed by the addition of chlorine to a slurry of 'milk of lime' (calcium hydroxide).

Calcium hypochlorite granules dissolve in water to form hypochlorous acid (HOCl), which partially dissociates to the hypochlorite ion (OCl^-).

$$Ca(OCl)_2 + 2H_2O \rightarrow 2HOCl + Ca(OH)_2$$

$$Ca(OCl)_2 \rightarrow Ca^{2+} + 2OCl^-$$

$$OCl^- + H2O \Leftrightarrow HOCl + OH^-$$

As with the addition of chlorine gas, the relative distribution of hypochlorous acid and hypochlorite ion resulting from the addition of calcium hypochlorite to water will depend on pH and temperature.

Calcium hypochlorite is a base and therefore raises the pH of water, whereas chlorine gas produces an acidic reaction that lowers the pH of the solution. The extent of the pH change depends on the alkalinity of the water.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Calcium hypochlorite is generally used as a disinfectant in smaller water treatment plants or in new water mains or in-system reservoirs.

As a disinfectant in water systems, calcium hypochlorite must be dissolved in water before it is added to the main supply. Doses usually range from 1 to 5 mg/L (as available chlorine), with 2–3 mg/L typical. Selection of the appropriate chlorine dose should take into account the C.*t* (disinfectant concentration × contact time) and chlorine residual required, and the levels of disinfection by-products likely to be formed. A free chlorine residual of ≥0.2 mg/L throughout the distribution system is preferred. Superchlorination (doses of 10 to 50 mg/L) may be used to disinfect or clean tanks and pipelines.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. Chemical contaminants that may be present in calcium hypochlorite include:

- aluminium
- arsenic
- barium
- cadmium
- chromium
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- selenium
- silica
- silver

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, calcium hypochlorite should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

The use of calcium hypochlorite as a disinfectant results in the formation of free chlorine, combined chlorine residuals and disinfection by-products. The by-products formed include trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones, chloral hydrate and chloropicrin. Although many specific chlorine disinfection by-products have been identified, many of the total organic halogens are as yet unidentified.

Among the many factors affecting the species formed as disinfection by-products are pH, temperature and levels of total organic carbon (TOC), bromide and chlorine. THMs (e.g. chloroform, bromodichloromethane, dibromochloromethane and bromoform) are the most widely known chlorination by-products. Chlorinated THM, HAA and HAN species are generally found at higher levels than brominated species; however, brominated species predominate in waters containing high levels of bromides.

The disinfection by-products most likely to occur and to be of concern to health are total THMs and THM species, total HAAs and HAA species.

STATUS

Calcium hypochlorite was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B300-99. AWWA CD-ROM (April 2003). Details at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington, DC.

Connell GF (1996). *The Chlorination/ Chloramination Handbook*. Water Disinfection Series, American Water Works Association, Denver.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Calcium oxide

(endorsed 2005)

Calcium oxide is used (after hydrating to produce 'slaked lime') to correct pH and adjust alkalinity, for coagulation optimisation, corrosion control and water softening. It can also be used to assist in the dewatering of sludge.

GENERAL DESCRIPTION

Calcium oxide, CaO, is also known as calx, quicklime, unslaked lime and burnt lime. It is a grey-white, hard, odourless solid, which sometimes has a yellowish or brownish tint due to the presence of iron. It crumbles on exposure to moist air and is soluble in acid. Calcium oxide reacts with water to form calcium hydroxide (slaked lime), releasing heat as it does so.

Calcium oxide is available in several grades, and is the least expensive way of obtaining calcium hydroxide. Quicklime has a specific gravity of 3.2–3.4. Its bulk density is 1030 kg/m^3 for pebble quicklime or 1050 kg/m^3 for powder quicklime; it usually contains approximately 94% calcium oxide.

Appropriate handling materials for calcium oxide include fibreglass-reinforced plastic, polyethylene, polyvinyl chloride, cast iron and steel. Rubber-lined containers can also be used.

CHEMISTRY

Calcium oxide is formed by calcination of limestone, and it can also contain magnesium oxide, MgO. Before being used in drinking water treatment, calcium oxide must be hydrated or 'slaked' to calcium hydroxide or slaked lime:

$$CaO + H_2O \rightarrow Ca(OH)_2$$

Slaked lime is added to water to provide hydroxide ions to raise pH and alkalinity, and to neutralise free carbon dioxide or carbonic acid. It reacts with carbon dioxide to form calcium bicarbonate:

$$Ca(OH)_2 + 2CO_2 \rightarrow Ca(HCO_3)_2$$

To remove carbonate hardness, hydroxide ions are used to raise the pH of water. This causes precipitation, as bicarbonate ions are converted to the carbonate (at pH > 10), precipitating calcium carbonate.

$$H_2CO_3 + Ca(OH)_2 \rightarrow CaCO_3\ (s) + 2H_2O$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In production of drinking water, slaked lime is used:

- at the start of the water treatment process, to adjust pH and boost alkalinity in order to assist coagulation
- at the end of the treatment process, to adjust final pH and alkalinity, and to minimise corrosion
- to soften hard waters by raising the pH, thus precipitating calcium carbonate;
- with carbon dioxide, to increase soft water's resistance to pH changes during distribution and to decrease its corrosivity
- to reduce the moisture content of sludge — if the concentration of calcium hydroxide is sufficiently high it will collapse the sludge structure, helping to reduce the water content of the sludge.

NOTE: Important general information is contained in PART II, Chapter 8

Slaked lime is usually made up as a solution or a slurry of up to 10% concentration; a slurry with a concentration of 1–5% is most commonly employed.

Typical slaked lime concentrations used in drinking water treatment depend on the quality of the water to be treated and the purpose of the treatment (e.g. water softening, pH adjustment or alkalinity increase). Slaked lime concentrations can vary from 5 to 500 mg/L, and the appropriate concentration should be determined by laboratory trials.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the source of the raw materials and on the manufacturing process. The following chemical contaminants may be present in calcium oxide (JECFA, KIWA 1994, NRC 1982):

- aluminium
- arsenic
- barium
- cadmium
- chromium
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- selenium
- silica
- silver

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, calcium oxide should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Adding slaked lime to water can significantly raise the turbidity and the concentrations of iron, aluminium and manganese. Thus, it is often best to add slaked lime at the start of the water treatment process, if possible, so that any impurities added with it can be removed during the treatment process.

STATUS

Calcium oxide was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B202-02. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jefca/database/cover.htm>

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

NOTE: Important general information is contained in PART II, Chapter 8

National Lime Association (1992). *Chemical Lime Facts*, 6th edition. National Lime Association, Washington, DC.

National Lime Association (1995). *Lime: Handling, Application and Storage*, 7th edition. National Lime Association, Arlington, Virginia.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC.

NOTE: Important general information is contained in PART II, Chapter 8

Carbon, granulated activated

(endorsed 2005)

Granular activated carbon is used in drinking water treatment to adsorb or biologically degrade dissolved organic matter, pesticides, algal toxins and compounds causing taste or odour problems. Use of activated carbon used before disinfection reduces the formation of disinfection by-products, by reducing the amount and reactivity of organic precursors of these by-products.

GENERAL DESCRIPTION

Granular activated carbon (GAC) is a black, solid, extremely porous material that can adsorb impurities and contaminants from air and water. It has a complex, porous internal structure, with internal surface areas averaging about 900 m^2/g and a bulk density of 250–600 kg/m^3. Activated carbon is insoluble in water and organic solvents.

The properties of activated carbon depend on its degree of activation and the raw material from which it is produced. Coal, wood and coconut-based activated carbons each have different pore structures and different characteristics.

GAC may act as a biological carrier by housing bacteria in its internal honeycomb structure. When GAC filters are used in an enhanced biological mode, they are referred to as biological activated carbon (BAC) filters. BAC filters work through two mechanisms: biodegradation of contaminants (e.g. taste and odour compounds, and organics) and biological regeneration of the carbon's adsorption sites.

Dry activated carbon can be stored in cast iron or steel silos. Wet activated carbon can be stored in plastic, rubber or silicon-lined containers, or in stainless steel (type 316), monel or bronze.

CHEMISTRY

Carbon is 'activated' by heating carbonaceous material such as wood, coal or coconut husks to high temperatures in a controlled atmosphere of steam, or at moderate temperatures in the presence of chemicals such as acid.

The adsorptive properties of GAC vary with pore size, pore-size distribution, internal surface area of the pores and surface properties. The properties of the GAC available in the market are variable. In selecting an activated carbon product, it is important to consider factors such as the adsorptive capacity of the activated carbon, the desired application, abrasion resistance during backwashing and cost. The quality of the activated carbon can be determined by its ability to remove contaminants such as 2-methylisoborneol (MIB), geosmin, toxins and pesticides, and by a number of other factors that are listed below, together with typical ranges (actual values will depend on the raw material and the activation processes):

- iodine number: 900–1300 mg/g carbon
- apparent density: 0.2-0.6 g/cc
- moisture content: 3-8%
- abrasion resistance: 75-99%
- particle size distribution: 5% maximum on upper sieve
  90% minimum between sieves
  5% maximum through lower sieve
- ash content: 3–15%

NOTE: Important general information is contained in PART II, Chapter 8

The adsorptive capacity of activated carbon can be inferred from the iodine number, methylene blue number or molasses number.

Effective sizes of GAC are typically 0.7–1.2 mm, with a uniformity coefficient (UC) generally specified to be less than 1.8. The GAC is installed over supporting layers of sand and gravel.

After installation in the filter bed, the GAC is carefully wetted over several hours. In some carbons, significant flotation of the carbon may occur in the wetting phase, and the floating portion of GAC is removed and disposed of. The floatable component of the GAC may vary between 0 and 30%.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In water treatment, activated carbon is used to control taste and odour-causing compounds, and to remove contaminants such as nitrates, pesticides, algal toxins, disinfection by-products, organic carbon and other trace organic chemicals.

GAC is generally used as a filter medium in beds or tanks, with the water being treated as it passes through the filter. Contaminants are removed through adsorption and biological degradation. Many taste and odour compounds (e.g. 2-methylisoborneol (MIB), geosmin and 20–50% of natural organics) can be biologically degraded, and GAC filters used in this way can operate for 10–15 years. If the contaminant is not biodegradable, the GAC medium can be used continuously until its adsorption capacity is exhausted; and can then be reactivated using a thermal process (currently not available in the Australian drinking water industry). In adsorption mode, a GAC bed is effective for about 1 month to 2 years, depending on the concentration of contaminants in the water.

GAC beds can be used either before or after conventional treatment (i.e. pre-filtration or post-filtration). GAC can also be used for a combination of filtration and adsorption, either as a full GAC bed, or as a layer of sand topped with a layer of GAC medium. The process can be preceded by ozonation, which encourages biological activity on the filter (creating a BAC filter), thus prolonging the life of the filter. Ozonation generally produces water that is more biologically stable and has a lower chlorine demand.

GAC and BAC filters are designed for a specific empty bed contact time (EBCT), which typically ranges from 5 to 25 minutes. The most economic EBCT can be determined by analysing particular contaminants of concern, at either laboratory or pilot scale.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in the ash that may be found in activated carbon:

- aluminium
- arsenic
- chromium
- iron
- lead
- manganese
- mercury
- phosphorus
- silver
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, activated carbon should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

NOTE: Important general information is contained in PART II, Chapter 8

Degraded GAC can pass through a water treatment plant, causing black specks and deposits in the distribution system, although it is unlikely that significant quantities of carbon residues will be present in finished water.

STATUS

Activated carbon was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard B604-96. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Gosselin RE, Smith, RP and Hodge HC (1984). *Clinical Toxicology of Commercial Products*, 5th edition. Williams and Wilkins, Baltimore, II-94.

IARC (International Agency for Research on Cancer) (1984). *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*. World Health Organization, Geneva, Switzerland.

NIOSH (National Institute for Occupational Safety and Health) (1984). Method 5000, Carbon Black (issued 2-15-84). In: *NIOSH Manual of Analytical Methods. Methods A-Z & Supplements*, 4th edition.

U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. U.S. Government Printing Office ,Washington, DC.

Snoeyink VL and Summers RS (1999). Adsorption of Organic Compounds. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed). American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 13.1–13.76.

Urfer D, Huck PM, Booth SDJ, and Coffey BM (1997) Biological Filtration for BOM and particle removal: a critical review. *AWWA Journal* 89(12) 83-98.

NOTE: Important general information is contained in PART II, Chapter 8

Carbon, powdered activated

(endorsed 2005)

Powdered activated carbon is used in drinking water treatment to adsorb dissolved organic matter, pesticides, algal toxins and compounds causing taste or odour problems. Adding activated carbon before disinfection reduces the formation of disinfection by-products, by reducing the amount and reactivity of organic precursors of these by-products.

GENERAL DESCRIPTION

Powdered activated carbon (PAC) is a black, solid, extremely porous material that can adsorb impurities and contaminants from air and water. It has a complex, porous internal structure, with internal surface areas averaging about 900 m^2/g and a bulk density of 250–600 kg/m^3. Activated carbon is insoluble in water and organic solvents.

The properties of activated carbon depend on its degree of activation and the raw material from which it is produced. Coal, wood and coconut-based activated carbons each have different pore structures and different characteristics.

Dry activated carbon can be stored in cast iron or steel silos. Wet activated carbon can be stored in plastic, rubber, or silicon-lined containers, or in stainless steel (type 316), monel or bronze.

CHEMISTRY

Carbon is 'activated' by heating carbonaceous material such as wood, coal or coconut husks to high temperatures in a controlled atmosphere of steam, or at moderate temperatures in the presence of chemicals such as acid.

The adsorptive properties of PAC vary with particle size, pore size, pore-size distribution, internal surface area of the pores and surface properties. The properties of the PAC available in the market are variable. In selecting an activated carbon product, it is important to take into account factors such as the adsorptive capacity of the activated carbon, the desired application and the cost. The quality of the activated carbon can be determined by its ability to remove contaminants such as 2-methylisoborneol (MIB), geosmin, toxins and pesticides, and by a number of other factors that are listed below, together with typical ranges (actual values will depend on the raw material and the activation processes):

- iodine number: 800–1400 mg/g carbon
- apparent density: 0.2-0.6 g/cc
- moisture content: 3-8%
- particle size distribution: 90% minimum through 100 µm mesh
  95% minimum through 200 µm mesh
- ash content: 3–15%

The adsorptive capacity of activated carbon can be inferred from the iodine number, methylene blue number or molasses number.

Effective sizes of PAC are typically 20–50 µm.

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, PAC can be added as a powder by dry-feed equipment; for higher dosing, it can be added as a slurry by metering pumps or feeders. It is important to add PAC early in the treatment process, before addition of chemicals such as chlorine, to ensure sufficient contact time and to avoid chemicals being adsorbed onto the carbon. For intermittent or low dosing, Ideally, PAC is added 30 minutes before coagulation; often near the raw water source. Care should be taken to avoid areas where PAC may build up (e.g. low-velocity pipes). The carbon is mixed for a short time before being removed by settling or filtration.

If PAC is added in the coagulation zone, additional PAC may be required, because the carbon can become bound in flocs, diminishing its effectiveness. Jar testing reflecting the operating conditions can determine the effective dose rate and contact time for optimal performance of PAC.

Occasionally, PAC is dosed immediately before filtration, where it reacts with organics above and within the filter bed. Care should be taken to avoid breakthrough of PAC caused by normal sludge removal processes (e.g. clarifier sludge blowdowns, flotation or filter backwashing).

The amount of PAC required will depend on the type and concentration of organics in the water. Typical values range from 2 to 60 mg/L, but can be as high as 100 mg/L. A contact time of 10–30 minutes between the PAC and the water generally removes most taste and odour compounds, but a longer time may be needed for removal of MIB and geosmin (the compounds most often linked with tastes and odours — see the fact sheet on taste and odour).

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in the ash that may be found in activated carbon:

- aluminium
- arsenic
- chromium
- copper
- iron
- lead
- manganese
- mercury
- phosphorus
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, PAC should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Powdered carbon slurry applied to raw water is easily removed by other water treatment processes (e.g. by settled sludge, floated sludge or filtration). PAC can pass through a water treatment plant, causing black specks and deposits in the distribution system, although it is unlikely that significant quantities of carbon residues will be present in finished water.

STATUS

Activated carbon was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

NOTE: Important general information is contained in PART II, Chapter 8

REFERENCES

AWWA (American Water Works Association)/ANSI (American National Standards Institute) (1997). Standard B604-96. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Gosselin RE, Smith, RP and Hodge HC (1984). *Clinical Toxicology of Commercial Products*, 5th edition. Williams and Wilkins, Baltimore, II-94.

IARC (International Agency for Research on Cancer) (1984). *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*. World Health Organization, Geneva, Switzerland.

NIOSH (National Institute for Occupational Safety and Health) (1984). Method 5000, Carbon Black (issued 2-15-84). In: *NIOSH Manual of Analytical Methods. Methods A-Z & Supplements*, 4th edition. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. U.S. Government Printing Office ,Washington, DC.

Snoeyink VL and Summers RS (1999). Adsorption of Organic Compounds. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed). American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 13.1–13.76.

NOTE: Important general information is contained in PART II, Chapter 8

Chlorine

(endorsed 2005)

Chlorine is widely used as a primary disinfectant in the treatment of drinking water and to provide secondary disinfection in reticulation. It is also used to oxidise metals, to break down organics and to minimise biofouling. Chlorine produces potentially harmful disinfection by-products with some organics.

GENERAL DESCRIPTION

Chlorine, Cl_2, is a dense, greenish-yellow, diatomic gas with a pungent and irritating odour. It is noncombustible, but supports combustion as an oxidizing agent. The liquefaction pressure of chlorine is 7.86 atm (25°C).

Chlorine is relatively inexpensive and easy to use, although the risks associated with its transportation, storage and handling must be managed. Liquefied chlorine gas is supplied in pressurised containers of varying sizes, typically 70 kg and 990 kg. Free chlorine can also be generated on-site from electrolysis of sodium chloride solutions (brine).

Appropriate materials for handling chlorine gas include steel, copper and black iron. Aqueous chlorine can be stored in fibreglass-reinforced plastic or polyvinyl chloride.

CHEMISTRY

Chlorine is manufactured by the electrolytic dissociation of salt (sodium chloride), using mercury, diaphragm or membrane cells.

The dissolution of chlorine gas in water results in rapid hydrolysis, forming chloride ion (Cl^-), and hypochlorous acid (HOCl). Being a weak acid, HOCl is partially dissociated to hypochlorite ion (OCl^-). The degree of dissociation in equation 2 varies with temperature and pH. An increase in pH will shift the equilibrium to the right.

$$Cl_2 + H_2O \rightarrow HOCl + H^+ + Cl^- \qquad (1)$$

$$HOCl \Leftrightarrow H^+ + OCl^- \qquad (2)$$

The sum of the three species (i.e. Cl_2, HOCl and OCl^-) is referred to as 'free available chlorine' (FAC). The concentrations of the individual species and their sum are expressed as Cl_2, in units of mg/L.

At 25°C, hypochlorous acid is the predominant species between pH 1 and pH 7.5, and hypochlorite ion predominates at pH values greater than 7.5. Oxidation reactions and disinfecting properties of chlorine tend to be more effective at low pH values, because of the predominance of hypochlorous acid, which is a stronger oxidant.

The pH of water dosed with chlorine is affected by the amount used and the alkalinity in the water. In water with low alkalinity, the pH will drop after addition of gaseous chlorine, although it will rise if sodium hypochlorite is added.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Chlorine is employed as a strong oxidant or disinfectant, and also to provide a disinfectant residual in water distribution systems.

NOTE: Important general information is contained in PART II, Chapter 8

Chlorine can be added at various points of the treatment process:

- for oxidation of organics or metals
- for disinfection purposes
- to maintain a chlorine residual in the distribution system (pre-coagulation, intermediate or postfiltration chlorination).

Doses are usually 1–5 mg/L, although 2–3 mg/L is typical. The selection of the appropriate chlorine dose should take into account the amount of disinfection by-products formed and the required C.*t* value (concentration × contact time) and chlorine residual; the WHO recommendation is 0.5 mg/L for 30 minutes. A free chlorine residual of ≥0.2 mg/L throughout the distribution system is preferred. In some systems, rechlorination is employed within the distribution system, where chlorine is added after water has left the treatment plant, to boost chlorine residuals.

Superchlorination (10–50 mg/L) may be used to disinfect or clean tanks or pipelines, or to temporarily treat tastes and odours associated with high ammonia levels. This process is usually followed by dechlorination, to chemically remove excess chlorine. Knowledge of the breakpoint phenomenon (whereby chlorine applied in sufficient doses will oxidise ammonia and eliminate chloramines, resulting in the formation of a free chlorine residual) is also necessary when dealing with water containing ammonia.

The fact sheet on ammonia discusses the use of chorine with ammonia to produce chloramines.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in chlorine (NRC 1982, JECFA):

arsenic	manganese
carbon tetrachloride	mercury
lead	trihalomethanes

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, chlorine should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

The use of a disinfectant such as chlorine results in the formation of free chlorine and combined chlorine residuals and disinfection by-products, including trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones, chloral hydrate and chloropicrine. Although many specific chlorine disinfection by-products have been identified, several of the total organic halogens have yet to be identified.

Factors affecting the distribution of disinfection by-product species include pH, temperature and the levels of total organic carbon (TOC), bromide and chlorine. THMs (e.g. chloroform, bromodichloromethane, dibromochloromethane and bromoform) are the best known chlorination by-products. Chlorinated THM, HAA and HAN species generally dominate over brominated species. However, brominated species predominate in high-bromide waters.

NOTE: Important general information is contained in PART II, Chapter 8

STATUS

Chlorine was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B301-99. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington, DC.

Connell GF (1996). *The Chlorination/ Chloramination Handbook*. Water Disinfection Series, American Water Works Association, Denver, Colorado.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jefca/database/ cover.htm>

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Copper sulfate

(endorsed 2005)

Copper sulfate is an active constituent in registered algicide products used in drinking water reservoirs. There are different State and Territory environment protection regulations on the use of copper sulfate in reservoirs. Further information should be sought form the relevant State or Territory agency.

GENERAL DESCRIPTION

Copper sulfate, $CuSO_4$, is a blue crystal, or blue crystalline granule or powder, but is white when dehydrated. The chemical has a nauseous metallic taste and is poisonous. The anhydrous form contains nearly 50% copper; the commonly used pentahydrate form ($CuSO_4 \cdot 5H_2O$) contains 25.5% copper.

Appropriate handling materials for copper sulfate include fibreglass-reinforced plastic, polyethylene, polyvinyl chloride, cast iron and stainless steel. Rubber-lined and silicon-lined containers can also be used.

CHEMISTRY

Copper sulfate is the product of the reaction of sulfuric acid with copper metal, cupric oxide or basic copper salts.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Copper sulfate is an algicide, and is used to treat toxic or odorous algal blooms in water reservoirs and other water supply storages. Copper sulfate may kill aquatic plants, insects, invertebrates and fish. Copper sulfate is subject to registration and labelling requirements of the Australian Pesticides and Veterinary Medicines Authority. Copper sulfate is not registered for general use as an algicide in all jurisdictions therefore, before copper sulfate is used in a water storage system, the State or Territory environment protection authority must be advised. In some States and Territories, a licence must be obtained for its use. The *Australian and New Zealand Guidelines for Fresh and Marine Water Quality* (2002) contain information on the effect of copper sulfate on various ecosystems. There is a range of alternative water treatment and storage management methods for controlling the risks of toxic algal bloom including reducing the amounts of nutrient inflow to water reservoirs.

The application of copper sulfate products to storages should be in accordance with the registered chemical label. Copper sulfate can be applied by:

- dissolving crystals of the chemical into the water using porous bags pulled by a boat
- applying the crystals directly using a hopper feeder
- spraying dissolved copper sulfate on the water surface.

To determine the appropriate dose rate and ensure efficient application, knowledge of algal habitat and distribution is needed. Experience with the use of copper sulfate to treat cyanobacteria indicates that it is best to start applying the chemical early in the morning, and to apply it during calm conditions. This is because cyanobacteria tend to be most buoyant at this time, and are likely to be near the surface.

For a stratified reservoir, calculation of the total amount of algicide to be added is based on the amount needed to treat the surface of the water body, because this is where most cyanobacteria will be located. Treatment of algae should be concentrated in areas of algae scum.

NOTE: Important general information is contained in PART II, Chapter 8

The amount of copper sulfate required will depend on various factors, such as pH, alkalinity and water temperature (algae are more likely to bloom in warm water).

Copper sulfate is most effective at pH values of around 8, and alkalinity less than 50 mg/L. In conditions of high alkalinity or pH, addition of an acid (e.g. citric acid) may also be needed for the copper sulfate to be effective. The concentrations of copper sulfate added are typically in the range 0.2–1 mg Cu/L, depending on the specific type of organism being controlled.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. Chemical contaminants that may be present in copper sulfate include (JECFA):

- arsenic
- chloride
- iron
- lead
- nickel

RESIDUE AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, copper sulfate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*. A limit of 2 mg/L based on health considerations and of 1 mg/L for aesthetic considerations has been established for copper residues resulting from the use of copper sulfate.

Copper sulfate breaks down algae, resulting in the release of algal toxins and odorous substances that decay over time. Hence, a withholding period is needed after copper sulfate has been used as an algicide, and it may be necessary to monitor copper residues, toxins and odours during a follow-up period.

Copper sulfate products should not be used to treat more than half of a lake or pond at one time, in order to avoid depletion of oxygen caused by decaying vegetation. One to two weeks should be allowed between copper sulfate treatments to allow water oxygen levels to recover.

Copper entering a water treatment plant may be removed to some degree through coagulation with clarification/filtration. Elevated pH assists in copper removal.

STATUS

Copper sulfate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B602-02. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

ANZECC (Australia and New Zealand Environment and Conservation Council)/ARMCANZ (Agriculture and Resource Management Council of Australia and New Zealand) (2002). *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*. National Water Quality Management Strategy, ANZECC/ARMCANZ, Canberra.

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York, 315.

National Registration Authority for Agricultural and Veterinary Chemicals (1996). *The Requirements Manual for Agricultural Chemicals*. National Registration Authority, Canberra.

Ramadan T (2000). Algae control solves aesthetic problems. *Opflow* 26(8): 1–4.

Ferric chloride

(endorsed 2005)

Ferric chloride is used as a primary coagulant in the treatment of drinking water, particularly when a broad coagulation pH range is required It is used to remove turbidity, natural organic matter (NOM) (including colour), microorganisms and many inorganic chemicals. Removal of NOM reduces the formation of disinfection by-products, because it removes the organic precursors of the by-products.

GENERAL DESCRIPTION

Ferric chloride, $FeCl_3$ (anhydrous) or $FeCl_3 \cdot 6H_2O$ (crystalline), has a brownish-yellow or orange colouration when in crystalline form and is very hygroscopic. In solution, it has the appearance of a dark-brown syrup. Solutions of ferric chloride are acidic and corrosive to most metals. The typical pH range of a 1% solution of ferric chloride is 3–4. The chemical is significantly more soluble in hot water (535.7 g/100 mL at 100°C) than in cold water (74.4 g/100 mL at 0°C), and is very soluble in alcohol, ether and methanol.

Ferric chloride is available as a powder and in solution at 30–42%. A 42% solution of ferric chloride has a specific gravity of 1.45 at 20°C, contains 14.5% iron and has a pH of 1–2.

Ferric chloride is highly corrosive to most metals, including stainless steel; however, it can be stored or transported in fibreglass, rubber-lined carbon steel, polyvinyl chloride, polyethylene or polypropylene.

Polytetrafluoroethylene and polyvinylidene difluoride are also suitable as lining materials.

CHEMISTRY

Ferric chloride is obtained from ores containing iron and titanium oxides. It is also produced through the reaction of chlorine gas with iron, ferrous sulfate or ferrous chloride.

The positively charged Fe species are available to interact with negatively charged colloidal matter in water. Such matter includes inorganic turbidity particles and the high molecular weight fraction of organic compounds present in natural organic matter (NOM). Fe cations interact with the natural alkalinity to form hydroxides that then act in a charge neutralisation fashion similar to that for aluminium. Charge neutralisation destabilises the repulsive forces between the negatively charged particles, allowing them to approach closely, collide and agglomerate. Metal hydroxides precipitate and can enmesh any colloidal particles. Iron floc is generally large and settles rapidly though it may be weaker than alum floc. As for aluminium, sweep coagulation can also occur at higher doses.

The stoichiometry of the precipitation of iron hydroxide is described as follows:

$$FeCl_3\ (s) \rightarrow Fe^{+3} + 3Cl^-$$

$$Fe^{+3} + 3OH^- \rightarrow Fe\ (OH)_3(s)$$

Ferric chloride is an effective coagulant at a pH between 4 and 11. When added to water, ferric chloride consumes more alkalinity than does alum.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Ferric chloride is used as a primary coagulant, especially when a broader coagulation pH range is required.

The amount of ferric chloride added depends on the properties of the raw water, including factors such as turbidity, NOM, temperature and alkalinity.

Typical ferric chloride doses are 2–100 mg/L $FeCl_3 \cdot 6H_2O$, although higher doses may be required if water

NOTE: Important general information is contained in PART II, Chapter 8

is particularly dirty. At high doses, product water should be tested to ensure that maximum contaminant levels have not been exceeded.

The dose rate for ferric chloride may refer to crystalline or anhydrous ferric chloride, supplied as liquid or as iron. Care should be taken when interpreting dose rates to ensure that comparisons are relevant.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (KIWA 1994, NRC 1982:

- antimony
- arsenic
- cadmium
- chromium
- cobalt
- copper
- cyanide
- lead
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- titanium
- vanadium
- zinc

Manganese concentrations in ferric chloride may be high enough to affect the treated water.

RESIDUE AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ferric chloride should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Conventional water treatment processes, if optimised, remove almost all of the ferric ions produced when ferric chloride is used for coagulation. Residual chloride is usually at low levels, which do not adversely affect drinking water quality.

The presence of any ferrous iron in the product reduces its effectiveness in water treatment and increases the possibility of soluble iron carry over. This could cause post precipitation of ferric hydroxide (red water) in the distribution system.

STATUS

Ferric chloride was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B407-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater,* 20th edition. American Public Health Association, Washington, DC.

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Ferric sulfate

(endorsed 2005)

Ferric sulfate is used as a primary coagulant in the treatment of drinking water, particularly when a broad coagulation pH range is required. It is used to remove turbidity, natural organic matter (NOM) (including colour), microorganisms and many inorganic chemicals. Removal of NOM reduces the formation of disinfection by-products, because it removes the organic precursors of the by-products.

GENERAL DESCRIPTION

Ferric sulfate, $Fe_2(SO_4)_3$, is a yellow crystal or greyish-white powder that is soluble in water. In water treatment, it is usually supplied as an aqueous solution of 39–45% w/w ferric sulfate (11–12.5% Fe). The liquid solution has a specific gravity of 1.5–1.6 and is red-brown in colour. A 1% solution of ferric sulfate is acidic (pH 3–4).

Ferric sulfate is also available in granular form with an iron content of 18.5–20% and a pH of less than 1. It is not as corrosive as ferric chloride. Ferric sulfate can be stored or transported in stainless steels, lead, fibreglass, rubber-lined carbon steel, polyvinyl chloride, polyethylene or polypropylene.

Ferric sulfate can be used in a system built for alum dosing, whereas ferric chloride cannot.

CHEMISTRY

Ferric sulfate is produced by the oxidation of ferrous sulfate or by dissolving ferric oxide in sulfuric acid. In water, the ferric (iron III) ion hydrolyses and precipitates, to an extent that depends on pH and dosage. Iron precipitates formed are goethite, $HFeO_2$, and iron hydroxide, $Fe(OH)_3$, which are less soluble than aluminium precipitates. At equilibrium, the concentration of the soluble species is very low. Fe cations interact with the natural alkalinity to form hydroxides that then act in a charge neutralisation fashion similar to that for aluminium. Charge neutralisation destabilises the repulsive forces between the negatively charged particles, allowing them to approach closely, collide and agglomerate. Metal hydroxides precipitate and can enmesh any colloidal particles. Iron floc is generally large and settles rapidly though it may be weaker than alum floc. As for aluminium, sweep coagulation can also occur at higher doses.

The stoichiometry of the precipitation of iron hydroxides is described as follows:

$$Fe_2 (SO_4)_3 \rightarrow 2Fe^{+3} + 3SO_4^{2-}$$

$$Fe^{+3} + 3OH^- \rightarrow Fe (OH)_3 (s)$$

Ferric sulfate is an effective coagulant at pH values between 4 and 11.

TYPICAL USE IN DRINKING WATER TREATMENT

Ferric sulfate is used as a primary coagulant in the treatment of drinking water, particularly when a broad coagulation pH range is required.

The dose of ferric sulfate used depends on the properties of the raw water, including factors such as turbidity, natural organic matter (NOM), temperature and alkalinity.

Typical ferric sulfate doses, expressed as mg/L $Fe_2(SO_4)_3$, range from 2 mg/L to 100 mg/L although higher doses may be required if the raw water is excessively dirty. At high doses, product water should be tested to ensure that maximum contaminant levels have not been exceeded.

The dose rate of ferric sulfate may be expressed as crystalline ferric sulfate, as supplied liquid or as iron. Care should be taken when interpreting dose rates to ensure that any comparisons made are relevant.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies depending on the manufacturing process. Chemical contaminants that may be present in ferric sulfate are (JECFA, KIWA 1994, NRC 1982):

- antimony
- arsenic
- cadmium
- chromium
- cobalt
- copper
- cyanide
- lead
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- titanium
- vanadium
- zinc

In some products, manganese concentrations may be high enough to affect the treated water.

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ferric sulfate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Almost all of the ferric ions used for coagulation are removed by optimised conventional water treatment processes. Residual sulfate is usually at low levels which do not adversely affect drinking water quality.

The presence of any ferrous iron in the product reduces its effectiveness in water treatment and increases the possibility of soluble iron carry over. Iron residuals after filtration can cause floc to form in the distribution system, which can give rise to customer complaints. To minimise residual levels of iron, pH and dosage conditions should exceed the solubility of iron. Poor dosage selection or inadequate mixing also leads to elevated iron residuals.

STATUS

Ferric sulfate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and* ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B406-97. AWWA CD-ROM (April 2003). Details at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/ cover.htm>

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Hydrochloric acid

(endorsed 2005)

Hydrochloric acid is used to correct pH, regenerate deionisers and generate chlorine dioxide on site.

GENERAL DESCRIPTION

Hydrochloric acid, HCl, also known as spirits of salts, is a colourless or slightly yellow, fuming, pungent liquid. This strong and highly corrosive acid should be handled with extreme caution (particularly when adding the concentrated acid to water), as it can cause severe burns and eye damage. Hydrochloric acid is generally available as a 25–42% solution. A 28% solution has a specific gravity of 1.14 at 20°C. The acid is soluble in water and benzene, and is noncombustible.

Hydrochloric acid is highly corrosive to most metals or alloys, liberating extremely flammable hydrogen gas. Chlorine gas may also be liberated in reactions with oxidants or sodium hypochlorite. Hydrochloric acid may be stored and piped in rubber-lined carbon steel, fibreglass-reinforced plastic with acid-resistant resins, plastic liners and pipes (u-polyvinyl chloride, polythene and polypropylene).

CHEMISTRY

Hydrochloric acid is manufactured by the combustion of chlorine gas in hydrogen to produce hydrogen chloride gas, which is then dissolved in water.

Hydrochloric acid disassociates in water to produce a strong acid:

$$HCl \Leftrightarrow H^+ + Cl^-$$

To reduce fuming, the acid should be diluted (by adding acid to water) to about 20% HCl.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, hydrochloric acid is used to correct pH (for softening, corrosion control, coagulation, prevention of post-precipitation), regenerate deionisers and generate the disinfectant chlorine dioxide on site.

Doses of hydrochloric acid required vary widely, depending on the application and conditions.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA, KIWA 1994):

- arsenic
- chlorine
- chromium
- iron
- lead
- lead
- methylene chloride
- nickel
- sulfate
- sulfur dioxide

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

NOTE: Important general information is contained in PART II, Chapter 8

When employed in drinking water treatment, hydrochloric acid should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Hydrochloric acid was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

Clesceri *Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

NOTE: Important general information is contained in PART II, Chapter 8

Hydrofluorosilicic acid

(endorsed 2005)

Hydrofluorosilicic acid is used to artificially fluoridate water, to reduce the occurrence of dental caries. When dissolved in water, hydrofluorosilicic acid forms the fluoride ion^{-}.

GENERAL DESCRIPTION

Hydrofluorosilicic acid, H_2SiF_6 (also known as fluorosilicic acid, hexafluorosilicic acid), is a colourless to pale yellow liquid, poisonous and corrosive, with a pungent odour and irritating fumes. It can etch glass. It has a specific gravity of 1.18 at 20°C at 22% strength.

The acid is usually delivered by road tanker but can be supplied in drums. It is incompatible with glass and stoneware but can be stored in polythene drums, rubber-lined mild steel or polyvinyl chloride-lined plastic tanks.

CHEMISTRY

Hydrofluorosilicic acid is a by-product of the preparation of chemical fertilisers from phosphate rock. The rock is ground up and treated with sulfuric acid, forming a gas by-product, which then reacts with water to produce a weak acid. This hydrofluorosilicic acid solution is subsequently concentrated to strengths of up to 30%. Manufacture of hydrofluorosilicic acid is limited, but because the acid is a by-product of the agricultural industry, it is generally readily available in Australia.

The dissolution of hydrofluorosilicic acid in water forms the fluoride ion (F^-) as follows:

$$H_2SiF_6 \Leftrightarrow 2H^+ + SiF_6^{2-}$$

$$SiF_6^{2-} \Leftrightarrow SiF_4 + 2F^-$$

$$SiF_4 + 2H_2O \Leftrightarrow SiO_2 + 4F^- + 4H^+$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Hydrofluorosilicic acid is used to fluoridate drinking water, to reduce the occurrence of dental caries. In each State and Territory, except for South Australia, the fluoridation of drinking water is regulated by an Act of Parliament; New South Wales and Queensland also have regulations in force.

In adding hydrofluorosilicic acid to drinking water, it is good practice to add the chemical after the water has been treated, because fluoride ions may be adsorbed onto the surfaces of suspended matter in water. In water that has been treated and disinfected, fluoridation is usually accomplished with a 20% hydrofluorosilicic acid stock solution. The acid solution, despite its pH of 1.2, has little effect on the pH of highly alkaline water, because relatively low amounts are used. However, the pH effect can be significant with water of low alkalinity.

The target levels of fluoride in fluoridated water in Australia vary between 0.7 and 1.0 mg/L. The lower concentrations apply in warmer climates, where more water is consumed. For an acid solution of 20% strength (15.8% F^-), this range translates to a dose of hydrofluorosilicic acid of 4.4–6.3 mg/L.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

Chemical contaminants that may occur in hydrofluorosilicic acid solutions include inorganic and organic substances, and the following chemicals:

- arsenic
- lead

The concentrations of contaminants depend on the purity of the raw materials used in fertiliser production. Hydrofluorosilicic acid solutions also contain free hydrofluoric acid, which prevents the precipitation of solid silica when the acid is diluted in water.

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, hydrofluorosilicic acid should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Fluoride forms precipitates with many metals and other elements, but is notably insoluble with calcium; thus, scaling can occur when concentrated lime solution and concentrated fluoride solution come into contact. Points for adding these solutions should be separated, to avoid this situation.

STATUS

Hydrofluorosilicic acid was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B703-00. AWWA CD-ROM (April 2003). Details at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Department of Health, South Africa (2003). *Water fluoridation, a manual for water plant operators.* Available at <www.doh.gov.za/docs/misc/fluoridation/>

NSW Health (1957). Code of Practice for the fluoridation of public water supplies. *NSW Fluoridation of Water Supplies Act 1957*, NSW Government Gazette No. 135.

NOTE: Important general information is contained in PART II, Chapter 8

Hydrogen peroxide

(endorsed 2005)

Hydrogen peroxide is used as an oxidant in the treatment of drinking water (often in conjunction with ozone) to oxidise metals or organics, reduce tastes and odours, or act as an algicide, disinfectant or biocide. It can also be used to destroy ozone residual.

GENERAL DESCRIPTION

Hydrogen peroxide, H_2O_2, is a colourless syrupy liquid that is available in concentrations ranging from 20 to 60%, with a specific gravity between 1.07 and 1.24 at 20°C and pH 1–4.

There are strict handling and storage requirements that must be adhered to for hydrogen peroxide, which is especially dangerous at concentrations over 52%, because it is a strong oxidant and extremely corrosive. Materials suitable for handling and storing hydrogen peroxide include passivated aluminium or stainless steel (types 304L and 316L). Plastic piping (polyvinyl chloride or polyethylene) is only suitable for short-term use.

CHEMISTRY

Hydrogen peroxide is manufactured by electrolytic or organic auto-oxidation processes. A common example is the auto-oxidation of alkylated anthraquinones through hydrogenation with oxidation in the presence of a catalyst.

Used with ozone, hydrogen peroxide produces the powerful hydroxyl radical:

$$2H_2O_2 + 2O_3 = 4OH^{\bullet} + 3O_2$$

For the destruction of ozone in water, this reaction proceeds to water and oxygen.

Hydrogen sulfide, a common taste and odour compound, is oxidised to sulfate by hydrogen peroxide as follows, or to colloidal sulfur.

$$4H_2O_2 + H_2S \Leftrightarrow SO_4^{2-} + 4H_2O + 2H^+ \qquad \text{pH} > 8$$

$$H_2O_2 + H_2S \Leftrightarrow S + 2H_2O \qquad \text{pH } 7$$

Hydrogen peroxide also oxidises iron and manganese, which are then precipitated.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In water treatment, hydrogen peroxide is used with ozone to produce the hydroxyl radical, which is a powerful oxidant. The combination of hydrogen peroxide and ozone is used to:

- oxidise iron, manganese, sulfide and hazardous synthetic organic compounds such as trichloroethylene and atrazine
- remove taste and odour-causing substances, such as hydrogen sulfide (H_2S) which is commonly found in groundwater
- reduce colour and natural organic matter
- improve the performance of coagulants, or reduce the required amount of coagulants.

NOTE: Important general information is contained in PART II, Chapter 8

Hydrogen peroxide is a biocide, and can be used before treatment to control the growth of aquatic organisms such as algae in the pre-treatment basin. It may also be used as a primary disinfectant to meet the C.*t* (disinfectant concentration × contact time) requirements. Alternatively, hydrogen peroxide can be used after the ozonation stage to destroy ozone residual and minimise its release to the atmosphere.

Hydrogen peroxide is often added at the head of a treatment plant, before or at the rapid mix basin. However, it can also be added after clarification and before filtration, when a substantial portion of the oxidant demand has been removed.

To determine the optimum hydrogen peroxide concentration for a particular application, it is best to undertake pilot-plant and jar-testing trials. For use with ozone, the hydrogen peroxide to ozone ratio is typically 0.4–0.5; whereas, for destroying ozone residual, a concentration of 1.4 mg/L of H_2O_2 (50% strength) would be required for each mg/L of ozone.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in hydrogen peroxide (JECFA):

- acetanilide
- acetophenetidin
- arsenic
- copper
- iron
- sulfuric acid
- tin

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

Hydrogen peroxide decomposes to oxygen and water.

When employed in drinking water treatment, hydrogen peroxide should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Hydrogen peroxide was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

IARC (International Agency for Research on Cancer) (1999). *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man*. World Health Organization, Geneva, 71: 683.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Rueff J et al. (1993). DNA strand breaks and chromosomal aberrations induced by H_2O_2 and ^{60}Co gamma-radiation. *Mutation Research* 289 (2): 197–204.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.]

NOTE: Important general information is contained in PART II, Chapter 8

Hydroxylated ferric sulfate

(endorsed 2005)

Hydroxylated ferric sulfate is used as a coagulant for the treatment of drinking water. It is effective over a broad pH range and generally produces a stronger floc than other ferric salts.

GENERAL DESCRIPTION

Hydroxylated ferric sulfate (HFS), $Fe_x(SO_4)_y(OH)$, also known as polymerised ferric sulfate, is one of several hydroxylated iron coagulants produced from ferrous sulfate. It is a translucent, dark reddish liquid, with no odour. It is available in various ferric iron and basicity concentrations, but typically contains 12.5 % Fe.

HFS has a pH of less than 2 and a specific gravity of 1.45–1.6 at 25°C. It is corrosive, but can be stored in fibreglass, rubber-lined steel, stainless steel, polyethylene, polyvinyl chloride or polytetrafluoroethylene.

For commercial coagulant solutions, the basicity varies from about 5 to 15% for prehydrolysed iron salts. As the basicity exceeds 15%, it becomes increasingly difficult to keep the metal hydroxide precipitate from forming in the product solution during shipping and extended storage. The typical basicity for HFS is 10%.

CHEMISTRY

HFS is produced by oxidising ferrous sulfate. It can also be produced by dissolving ferrous oxide in sulfuric acid under controlled conditions. The chemical is similar to ferric sulfate, but its small polymeric chains provide additional coagulation properties, so it may be preferable to ferric sulfate for water that is difficult to treat.

In water, ferric (iron III) ion hydrolyses and precipitates, to an extent that depends on pH and dosage. Iron precipitates formed are goethite (HFeO2) and iron hydroxide ($Fe(OH)_3$), which are less soluble than aluminium precipitates. At equilibrium, the concentration of the soluble species is very low. The stoichiometry of the precipitation of iron hydroxide is described as follows:

$$Fe^{+3} + 3OH^- \rightarrow Fe\ (OH)_3\ (s)$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

HFS coagulates relatively quickly over a wide pH range (pH 4–11). It forms a dense floc and does not cause significant variation in pH. The floc produced is usually similar in characteristics to alum floc, but has less impact on pH and alkalinity. HFS generally produces a more robust floc than other iron salts. It is often preferred over conventional coagulants for low alkalinity waters containing colour because it does not consume as much alkalinity as other coagulants; thus, the need to add alkali in addition to coagulant is reduced.

The dose of HFS used depends on the properties of the raw water, including factors such as turbidity, natural organic matter, temperature and alkalinity. As with other coagulants, higher doses are required as turbidity and colour increase, and colder temperatures slow down reaction times.

Typical HFS doses (expressed as supplied HFS in mg/L) are 5–100 mg/L, although higher doses may be required if the water is particularly dirty. At high doses, product water should be tested to ensure that maximum contaminant levels have not been exceeded.

NOTE: Important general information is contained in PART II, Chapter 8

The dose rate of HFS may be expressed as ferric sulfate, supplied HFS liquid or iron. Care should be taken when interpreting dose rates to ensure that relevant comparisons are made. Because of its reactivity, HFS should be used neat if possible, or not pre-diluted such that is hydrolyses before contact with the water to be treated.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in HFS:

- antimony
- arsenic
- cadmium
- chromium
- cobalt
- copper
- cyanide
- lead
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- titanium
- vanadium
- zinc

In some products, manganese concentrations may be significant.

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, HFS should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Almost all ferric ions used for coagulation are removed by conventional water treatment processes. Residual sulfate is usually at low levels that do not adversely affect drinking water quality.

STATUS

HFS was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). Standard Methods for the Examination of Water and Wastewater, 20th edition. American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Ozone

(endorsed 2005)

Ozone is used as an oxidant and disinfectant in the treatment of drinking water. It can oxidise metals and organic compounds, including algal toxins, tastes and odours. Ozonation does not produce a residual so cannot be used to maintain a disinfection residual in the distribution system.

GENERAL DESCRIPTION

Ozone (O_3) is an unstable blue or colourless gas with a pungent odour. It can be liquefied at –12°C, and has a boiling point of –112°C and a freezing point of –192°C. It is more soluble in water than oxygen. As a strong oxidant and disinfectant, ozone effectively inactivates bacteria, viruses and protozoa (*Cryptosporidium* and *Giardia*), controls tastes and odours, and breaks down organic contaminants and algal toxins. Ozone also aids in coagulation and flocculation, by breaking down organic chains and starting microflocculation. Ozone does not produce halogenated disinfection by-products, except in bromide-rich waters where bromate ion is generated.

Disadvantages of ozone are that it is relatively costly and does not produce a persistent disinfectant residual (and therefore cannot be used to maintain a disinfection residual in the distribution system).

Also, ozone produces biodegradable organic material that increases biofouling problems in the water distribution system. This biodegradable material can be achieved by using biologically activated carbon (BAC) filters after ozone treatment.

Ozone in water is highly corrosive; therefore, only it can only be used with certain materials, such as 316 and 305 stainless steel, glass and Teflon. Ozone is produced on site using electrical discharges in the presence of oxygen. The maximum concentration of ozone generated is 50 g/m^3 and the maximum practical solubility of ozone in water is approximately 40 mg/L.

CHEMISTRY

Ozone is produced on site, as described above, and is highly unstable in the gaseous phase. Ozone has a half-life of 20–100 hours in clean vessels, at room temperature.

Two types of reactions occur when ozone is added to water:

- direct oxidation (a slow and extremely selective reaction favoured by low pH conditions)
- auto-decomposition to the hydroxyl radical (a reaction catalysed by hydroxyl radicals, organic radicals, hydrogen peroxide, ultraviolet light or high concentrations of hydroxide ion; and favoured by high pH conditions or high concentrations of organic matter).

Ozone breaks down more slowly in water that has a high concentration of bicarbonate or carbonate. Therefore, an ozone residual will last longer in highly buffered water with low pH.

The reaction of ozone with contaminants in the water requires a sufficient contact time and a high transfer efficiency coefficient, which can be provided by well-designed ozone contactors and mixing devices. The gas vented from the contactors contains ozone, which has to be destroyed or re-injected before the air is released to the atmosphere.

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL VALUES USED IN AUSTRALIAN DRINKING WATER TREATMENT

Ozone is a very strong oxidant that is moderately soluble in water. Typical concentrations used in drinking water are 0.5–5 mg (O_3)/L, depending on the organic content of the water. The required dose should be determined through bench-scale ozone demand tests or pilot-plant testing, using available C.*t* (concentration × contact time) data for the inactivation of various microorganisms. The contact time required for ozone inactivation of microorganisms varies from seconds to minutes (the longer time being required for inactivation of protozoan cysts) and is temperature dependent; it is significantly shorter than the contact time required for chlorine or chloramines.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process and impurities in the air or oxygen used to generate the ozone. The following chemicals may be present in ozone:

- acetylene
- argon
- carbon dioxide
- carbon monoxide
- hydrocarbons
- nitrous oxide

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, ozone should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Ozone can react with bromide to form brominated ozone, which includes bromate ion (BrO_3^-). If natural organic material is present, nonhalogenated organic disinfection by-products are formed. These include aldehydes (formaldehyde being dominant), ketoacids and carboxylic acids. If both natural organic material and bromide are present, hypobromous acid is formed, together with brominated organohalogen compounds.

STATUS

Ozone was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

APHA (1998). APHA 4500-O3, Ozone (residual), in Standard Methods for the Examination of Water and Wastewater, 20th Edition, American Public Health Association, Washington, DC.

Water Treatment Plant Design (1990). American Water Works Association, 3rd Ed. McGraw-Hill Companies, Inc.

NOTE: Important general information is contained in PART II, Chapter 8

Polyacrylamide

(endorsed 2005)

Polyacrylamide is used in water treatment as an aid to coagulation, flocculation, clarification, filtration or handling of sludge.

GENERAL DESCRIPTION

Polyacrylamide, $(CH_2CHCONH_2)_n$, is a white crystalline solid. It is hydrophilic, with molecular weights of 1–30 million daltons, and chain lengths of 1.4×10^4 to 4.2×10^5 monomer units. Polyacrylamide is available in anionic, cationic or non-ionic forms, and in a variety of molecular weights and charge densities, to suit the particular characteristics of the water to be treated. It may be supplied as a powder, as an aqueous solution, dispersed in a light mineral oil or bound up in a solid cake that slowly dissolves when immersed.

Appropriate handling materials for polyacrylamide include fibreglass-reinforced plastic, polyethylene, polypropylene, polyvinyl chloride, stainless steel and coated steel.

CHEMISTRY

Polyacrylamide is usually manufactured by the polymerisation of the acrylamide monomer (AM) to form a non-ionic polymer, polymerisation of AM with acrylic acid salts to form an anionic polymer, or polymerisation of AM with cationic monomer to form a cationic polymer.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, polyacrylamide may be added:

- as a coagulation aid, immediately after coagulation, to strengthen the precipitate formed
- as a flocculation aid, at the start of flocculation, to increase the agglomeration of the floc
- as a clarification aid, before clarification, to help settle floc, bind dissolved air bubbles to floc (in dissolved air flotation, DAF) or bind floc to microsand
- as a filtration aid, before filtration, to minimise floc shearing and to improve adsorption of floc onto the filter medium
- to backwash water, to minimise filter ripening periods
- to sludge for thickening or dewatering, to improve performance.

As a coagulation, flocculation or clarification aid, polyacrylamide is typically used at concentrations of 0.05–0.3 mg/L. As a filter aid, it is usually applied in lower doses (0.01–0.1 mg/L). For sludge handling, typical doses of polyacrylamide are 0.5–2 kg per tonne of dry solids for thickening, and 1–4 kg for dewatering.

High doses of polyacrylamide can cause clogging and blockages, particularly in filter beds. Therefore, where high doses of polymers are used in water treatment, it is best to clean filters by both air scouring and water washing. Even with relatively low doses of polyacrylamide, filter beds should be inspected regularly for signs of polymer build-up. Regular measurement of the headloss accumulation rate in a filter is also useful.

Care should be taken in making up polymer solutions to minimise the formation of lumps of undissolved polymer (referred to as 'fish eyes'). The polymer should be mixed with the water using a well-designed eductor, so that each grain of polymer is separately introduced to the water.

NOTE: Important general information is contained in PART II, Chapter 8

The polymer solution should also be suitably aged before dosing to obtain best performance. Aging requires gentle mixing of the polymer solution for 1–4 hours (refer to manufacturer for specific polymer aging times).

While most polymers are at least chlorine resistant, making up polymer solutions with chlorinated water can reduce their effectiveness.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product, depending on the raw materials used:

- acetamide
- acetone
- acrylamide
- acrylic acid
- acrylonitrile
- copper
- hydroquinone
- methacrylamide
- methyl ether hydroquinone
- peroxide
- propanamide
- sulfate

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

Polyacrylamides contain varying residual amounts of unreacted acrylamide monomer.

When employed in drinking water treatment, polyacrylamide should be used in such a way that any contaminants or by-products formed by the use of the chemical do not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Polyacrylamide, acrylic acid polymers and copolymers were endorsed by the NHMRC as drinking water treatment chemicals in 1977 and 1979. The revision undertaken in 2003 did not result in any change to the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B453-01. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Brown L and Rhead MM (1979). Liquid chromatographic determination of acrylamide monomer in natural and polluted aqueous environments. *Analyst* 104:391–399.

Letterman RD and Pero (1990). Contaminants in Polyelectrolyets used in Water Treatment. *American Water Works Association* 82(11): 87–97.

NICNAS (National Industrial Chemicals Notification and Assessment Scheme) (2002). *Priority Existing Chemical Assessment No 23: Acrylamide*, NICNAS, Canberra.

NOTE: Important general information is contained in PART II, Chapter 8

Polyaluminium chloride

(endorsed 2005)

Polyaluminium chloride is used as a primary coagulant in the treatment of drinking water. It is effective over a range of pH values. It is particularly effective on some waters and usually requires a lower dose than alum.

GENERAL DESCRIPTION

Polyaluminium chloride (PACl), $Al_2(OH)_3Cl_3$, is also known as aluminium hydroxy chloride or basic aluminium chloride. In solution, PACl is colourless to pale yellow, clear to slightly cloudy liquid. It is usually supplied with a minimum of 10% Al_2O_3 content, a pH of 2.2–2.8 and a basicity of about 50% (w/w). PACl solution has a specific gravity of 1.18–1.22 at 20°C and is completely soluble in water. Its use requires less alkalinity adjustment than most coagulants because of its basicity.

The formula $Al_2(OH)_3Cl_3$, is simply a representation of the proportions of aluminium, hydroxide and chloride in the solution. A generic formula for the PACl species may be given as $Al_2(OH)_mCl_{(6-m)}$ where the value of m typically ranges from 2.5 to 3.5.

PACl can be stored in fibreglass or plastics (polyethylene, polypropylene or polyfluorene), but is corrosive to most materials, including stainless steel (although 316 stainless steel can be used).

CHEMISTRY

PACl is manufactured by the reaction of hydrochloric acid with aluminium-containing raw materials such as aluminium metal, alumina trihydrate, aluminium chloride or aluminium sulfate.

PACl solution is a complex, dynamic mixture of positively charged polynuclear aluminium species, with no single species predominating. When applied to water, these species interact with and destablise negatively charged colloidal matter, such as inorganic particles and the high molecular weight organic compounds that largely constitute natural organic matter. The polynuclear species also hydrolyse to form dense flocs of aluminium hydroxides that further act to entrap particles and remove some organic.

An example of one of the many polynuclear species that may be present in PACl solution is the so called Al-13 ion that has the formula $[AlO_4.Al_{12}(OH)_{24}(H_2O)_{12}]^{13+}$.

The hydrolysis of PACl produces less acid than the hydrolysis of aluminium sulfate owing to the high degree of hydroxylation of the aluminium. As a result, PACl generally requires less pH correction with alkali than if alum were the coagulant.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

PACl is used as a primary coagulant to reduce turbidity, metals, colour and natural organic matter.

The amount of PACl added as a coagulant depends on the properties of the raw water, including factors such as turbidity, dissolved organic carbon, temperature and alkalinity.

Typical PACl doses (with 10% Al_2O_3 content) are 5–100 mg/L, although higher doses can be required if the water is particularly dirty. Doses should be determined by laboratory trials.

PACl is the next most commonly used aluminium salt after alum. Compared to alum, it produces a relatively robust floc, generally requires lower doses and is effective over a wider pH range.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

PACl solution is usually low in trace metals, because it is made from clean raw materials. However, the following chemical contaminants may be present in this product:

- antimony
- arsenic
- barium
- beryllium
- cadmium
- chromium
- copper
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- thallium
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, PACl should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Most of the aluminium ions resulting from the use of PACl as a coagulant are removed by conventional water treatment processes. Residual chloride will be present, but at low levels that do not adversely affect drinking water quality.

STATUS

Polyaluminum chloride was endorsed by the NHMRC for use as a drinking water treatment chemical in 1979. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B408-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th Edition. American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Polyaluminium silica sulfates

(endorsed 2005)

Polyaluminium silica sulfates are a relatively new group of coagulants in the treatment of drinking water. They are effective for removal of metals, colour and turbidity, and readily forms floc even in clean water.

GENERAL DESCRIPTION

Polyaluminium silicate sulfate, $Al_A(OH)_B(SO_4)_C(SiO_x)_D \cdot E(H_2O)$ (also known as aluminium hydroxide silicate sulfate) are pale yellow in colour and appears slightly cloudy to clear. It is usually supplied with a minimum of 9.8% Al_2O_3, a basicity of about 54% and a specific gravity of 1.32–1.36 (at 25°C). It has a pH of 2.8–3.6. It can be stored in fibreglass, plastics and stainless steel.

CHEMISTRY

Polyaluminium silicate sulfate is manufactured from alum, soda ash, sodium silicate and sodium aluminate.

Polyaluminium silicate sulfate solution is a polymerised coagulant solution containing aluminium in short chains. The high basicity of polyaluminium silicate sulfate assists in flocculation, because the coagulant does not require alkalinity to form the initial floc. The charge on colloidal particles and dissolved organics is neutralised by adsorption onto the very small flocs that form initially. Silicate compounds in polyaluminium silica sulfate help to form larger flocs faster than with many other coagulants.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Polyaluminium silicate sulfate is used as a coagulant in the treatment of water and wastewater and to assist sludge blanket formation at start up. Polyaluminium silicate sulfate forms floc rapidly, even in cold water. It tends to form floc even with clean dilution water; therefore, it should be added as supplied (i.e. undiluted).

Typical concentrations of polyaluminium silicate sulfate used in drinking water treatment depend on the quality of the water to be treated and the purpose of the treatment. Polyaluminium silicate sulfate doses are typically 5–100 mg/L, but may be higher if the water is particularly dirty. The appropriate concentration should be determined by laboratory trials. Polyaluminium silicate sulfate must be used undiluted in jar tests.

CONTAMINANTS

The following contaminants may be present depending on the manufacturing process:

- antimony
- arsenic
- barium
- beryllium
- cadmium
- chromium
- copper
- fluoride
- iron
- lead
- magnesium
- manganese
- mercury
- nickel
- phosphorus
- selenium
- silver
- thallium
- zinc

NOTE: Important general information is contained in PART II, Chapter 8

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, polyaluminium silicate sulfate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Polyaluminium silicate sulfate was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Clifford DA (1999). Ion Exchange and Inorganic Adsorption. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 9.1–9.91.

Lewis RJ Sr (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

McGregor S (2002) Pass for P.A.S.S. on OHS and treatment. WaterWorks, December pp 12-15.

NOTE: Important general information is contained in PART II, Chapter 8

Polydiallyldimethylammonium chloride

(endorsed 2005)

Polydiallyldimethylammonium chloride (polyDADMAC) is used in the treatment of drinking water as a primary coagulant or, together with an inorganic coagulant, as a coagulation aid. PolyDADMAC reduces the quantities of floc and sludge produced.

GENERAL DESCRIPTION

Polydiallyldimethylammonium chloride $(C_8H_{16}N{\cdot}Cl)_n$, (also known as polyDADMAC), is a cationic polyelectrolyte with a medium molecular weight range of 105–106 and a high charge density (50–100%). The chemical is available as a powder or aqueous solution (10–60%). PolyDADMAC is not pH sensitive and is chlorine resistant.

Appropriate handling materials for polyDADMAC include fibreglass-reinforced plastic, polyethylene, polypropylene, polyvinyl chloride, stainless steel and coated steel.

CHEMISTRY

PolyDADMAC is produced from the diallyldimethylammonium chloride (DADMAC) monomer, which is made from allyl chloride and dimethylamine.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

PolyDADMAC can be used in a conventional treatment process as a primary coagulant for neutralisation and precipitation, in place of metal salts. The positively charged polyDADMAC reacts with turbidity particles and humic substances, which are generally negatively charged. The reaction eliminates the charge, allowing the particles to agglomerate. PolyDADMAC is usually most effective with particulate material; it may be less useful than aluminium and iron salts for treating dilute inorganic suspensions and water with significant amounts of colour.

PolyDADMAC can also be used as a secondary coagulant, to partially replace inorganic salts. A small dose of polyDADMAC may significantly reduce the amount of inorganic salt required (thus reducing floc volume and improving filter run times); often, it also improves treated water quality. PolyDADMAC is used particularly in direct and contact filtration processes, where the objective of coagulation is to produce small, high-density aggregates.

In treatment of drinking water, typical concentrations of polyDADMAC are 0.2–6 mg/L (as 100% polyDADMAC). When polyDADMAC is used, together with an inorganic salt, as a secondary coagulant, concentrations are usually lower (0.2–1 mg/L). The amount of polyDADMAC required should be determined through jar testing. The chemical can be added at concentrations of up to 10 mg/L, provided that the residual concentration of the monomer (DADMAC) does not exceed 2% of the polymer, and that the concentration of the residual monomer does not exceed 0.2 mg/L in the clarified water.

At concentrations above 40%, polyDADMAC is difficult to pump, because of its relatively high viscosity. Excessive polymer concentrations can adversely affect coagulation and filtration by re-dispersing the impurities.

NOTE: Important general information is contained in PART II, Chapter 8

Being highly charged, polyDADMAC should be diluted before it is added to the main water stream, so that it mixes more easily.

PolyDADMAC is usually supplied as a liquid. If supplied as a solid, individuals should seek advice from the supplier of the polymer as how to best prepare it.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product:

- 5-hexenal
- allyl chloride
- DAD monomers
- beryllium
- cadmium
- chromium
- copper
- fluoride
- iron
- lead
- diallyl ether
- dimethylamine
- mercury
- nickel
- phosphorus
- selenium
- silver
- thallium
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, polyDADMAC should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Diallyldimethylammonium chloride residues are present in polyDADMAC.

STATUS

PolyDADMAC was endorsed by the NHMRC for use as a drinking water treatment chemical in 1982. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

NSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B451-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Bolto, B. (August, 1994). Polymeric Flocculants in Water Purification. *Water Chemistry Supplement* in *Water Journal* 21(4): 431-433.

Letterman RD and Pero (1990). Contaminants in Polyelectrolytes used in Water Treatment. *American Water Works Association* 82(11): 87–97.

NOTE: Important general information is contained in PART II, Chapter 8

Potassium permanganate

(endorsed 2005)

Potassium permanganate is mainly used for the oxidation and removal of iron and manganese; it can also be used as a disinfectant, or to control tastes and odours.

GENERAL DESCRIPTION

Potassium permanganate, $KMnO_4$, is a dark purple crystal with a blue metallic sheen. It has a sweetish, astringent taste, is odourless and is an oxidant. The chemical is commercially available in crystalline form. Potassium permanganate is highly soluble in water, but heating is usually needed to prepare solutions with concentrations of more than 2.5%.

Appropriate handling materials include iron, steel, stainless steel, fibreglass-reinforced plastic, polyethylene and polyvinyl chloride.

CHEMISTRY

Potassium permanganate is produced by fusing manganese dioxide with potassium hydroxide to form potassium manganate; a solution of the manganate is then electrolysed at about 60°C using iron electrodes.

Under most treatment applications, permanganate (MnO_4^-) is reduced to insoluble manganese dioxide (MnO_2 (s)).

Divalent manganese (Mn^{2+}) is removed from water by the oxidation to insoluble manganese dioxide (MnO_2). As the oxidant, the permanganate ion MnO_4^- is itself reduced to manganese dioxide. The reaction proceeds as follows:

$$3Mn^{2+} + 2MnO_4^- + 4OH \rightarrow 5MnO_2 + 2H_2O$$

The stoichiometric ratio of $KMnO_4$ to soluble Mn^{2+} is 1.92:1; however, reactions with organics usually require significantly higher ratios. The alkalinity consumed is 1.2 mg of CaCO3 per milligram of Mn^{2+}, and the sludge produced (based on MnO_2 as the precipitate) is 2.6 kg/kg Mn^{2+}.

Potassium permanganate can also be used to oxidise iron and organics. The stoichiometric ratio of $KMnO_4$ to soluble Fe^{2+} is 0.94:1; however, reactions with organics usually require higher ratios. The alkalinity consumed is 1.5 mg per mg of Fe^{2+} and the sludge produced (based on $Fe(OH)_3$ as the precipitate) is 2.4 kg/kg Mn^{2+}.

Manganese dioxide resulting from permanganate reduction is an effective adsorbent for ferrous iron (Fe^{2+}), manganous manganese (Mn^{2+}), radium (Ra^{2+}) and other trace inorganic cationic species. These contaminants can be removed by permanganate treatment.

Manganese dioxide also adsorbs natural organic materials that serve as precursors for disinfection by-products. This characteristic of manganese dioxide is particularly pronounced in hard waters, presumably because of the bridging action of calcium and manganese.

NOTE: Important general information is contained in PART II, Chapter 8

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, potassium permanganate can be fed into solution directly using a dry chemical feeder or as a liquid bulk supply using dosing pumps. Alternatively, a concentrated solution can be prepared on site, from which the desired concentration is added to the water.

Permanganate is often added at the head of the treatment plant, as close to the intake as possible. This allows sufficient time for the permanganate to perform its oxidative function and to be reduced completely to solid manganese dioxide before filtration. In some cases, an alkali (usually lime) is added before, or soon after, addition of potassium permanganate, to assist in the oxidation process. Potassium permanganate can be effective over a range of pH values, but is most effective at pH 8.5 or higher.

The adsorptive property of $MnO_2(s)$ is the principle underlying the historical manganese greensand process, in which the filter medium is coated with manganese dioxide, which subsequently serves as an adsorbent for Fe^{2+}, Mn^{2+} and other metals in the filter influent. Filter media can be coated with manganese oxide by applying a potassium permanganate solution to the filter bed and oxidising it (through chlorination or aeration). Low doses of chlorine or permanganate are applied to the filter influent to catalyse the oxidation of the adsorbed metals, thereby creating additional adsorption sites. Alternatively, the filter backwash water may be treated with chlorine or permanganate.

Concentrations of potassium permanganate used in drinking water treatment depend on the concentrations of metals and organics, but are usually 0.3–5 mg/L. Overdosing of the chemical should be avoided because of the pink colour of unreacted permanganate. The potassium permanganate levels required should be determined by jar testing.

Advantages of potassium permanganate include its ease of use and the fact that it is effective for the oxidation of both iron and manganese and for certain types of taste and odour. As a disinfectant, it produces no halogenated disinfection by-products, but has only a limited disinfection capability.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (NRC 1982):

- cadmium
- chloride
- chromium
- mercury
- sulfate

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, potassium permanganate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

The manganese dioxide produced is a black precipitate that can be removed by a conventional clarification or filtration process. If manganese dioxide is not properly removed, the precipitates will create particulate deposits in the distribution system and on household plumbing fixtures. At manganese concentrations above 0.02 mg/L, an increase in consumer complaints is common.

STATUS

Potassium permanganate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

NOTE: Important general information is contained in PART II, Chapter 8

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B603-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium aluminate

(endorsed 2005)

Sodium aluminate is used as a primary coagulant in drinking water treatment, especially in water with low alkalinity; it can also be used in combination with alum to control alkalinity and pH.

GENERAL DESCRIPTION

Sodium aluminate, $Na_2Al_2O_4$, is a white powder that is hygroscopic, soluble in water and strongly alkaline. The aqueous solution is a clear, colourless to pale amber liquid, with a pH of 14.

Sodium aluminate can be supplied as a powder or as a solution. The solid product contains 70–90% $Na_2Al_2O_4$, whereas the liquid form contains 29–35% $Na_2Al_2O_4$. The liquid solution has a specific gravity of 1.4–1.6, with an excess alkali (as sodium hydroxide, NaOH) of 8–13% and Al_2O_3 equivalent of 18–21%.

Appropriate handling materials for sodium aluminate include iron, fibreglass-reinforced plastic, polyethylene, rubber, steel, stainless steel and concrete.

CHEMISTRY

Sodium aluminate is produced by combining aluminium oxide with excess caustic soda.

The aluminium ion neutralises the negative charges on turbidity particles and also forms insoluble metal hydroxides that agglomerate the neutralised particles:

$$Na_2Al_2O_4 + 4H_2O \rightarrow 2Na^+ + 2Al^{+3} + 8OH^-$$

$$Al_2O_3 + 3H_2O \Leftrightarrow 2Al(OH)_3(s)$$

1 mg/L of $Na_2Al_2O_4$ (88%) increases the alkalinity of the water by 0.54 mg/L and reduces carbon dioxide, CO_2, by 0.47 mg/L.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sodium aluminate is used as a primary coagulant, especially in water with low alkalinity. It can also be used in combination with alum to control alkalinity and pH. An advantage of sodium aluminate is that the chemical provides both aluminium and alkali. However, its use as a coagulant in water treatment is limited by cost and by its chemical properties, which make it more difficult to handle than alum or other metal salts.

Because sodium aluminate contains a high percentage of aluminium, a concentration of 1 mg/L of $Na_2Al_2O_4$ is equivalent to 3.5 mg/L of alum (on a dry weight basis).

Typical concentrations used are 2–60 mg/L (as $Na_2Al_2O_4$). The appropriate level should be determined by jar testing.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product:

- arsenic
- cadmium
- chromium
- iron
- lead
- mercury
- selenium
- silver

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium aluminate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Aluminium residuals remaining after filtration can cause floc to form in the distribution system, which can lead to customer complaints.

STATUS

Sodium aluminate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

NSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B405-00. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium bicarbonate

(endorsed 2005)

Sodium bicarbonate is used to correct pH, control corrosion, soften water for coagulation and prevent post-precipitation.

GENERAL DESCRIPTION

Sodium bicarbonate, $NaHCO_3$ (also known as baking soda, bicarbonate of soda, sodium acid carbonate or sodium hydrogen carbonate), is in the form of a white powder or crystalline lumps, and has a slightly alkaline taste. It is soluble in water (96 g/L at 20°C) and stable in dry air, but slowly decomposes in moist air. Its specific gravity is 2.159 at 20°C, with a bulk density of 1000 kg/m^3. Sodium bicarbonate is available in several grades, but is usually supplied as > 99% sodium bicarbonate. A 10 g/L solution has a pH of 8.4. The chemical decomposes with heat (> 50°C) and reacts with acid to release carbon dioxide.

Suitable storage materials for sodium bicarbonate include rubber linings and stainless steel.

CHEMISTRY

Sodium bicarbonate is most economically produced by bubbling carbon dioxide gas through a solution of purified sodium carbonate; the bicarbonate precipitates out and can be collected and dried. Sodium bicarbonate is also an intermediate product in the Solvay process for making sodium carbonate.

Sodium bicarbonate provides bicarbonate alkalinity without significantly changing the pH of the water:

$$NaHCO_3 \Leftrightarrow Na^+ + HCO_3^-$$

It can further break down to carbon dioxide in the presence of acid:

$$HCO_3^- + H^+ \Leftrightarrow CO_2 + H_2O$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sodium bicarbonate is used to correct pH, control corrosion, soften water for coagulation and prevent post-precipitation. It is used as a source of alkalinity for the treatment of waters with low alkalinity, but is more expensive than soda ash or lime. When it is used to improve coagulation, additional alkalinity or pH adjustment is often required.

The concentration of sodium bicarbonate required depends on the alkalinity and pH of the raw water and the targets for the treated water. Jar testing should be used to determine requirements.

Sodium bicarbonate can increase alkalinity with little increase in pH. It imparts a change of 0.60 g/L $CaCO_3$ alkalinity per mg/L as $NaHCO_3$.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water will vary depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA):

- ammonium
- arsenic
- chloride
- iron

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

NOTE: Important general information is contained in PART II, Chapter 8

When employed in drinking water treatment, sodium bicarbonate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Sodium, alkalinity, carbonate and carbon dioxide are the only significant residues that are expected to occur from sodium bicarbonate, but none of these is likely to become a problem at normal doses.

STATUS

Sodium bicarbonate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Singer PC and Reckhow DA (1999). Chemical oxidation. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 12.1–12.51.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium carbonate

(endorsed 2005)

Sodium carbonate is used to correct pH, control corrosion, soften water for coagulation and prevent post-precipitation.

GENERAL DESCRIPTION

Sodium carbonate, Na_2CO_3 (also known as soda ash), is a hygroscopic, greyish-white powder. It is supplied in the form of crystalline granules containing more than 99% sodium carbonate. It is soluble in water (to 250 g/L) and noncombustible. The chemical is available in different grades. Dense soda ash (specific gravity 2.15, bulk density 1000 kg/m^3) is most commonly employed in the water industry, but light soda ash (specific gravity 2.53, bulk density 500 kg/m^3) may also be used. Liquid soda ash is also available as a solution of various concentrations. Liquid soda ash is typically supplied as a 10 % w/v solution that has a specific gravity of 1.1(25°C) and a pH of up to 12.5. A 1% solution has a pH of 11.3.

Appropriate materials for handling sodium carbonate include rubber linings, iron, steel, fibreglass reinforced plastic and polyethylene.

CHEMISTRY

Sodium carbonate is found in natural deposits and is mined. It is also recovered, with other chemicals, from lake brines. However, most is produced through the Solvay process, in which ammonia and carbon dioxide are passed into a saturated sodium chloride solution, forming first ammonium hydrogen carbonate, then soluble ammonium chloride and a precipitate of sodium hydrogen carbonate (sodium bicarbonate). The precipitate is filtered off and heated to produce sodium carbonate.

Sodium carbonate produces hydroxide and bicarbonate ions in water:

$$Na_2CO_3 + H_2O \Leftrightarrow 2Na^+ + HCO_3^- + OH^-$$

Sodium carbonate is used together with lime to remove noncarbonate hardness (that portion of calcium and magnesium present as noncarbonate salts) as shown below:

$$MgSO_4 + Ca(OH)_2 \Leftrightarrow Mg(OH)_2 + CaSO_4$$

$$CaSO_4 + Na_2CO3 \Leftrightarrow CaCO_3 + Na_2SO_4$$

The solubility of magnesium hydroxide varies with pH. A pH of 11–11.3 is usually needed to remove magnesium effectively; this will require a concentration of lime higher than the stoichiometric requirement.

The quantity of sodium carbonate needed to remove noncarbonate hardness can be estimated using the following equation:

$$Na_2(CO)_3 \text{ (mg/L)} = 1.05 \text{ x (noncarbonate hardness removed (mg/L))}$$

Noncarbonate hardness is expressed as $CaCO_3$.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sodium carbonate is used mainly as a source of alkalinity and pH adjustment. It is more expensive than lime but is generally easier to handle, because of its higher solubility. If hard water is used for making up or diluting a solution of sodium carbonate, calcium carbonate may precipitate. This reduces the strength of the solution and can produce scale in the delivery pipelines. In this situation, the service water supplied to the soda ash system needs to be softened.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium carbonate is usually made up as a solution of up to 20% concentration. Concentrations of sodium carbonate used in drinking water treatment depend on the quality of the water to be treated and the purpose of the treatment (water softening, pH adjustment or alkalinity increase). Based on stoichiometry, 1 mg/L of sodium carbonate provides alkalinity equivalent to about 0.7 mg/L of hydrated lime. Typical sodium carbonate concentrations used can vary from 5 to more than 500 mg/L, and the appropriate concentration should be determined by laboratory trials.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in sodium carbonate (JECFA, KIWA 1994, NRC 1982):

- arsenic
- cadmium
- calcium
- chloride
- chromium
- iron
- lead
- magnesium
- mercury
- nickel
- sulfate

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium carbonate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*. Sodium residue derived from using sodium carbonate in water softening is 30–300 mg/L.

STATUS

Sodium carbonate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B201-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Benefi eld LD and Morgan JM (1999) Chemical Precipitation. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, 10.1–10.60.

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater,* 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary,* 12th edition. Van Nostrand Reinhold, New York.

NRC (National Research Council) (1982). *Water Chemicals Codex*. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium fluoride

(endorsed 2005)

Sodium fluoride is used to artificially fluoridate water, to reduce the occurrence of dental caries. Use of sodium fluoride is more common in small fluoridation facilities.

GENERAL DESCRIPTION

Sodium fluoride, NaF, is a white, odourless powder (or crystals), supplied in 25 kg bags. It is easily soluble in water, and the solubility varies little with temperature. It has a specific gravity of 2.78 at 20°C. The typical commercial grade of sodium fluoride is 97% purity, with about 44% fluorine. It has a bulk density of 1040–1440 kg/m^3. The pH of a 1% solution is 6.5; that of a 4% solution is 7.6. Suitable materials for handling sodium fluoride include iron, steel, fibreglass-reinforced plastic and polyethylene.

CHEMISTRY

Sodium fluoride is produced by neutralising hydrofluoric acid with either sodium carbonate or sodium hydroxide.

The dissolution of sodium fluoride in water forms fluoride ions (F^-) and sodium ions (Na^+) as follows:

$$NaF \Leftrightarrow Na^+ + F^-$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Sodium fluoride is used to artificially fluoridate water, to reduce the occurrence of dental caries. In each State and Territory, except for South Australia, the fluoridation of drinking water is regulated by an Act of Parliament; New South Wales and Queensland also have regulations in force.

Sodium fluoride can be used in solution feed systems at a strength of 1–2%, or in a saturator system where water is passed through a bed of sodium fluoride crystals, thus producing a saturated solution. The water used for dissolving sodium fluoride should not have a hardness greater than 75 mg/L (as calcium carbonate, $CaCO_3$), because the presence of calcium and magnesium causes the formation of insoluble fluorides which may cause clogging problems.

When using sodium fluoride, it is good practice to add the chemical after drinking water has been treated, because fluoride ions may be adsorbed onto the surfaces of suspended matter in water.

The target levels of fluoride in fluoridated water in Australia vary between 0.7 and 1.0 mg/L. The lower concentrations apply in warmer climates, where more water is consumed.

For sodium fluoride of 97% strength (44% F^-), this range translates to a dose of sodium fluoride of 1.6–2.3 mg/L.

CONTAMINANTS

Sodium fluoride can contain traces of free acid or alkali, and also:

- arsenic
- lead
- silicate
- sulfate

NOTE: Important general information is contained in PART II, Chapter 8

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium fluoride should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Fluoride forms precipitates with many metals and other elements, but is notably insoluble with calcium; thus, scaling can occur when concentrated lime solution and concentrated fluoride solution come into contact. Locations for adding concentrated lime and fluoride solutions should be separated, to avoid this situation.

STATUS

Sodium fluoride was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B701-99. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Department of Health, South Africa (2003). *Water fluoridation, A manual for water plant operators*. Available at <www.doh.gov.za/docs/misc/fluoridation/>

NSW Health (1957). Code of Practice for the fluoridation of public water supplies. *NSW Fluoridation of Water Supplies Act 1957*, NSW Government Gazette No. 135.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium fluorosilicate

(endorsed 2005)

Sodium fluorosilicate, Na_2SiF_6, is used to artificially fluoridate water, to reduce the occurrence of dental caries.

GENERAL DESCRIPTION

Sodium fluorosilicate (Na_2SiF_6, also known as sodium silicofluoride, sodium hexafluorosilicate and disodium hexafluorosilicate) is a white or yellowish white, odourless, crystalline powder with a specific gravity of 2.7. Sodium fluorosilicate has very low solubility in water. The chemical is usually supplied at 98.5% purity (59.5% F^-) in 25 kg bags. It has a bulk density of 880–1150 kg/m^3. Suitable handling material includes cast iron, rubber linings, steel and stainless steel, fibreglass-reinforced plastic, polyethylene and polyvinyl chloride.

CHEMISTRY

Sodium fluorosilicate is produced by neutralising hydrofluorosilicic acid with sodium carbonate or sodium hydroxide, and then evaporating the solution.

The dissolution of sodium fluorosilicate in water forms the fluoride ion (F^-), as follows:

$$Na_2SiF_6 \Leftrightarrow 2Na^+ + SiF_6^{2-}$$

$$SiF_6^{2-} \Leftrightarrow SiF_4 + 2F^-$$

$$SiF_4 + 2H_2O \Leftrightarrow SiO_2 + 4F^- + 4H^+$$

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

Sodium fluorosilicate is used to fluoridate drinking water, to reduce the occurrence of dental caries. In each State and Territory, except for South Australia, the fluoridation of drinking water is regulated by an Act of Parliament; New South Wales and Queensland also have regulations in force.

It is good practice to add sodium fluorosilicate after drinking water has been treated, because fluoride ions may be adsorbed onto the surfaces of suspended matter in water. In water that has been treated and disinfected, sodium fluorosilicate is usually added at a concentration of 0.2%. A good mixing system is required because sodium fluorosilicate has low solubility in water.

The targeted levels of fluoride in fluoridated water in Australia vary between 0.7 and 1.0 mg/L. The lower concentrations apply in warmer climates, where more water is consumed. For sodium fluorosilicate of 98.5% strength (59.5% F^-), this range translates to a dose of sodium fluorosilicate of 1.2–1.7 mg/L.

CONTAMINANTS

Sodium fluorosilicate may contain traces of free acid and moisture, and also:

- arsenic
- cadmium
- iron
- phosphorus

NOTE: Important general information is contained in PART II, Chapter 8

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium fluorosilicate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Fluoride forms precipitates with many metals and other elements, but is notably insoluble with calcium; thus, scaling can occur when concentrated lime solution and concentrated fluoride solution come into contact. Points for adding concentrated lime and fluoride solutions should be separated, to avoid this situation.

STATUS

Sodium fluorosilicate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B702-99. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Department of Health, South Africa (2003). *Water fluoridation, A manual for water plant operators*. Available at <www.doh.gov.za/docs/misc/fluoridation/>

NSW Health (1957). Code of Practice for the fluoridation of public water supplies. *NSW Fluoridation of Water Supplies Act 1957*, NSW Government Gazette No. 135.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium hexametaphosphate

(endorsed 2005)

Sodium hexametaphosphate can be used for control of corrosion, prevention of scale formation, and sequestration of unwanted precipitants.

GENERAL DESCRIPTION

Sodium hexametaphosphate, $Na(PO_3)_6$ (also known as SHMP, glassy phosphate or vitreous phosphate) is a white granular powder with a bulk density of 800–1500 kg/m^3. It is highly soluble in water.

Sodium hexametaphosphate can be stored in rubber-lined containers, or in plastics, fibreglass-reinforced plastic, or stainless steel (type 316).

CHEMISTRY

Sodium hexametaphosphate is produced by treating soda ash or caustic soda with phosphoric acid.

Polyphosphates keep metal ions in solution for a period of time, thus preventing deposition.

With time, sodium hexametaphosphate naturally reverts to orthophosphate, and thus loses its sequestering capability. This reversion can be accelerated by low pH, high temperature and the presence of oxides of certain materials (e.g. iron, calcium, copper and zinc). The reversion can occur in hot water systems or in reverse osmosis (RO) membranes, where it can cause fouling.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In water treatment plants, a thin layer of sodium hexametaphosphate formed on metal surfaces is used to control corrosion. The chemical is also used as a sequestering agent, to prevent unwanted precipitates or scales (e.g. iron, manganese, calcium or magnesium) from depositing.

Control of ferrous iron through sequestering is only effective up to concentrations of 3 mg/L ferrous iron. In water treatment, the amount of sodium hexametaphosphate should be controlled to ensure that concentrations do not exceed levels that would complex manganese or iron by more than 10%. Control of calcium carbonate ($CaCO_3$) scale rarely requires more than 1 mg/L of polyphosphate.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA):

- arsenic
- fluoride
- iron
- lead

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium hexametaphosphate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Sodium and orthophosphate residues are present in finished water. Sodium hexametaphosphate naturally reverts to orthophosphate over time. Residual orthophosphate encourages biological growth.

NOTE: Important general information is contained in PART II, Chapter 8

The use of sodium hexametaphosphate in the water supply adds to the phosphorous load at the sewage treatment plant. Its use should therefore be considered in consultation with the manager of the sewage treatment plant.

STATUS

Sodium hexametaphosphate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B502-01. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Gosselin RE, Smith RP and Hodge HC (1984). *Clinical Toxicology of Commercial Products*, 5th edition. Williams and Wilkins, Baltimore, II-121.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

NRC (National Research Council) (1981). *Drinking Water & Health*, Volume 4. National Academy Press, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium hydroxide

(endorsed 2005)

Sodium hydroxide is a commonly used alkali suitable for pH adjustment, water softening and corrosion control. It requires only a simple dosing system but needs care in handling.

GENERAL DESCRIPTION

Sodium hydroxide, NaOH (also known as caustic soda), is a white, deliquescent solid. It absorbs water and carbon dioxide from the air. The chemical is supplied as flake or pearl solids, or liquid (usually 30% or 46–50%). It has a specific gravity of 1.33 (at 30%) and 1.48 (at 46%). Liquid solutions of sodium hydroxide can freeze in cold climates, depending on concentration. Climate considerations are relevant for any caustic soda concentrations above 30%, because such solutions can freeze at temperatures above 0°C.

Appropriate handling materials for sodium hydroxide include rubber linings and steel, stainless steel, polyvinyl chloride, polypropylene, fibreglass-reinforced plastic.

CHEMISTRY

Sodium hydroxide is commonly produced by the electrolytic dissociation of sodium chloride, with chlorine gas as a by-product.

For pH and alkalinity adjustment, caustic soda simply produces hydroxide ions in water:

$$NaOH \Leftrightarrow Na^{+} + OH^{-}$$

The chemical reactions of sodium hydroxide–soda softening are as follows:

$$CO2 + 2NaOH \Leftrightarrow Na_2CO_3 + H_2O \quad (1)$$

$$Ca(HCO_3)_2 + 2NaOH \Leftrightarrow CaCO_3 + Na_2CO_3 + 2H_2O \quad (2)$$

$$Mg(HCO_3)_2 + 4NaOH \Leftrightarrow Mg(OH)_2 + 2Na_2CO_3 + 2H_2O \quad (3)$$

$$MgSO_4 + 2NaOH \Leftrightarrow Mg(OH)_2 + Na_2SO_4 \quad (4)$$

$$CaSO_4 + Na_2CO_3 \Leftrightarrow CaCO_3 + Na_2SO_4 \quad (5)$$

Sodium carbonate produced from equation (1) precipitates calcium noncarbonate hardness, as shown in equation (5). Sodium hydroxide can be used in combination with lime, depending on the amount of calcium noncarbonate to be removed.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sodium hydroxide is often used instead of powdered alkalis such as lime or soda ash, because the systems for adding sodium hydroxide are less complicated and require less maintenance. The chemical can also be used in place of lime to soften water by removing carbonate and noncarbonate hardness. Sodium hydroxide can also partially or fully substitute for the soda ash requirement.

Sodium hydroxide is used to raise pH and to convert excess carbon dioxide to alkaline species. Typical concentrations used are 2–100 mg/L (as caustic soda), but higher concentrations may be required with waters of poor quality.

Sodium hydroxide imparts a change of 1.55 mg/L calcium carbonate ($CaCO_3$) alkalinity per mg/L as

NOTE: Important general information is contained in PART II, Chapter 8

NaOH. Control of pH is difficult when sodium hydroxide is added to poorly buffered water.

For concentrations up to about 30%, caustic soda freezes at below 0°C. At 40% concentration, caustic soda will freeze at 15°C, dropping back to around 5°C at 46%. Concentrations above 50% freeze at 12°C or higher. In cold climates it may be necessary to dilute caustic solutions or heat caustic storage and delivery facilities. Softened water should be used for dilution to minimise scaling.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA, KIWA 1994, NRC 1982):

- arsenic
- iron
- cadmium
- lead
- chloride
- mercury
- chromium
- nickel

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium hydroxide should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

The amount of sodium added to water when sodium hydroxide is used to adjust pH is generally insignificant.

STATUS

Sodium hydroxide was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B501-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Benefi eld LD and Morgan JM (1999). Chemical Precipitation. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, New York, 10.1–10.60.

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

KIWA (1994) *Guideline quality of materials and chemicals for drinking water supplies*. Inspectorate of Public Health and Environmental Planning, Publication 94-01. Rijswijk, The Netherlands.

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

NRC (National Research Council) (1982). Water Chemicals Codex. Committee on Water Treatment Chemicals, Food and Nutrition Board, Assembly of Life Sciences, NRC, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium hypochlorite

(endorsed 2005)

Sodium hypochlorite is used as a disinfectant and oxidant in the treatment of drinking water. It provides available chlorine in a liquid form, with less risk than storing and handling chlorine gas.

GENERAL DESCRIPTION

Sodium hypochlorite, NaOCl, or liquid bleach, is a strong oxidising agent that is usually stored and used in solution. It has a disagreeable, sweetish odour and a pale greenish colour. Sodium hypochlorite solution releases vapours that cause corrosion in the presence of moisture.

Sodium hypochlorite is usually supplied as 10–13% w/v available chlorine. More concentrated solutions are not practical because of the instability of sodium hypochlorite, which forms chlorate and chlorite (both of which are of potential concern to health) as solution strength increases. Other factors that affect the stability of sodium hypochlorite are temperature, period of storage, impurities and exposure to light. The oxidising capability of 1 L of sodium hypochlorite (12.5% strength) is equivalent to the oxidising capability of 125 g of chlorine gas. Sodium hypochlorite is generated by combining chlorine and sodium hydroxide.

Suitable materials for storing and handling sodium hypochlorite include ceramics, glass, fibreglass reinforced plastic, polyethylene and polyvinyl chloride, and rubber or plastic linings.

CHEMISTRY

Sodium hypochlorite is generated by combining chlorine and sodium hydroxide.

Sodium hypochlorite hydrolyses in water forming hypochlorous acid (HOCl), which partially dissociates to hypochlorite ion (OCl^-):

$$NaOCl \rightarrow Na^+ + 2OCl^-$$

$$OCl^- + H_2O \Leftrightarrow HOCl + OH^-$$

The relative distribution of hypochlorous acid and hypochlorite ion resulting from these reactions depends on pH and temperature. At 25°C, hypochlorous acid is the predominant species between pH 1 and pH 7.5, and hypochlorite ion predominates at pH values greater than 7.5. Oxidation reactions and the disinfecting properties of chlorine tend to be more effective at low pH values, because of the predominance of hypochlorous acid, which is a stronger oxidant.

Sodium hypochlorite is a base, which will raise the pH of water, whereas chlorine gas produces an acidic reaction that lowers the pH of the solution. The extent of the pH change will depend on the alkalinity of the water.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sodium hypochlorite is used as a disinfectant. Sodium hypochlorite solution is more expensive than chlorine, but its use is becoming more widespread, because of concerns about the safe transport and handling of hazardous gaseous chlorine in pressurised tanks.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium hypochlorite can be added at various points of the treatment process:

- for oxidation of organics or metals
- for disinfection purposes
- to maintain a chlorine residual in the distribution system (pre-coagulation, intermediate or postfiltration chlorination).

Concentrations can range from 1 to 5 mg/L (as available chlorine), although 2–3 mg/L is typical. The selection of the appropriate chlorine dose should take into account the amount of disinfection by-products formed, and the required C.*t* (concentration × contact time) and chlorine residual; the World Health Organization (WHO) recommends 0.5 mg/L for 30 minutes. A free chlorine residual of more than 0.2 mg/L throughout the distribution system is preferred. In some systems, rechlorination is employed within the distribution system, with chlorine added after water has left the treatment plant, to boost chlorine residuals.

Superchlorination (10–50 mg/L as available chlorine) may be used to disinfect or clean tanks or pipelines. It can also be used to temporarily treat taste and odour issues caused by high ammonia levels. The process is usually followed by dechlorination, to chemically remove excess chlorine.

Knowledge of the breakpoint phenomenon (whereby chlorine applied in sufficient doses will oxidise ammonia and eliminate chloramines, resulting in the formation of a free chlorine residual) is necessary when dealing with water containing ammonia.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in sodium hypochlorite:

- chlorate
- iron
- manganese
- mercury
- nickel

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium hypochlorite should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

The use of a disinfectant such as chlorine results in the formation of free chlorine and combined chlorine residuals and disinfection by-products. By-products include trihalomethanes (THMs), haloacetic acids (HAAs), haloacetonitriles (HANs), haloketones, chloral hydrate and chloropicrine. Although many specific chlorine disinfection by-products have been identified, a significant percentage of the total organic halogens have yet to be identified.

Many factors affect the distribution of disinfection by-product species, including pH, temperature and levels of total organic carbon (TOC), bromide and chlorine. The THMs (chloroform, bromodichloromethane, dibromochloromethane, bromoform) are the best known chlorination by-products. Chlorinated THM, HAA and HAN species are generally present in higher concentrations than brominated species; however, brominated species predominate in high-bromide waters.

STATUS

Sodium hypochlorite was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

NOTE: Important general information is contained in PART II, Chapter 8

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B300-99. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Connell GF (1996). *The Chlorination/Chloramination Handbook*. Water Disinfection Series, American Water Works Association. Denver, Colorado.

White GC (1992). *Handbook of chlorination and alternative disinfectants*, 3rd edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium silicate

(endorsed 2005)

Sodium silicate, in the form of 'activated silica,' is used as a coagulant or a flocculation aid in the treatment of drinking water, in conjunction with a primary coagulant (e.g. alum). Soluble silicates (waterglass) can also be used to inhibit corrosion or sequester metals, and sodium silicate solution can be used to adjust pH in small water systems.

GENERAL DESCRIPTION

Sodium silicate, $Na_2O{\cdot}xSiO_2$, can be in the form of lumps of greenish glass (soluble in steam), white powders of varying degrees of solubility, or as cloudy or clear solutions of varying viscosity.

Soluble silicates can be differentiated by their ratio of silica to sodium oxide (SiO_2:Na_2O). This ratio, which ranges from 1.6 to 3.3 by weight, determines the physical and chemical properties of the product. Liquid silicates with a ratio of 1.6 have a pH of 13.2; whereas, at a ratio of 3.3 the pH is 11.0. The specific gravity of these solutions ranges between 1.4 and 1.6. The colloidal and polymeric properties of liquid silicates increase as the SiO_2:Na_2O ratio increases.

Appropriate materials for handling sodium silicate include cast iron, steel, fibreglass-reinforced plastic and polyethylene, and rubber linings.

CHEMISTRY

Sodium silicate is produced by fusing high purity silica sand with sodium carbonate or potassium carbonate at 1000–1500°C. This results in an amorphous glass, which can be dissolved in water to form silicate solutions or 'waterglass'.

In solution, silica is present in equilibrium between monomeric anionic species. The proportion of silica and alkali in a sodium silicate is usually expressed as the weight ratio of SiO_2 to Na_2O.

In drinking water treatment, solutions of activated or colloidal silica can be used for coagulation. Such solutions can be generated on site by partial or complete neutralisation of a dilute solution of sodium silicate by a mineral acid, an acid salt or chlorine. The activated silica solution obtained can be slightly alkaline or neutral, and is aged for a short time (1–2 hours) before use. The solution is then further diluted with 2–2.5 volumes of water. The activated silica solution has a shelf life of 1–2 days.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

At one time, activated silica was commonly used as a coagulant aid (after a primary coagulant such as alum or ferric chloride), because it forms heavy, tough flocs that settle fast. However, polyacrylamide polymers have now largely replaced activated silica in most water treatment plants.

Soluble silicates are also used to protect metals from the corrosive effects of water by depositing a thin molecular film of silica (SiO_2) on metal surfaces. Silicate treatment is effective for corrosion control of concrete and a variety of metals: lead, copper, cast iron, ferrous metals, steel, galvanised steel, bronze, red and yellow brass, and nickel alloys. The pH and alkalinity of the water determine which silicate is suitable for this application.

Sodium silicate can also be used to sequester iron and manganese. Following metal oxidation, sodium silicate is added to hold oxidised metals in a colloidal suspension.

NOTE: Important general information is contained in PART II, Chapter 8

Concentrations of activated silica used in drinking water treatment can range from 1 to10 mg/L (as SiO_2), and the concentration required varies with water quality, depending on factors such as pH, turbidity, colour, temperature and contaminant level.

The effectiveness of sodium silicate as a corrosion inhibitor depends on water quantities such as pH and bicarbonate concentration. The chemical is more effective under high-velocity flow conditions. Silicate is effective at high pH, and at a dosage over 15–20 mg/L (as SiO_2).

Silicate with a high ratio of Na_2O to SiO_2 will raise pH in weakly buffered waters. For corrosion control, relatively high concentrations (up to 24 mg/L) are required during the first 30–60 days of treatment, to form the initial protective coating. Thereafter, the silicate dosage is reduced incrementally in 30-day periods, until it reaches maintenance doses (4–8 mg/L).

As a metal sequestrant, sodium silicate (as SiO_2) should be added at up to 4–5 times the level of iron or manganese in the water.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. More than 20 elements are present as trace impurities in sodium silicate, including:

- aluminium
- cadmium
- calcium
- chloride
- iron
- magnesium
- manganese
- sulfate

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

Sodium and silicate residues are present in finished water. When employed in drinking water treatment, sodium silicate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Sodium silicate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B404-98. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

NOTE: Important general information is contained in PART II, Chapter 8

Sodium tripolyphosphate

(endorsed 2005)

Sodium tripolyphosphate is used in drinking water treatment to control corrosion and soften water; it is also used as a sequestering and descaling agent, and to stabilise or disperse calcium and iron in the water distribution system.

GENERAL DESCRIPTION

Sodium tripolyphosphate, $Na_5P_3O_{10}$, is a white powder or granular solid, and is odourless. A 1% aqueous solution of sodium tripolyphosphate has a pH of 9.8; the pH of a concentrated solution (slurry) is about 10.5.

Appropriate handling materials for sodium tripolyphosphate include cast iron, steel, fibreglass-reinforced plastic, polyethylene and polyvinyl chloride; rubber-lined containers can also be used.

CHEMISTRY

Sodium tripolyphosphate is manufactured by combining soda ash or caustic soda with phosphoric acid. The product is then heated to form crystalline solids.

Low concentrations of polyphosphate inhibit the precipitation of calcium salts, and therefore inhibit scale formation. If phosphate concentrations are increased, then calcium phosphate precipitates. A further increase in concentration results in the sequestration phenomenon, whereby calcium is sequestered, inhibiting scale formation. Sequestering is affected by pH, with a neutral to alkaline pH being more effective.

Sodium tripolyphosphate can be used as a corrosion inhibitor in combination with divalent cations such as calcium (Ca^{2+}). Positively charged colloidal complexes form, migrate to the cathode and create an amorphous polymeric film. This inhibition is most effective at a pH of 6.5–7.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

The chemical is used in drinking water treatment to control corrosion and soften water; it is also used as a sequestering and descaling agent, and to stabilise or disperse calcium and iron in the water distribution system.

Polyphosphates can change the characteristics of corrosion, making it more uniform rather than a pitting type of corrosion. Polyphosphates have also been used to control oxidation of ferrous iron dissolved from pipes, and to reduce the formation of 'red water' (caused by contamination with hydrated iron oxide). When mixed with orthophosphate, polyphosphates may assist in the formation of an orthophosphate film, by complexing calcium or manganese in hard waters that might otherwise cause unwanted orthophosphate precipitates.

Typical doses for protection against scale, corrosion and prevention of 'red water' range from 0.5 to 20 mg/L, although doses of up to 50 mg/L may be used during mains cleaning.

For corrosion control in a cast-iron distribution system, an initial feed of 5–10 mg/L may be applied for several weeks, followed by a maintenance dosage of 1–2 mg/L; or a continuous dosage of 1–5 mg/L may be used.

For sequestration applications, a ratio of 3.4–5 parts sodium tripolyphosphate per water hardness (as $CaCO_3$) is recommended by manufacturers.

NOTE: Important general information is contained in PART II, Chapter 8

Control of post-precipitation in softened water typically requires a dosage of 0.5–2 mg/L.

Laboratory or pilot trials should be undertaken to determine the appropriate doses.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA):

- arsenic
- fluoride
- iron
- lead
- phosphates

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sodium tripolyphosphate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

Sodium and orthophosphates are present in finished water and can cause problems. For example, phosphates increase biological activity in the distribution system, and polyphosphates both reduce the deposition of protective calcium-containing films and increase the solubility of metals, interfering with the formation of passivating films. Polyphosphates also soften asbestos-cement pipe by accelerating the depletion of calcium and inhibiting the formation of fibre-binding iron or manganese deposits. Similar effects can occur in cement-lined or concrete pipes.

The use of sodium tripolyphosphate in the water supply adds to the phosphorous load at the sewage treatment plant. Its use should therefore be considered in consultation with the manager of the plant.

STATUS

Sodium tripolyphosphate was endorsed by the NHMRC for use as a drinking water treatment chemical in 2005.

REFERENCES

ANSI (American National Standards Institute)/AWWA (American Water and Wastewater Association) Standard no B503-01. AWWA CD-ROM (April 2003). Available at <www.awwa.org>

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

NOTE: Important general information is contained in PART II, Chapter 8

Sulfuric acid

(endorsed 2005)

Sulfuric acid is used to correct pH in coagulation, water softening, corrosion control, prevention of post-precipitation and activation of silica.

GENERAL DESCRIPTION

Sulfuric acid (H_2SO_4) is a strongly corrosive, dense, oily liquid. It is colourless to dark brown, depending on purity, and is miscible with water. Sulfuric acid is generally available in concentrations of 28.5–98.5%, with corresponding specific gravity of 1.2–1.85 at 20°C. The acid is very reactive and dissolves most metals; the concentrated acid oxidises, dehydrates or sulfonates most organic compounds, often causing charring.

Sulfuric acid is highly corrosive to most metals and alloys, and is corrosive to mild steel at concentrations below 90%. It can be stored in fibreglass-reinforced plastic with acid resistant resins, polyethylene, porcelain, glass and rubber linings.

CHEMISTRY

Sulfuric acid is usually produced using the Contact process: sulfur dioxide is catalytically converted to sulfur trioxide, which is then dissolved in sulfuric acid and water.

Sulfuric acid disassociates in water to produce a strong acid:

$$H_2SO_4 \Leftrightarrow 2H^+ + SO_4^{2-}$$

Sulfuric acid is added to lime–soda softened waters to prevent post-precipitation of calcium carbonate and magnesium hydroxides in filters or in water distribution systems. These water usually have pH values of approximately 10.4, and are supersaturated with calcium carbonate ($CaCO_3$) and magnesium hydroxide ($Mg(OH)_2$). Sulfuric acid is therefore used to reduce excessive pH values and alkalinities as follows:

$$H_2SO_4 + 2CaCO_3(s) \Leftrightarrow Ca\ (HCO_3)_2 + CaSO_4$$

$$H_2SO_4 + Ca(OH)_2 \Leftrightarrow CaSO_4 + 2H_2O$$

Sulfuric acid is used to fortify hydrolysing metal salts (aluminium and iron). The typical acid-fortified alum product, also called acidulated alum or acid alum, contains 5–20% (weight basis) of sulfuric acid. For a given amount of metal ion added to the water, strong acid-fortified products react with more alkalinity and depress the pH to a greater extent than nonfortified metal salt solutions.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, sulfuric acid is used to correct pH in coagulation, water softening, corrosion control, prevention of post-precipitation and activation of silica.

Handling and adding concentrated sulfuric acid to water requires extreme caution, because it can cause severe burns and eye damage. Also, sulfuric acid has an exothermic reaction with water that may cause violent splattering. Careful design is required in dilution systems for sulfuric acid, because the significant heating that may occur could damage pipework.

Concentrations of sulfuric acid required vary widely, depending on the alkalinity of the water and the pH required. Low concentrations (1–30 mg/L) are usually adequate to adjust pH for coagulation; higher doses may be required for water softening.

NOTE: Important general information is contained in PART II, Chapter 8

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product (JECFA):

- antimony
- arsenic
- cadmium
- chloride
- chromium
- copper
- fluoride
- iron
- lead
- manganese
- mercury
- selenium
- sulfate
- sulfur dioxide
- zinc

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

When employed in drinking water treatment, sulfuric acid should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Sulfuric acid was endorsed by the NHMRC for use as a drinking water treatment chemical in 1983. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Waterand Wastewater*, 20th edition. American Public Health Association, Washington, DC.

JECFA (Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Joint Expert Committee on Food Additives). *Compendium of Food Additive Specifications*. FAO Food and Nutrition Papers 52 (two volumes). Available at <www.fao.org/es/esn/jecfa/database/cover.htm>

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

Schock MR (1999). Internal Corrosion and Deposition Control. In: *A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, 17.1–17,109.

NOTE: Important general information is contained in PART II, Chapter 8

Zinc orthophosphate

(endorsed 2005)

Zinc orthophosphate is used to inhibit corrosion of lead, copper and iron, and to prevent the release of asbestos or cement from water pipes.

GENERAL DESCRIPTION

Zinc orthophosphate, $Zn_3(PO_4)_2$, solution is a clear odourless liquid that is soluble in water; it is available in various ratios of phosphate to zinc.

Appropriate materials for handling zinc orthophosphate include cast iron, steel, fibreglass-reinforced plastic, polyethylene and polyvinyl chloride; rubber-lined containers can also be used.

CHEMISTRY

Zinc orthophosphate is manufactured using zinc salts (chloride or sulfate) and orthophosphate. Zinc orthophosphate limits the release of lead, copper and iron from metal surfaces by forming a microscopic protective film on these surfaces, and by electrochemical passivation. Water with a high pH (> 8.1) should not be treated with zinc orthophosphate because of zinc hydroxide precipitation.

Reactions between orthophosphate and lead in water result in the formation of several solids that are less soluble than basic lead carbonate over a wide range of pH values. The most likely solid phase formed is hydroxypyromorphite ($Pb_5(PO_4)_3OH$). Tertiary lead orthophosphate ($Pb_3(PO_4)_2$) is another solid formed. The formation of lead orthophosphate films depends on the concentration of dissolved inorganic carbon (DIC; e.g. carbonates), acidity, temperature and orthophosphate content. These phosphate films may not form as rapidly as the basic lead carbonate solids. Carbonate competes with orthophosphate for control of lead solubility. Hence, lead orthophosphate films can be formed in water with low levels of carbonate or DIC (these two characteristics are often found together), in which case the effectiveness of a phosphate control program may need to be evaluated over a longer time.

TYPICAL USE IN AUSTRALIAN DRINKING WATER TREATMENT

In drinking water treatment, zinc orthophosphate is used to inhibit corrosion. It is particularly effective at inhibiting lead corrosion, because it reduces lead solubility in waters with both low and high alkalinity. The chemical is used to treat waters that are soft and corrosive. Zinc orthophosphate suppresses corrosion of carbon steel, and the release of asbestos fibres from asbestos–cement (A–C) pipe. It also inhibits corrosion of cast iron, and mildly inhibits corrosion of copper.

A few milligrams per litre of orthophosphate are sufficient at pH values in the 7–9 range.

CONTAMINANTS

The purity of chemicals used in Australia for the treatment of drinking water varies, depending on the manufacturing process. The following chemical contaminants may be present in this product:

- chloride
- sulfate

RESIDUAL AND BY-PRODUCT FORMATION IN DRINKING WATER

NOTE: Important general information is contained in PART II, Chapter 8

When employed in drinking water treatment, zinc orthophosphate should be used in such a way that any contaminant or by-product formed by the use of the chemical does not exceed guideline values in the *Australian Drinking Water Guidelines*.

STATUS

Zinc orthophosphate was endorsed by the NHMRC for use as a drinking water treatment chemical in 1987. The revision undertaken in 2003 did not change the status of this chemical for the treatment of drinking water.

REFERENCES

Clesceri LS, Greenberg AE and Eaton AD (eds) (1998). *Standard Methods for the Examination of Water and Wastewater*, 20th edition. American Public Health Association, Washington, DC.

Gosselin RE, Smith RP, Hodge HC (1984). *Clinical Toxicology of Commercial Products*, 5th edition. Williams and Wilkins, Baltimore, II-121.

Lewis RJ (1993). *Hawley's Condensed Chemical Dictionary*, 12th edition. Van Nostrand Reinhold, New York.

NRC (National Research Council) (1981). *Drinking Water & Health*, Volume 4. National Academy Press, Washington, DC.

Schock MR (1999). Internal Corrosion and Deposition Control. In: *Water Quality and Treatment, A Handbook of Community Water Supplies*, Letterman RD (ed), American Water Works Association, 5th edition. McGraw-Hill Professional, 17.1–17.109.

Shibata H, Morioka T (1982). Antibacterial action of condensed phosphates on the bacterium *Streptococcus mutans* and experimental caries in the hamster. *Archives of Oral Biology* 27(10): 809–16.

NOTE: Important general information is contained in PART II, Chapter 8

APPENDICES

Appendix 1: Additional guidance on elements 2 and 3 of the Framework for management of drinking water quality

This appendix provides additional guidance on *Assessment of the drinking water supply system* (element 2) and *Preventive measures for drinking water quality management* (element 3) of the Framework for management of drinking water quality (the Framework). This appendix should be read in conjunction with Chapter 3, which provides a more comprehensive overview of these elements.

Users are also encouraged to draw on the numerous sources providing detailed technical guidance, a number of which have been listed in Section A9.

A1.1 Introduction

Effective management of drinking water quality requires appropriate attention to system analysis and system management. The objectives are to increase understanding of:

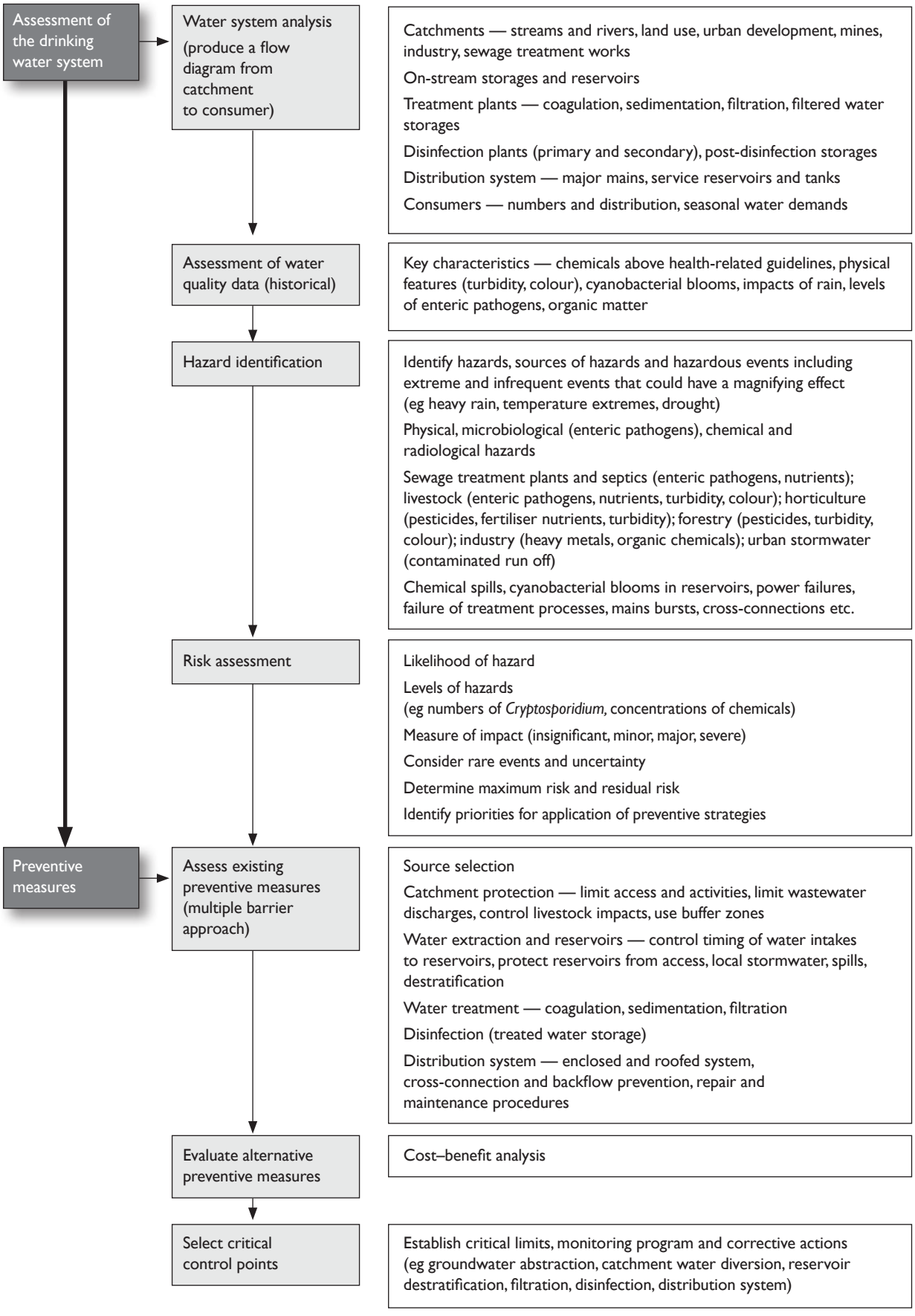
the entire water supply system from catchment to consumer

the hazards, sources and events that can compromise drinking water quality

the preventive measures needed to effectively control hazards, including the application of multiple barriers and the establishment of critical control points to reduce exposure to hazards.

Figure A1.1 provides a suggested roadmap to assist in the application of these aspects of the Framework. Further guidance on implementing these aspects is offered in the following text.

Figure A1.1 *Application of Framework elements 2 and 3*



A1.2 Water supply system analysis

Assessment of the drinking water system provides an important information base and is a prerequisite for subsequent steps in which strategies for prevention and control of hazards are planned and implemented. The purpose of this element is to develop a broad overview and basic understanding of the water supply system. It is not intended to be an extensive data collection exercise; rather, it is the characterisation of the system at an appropriate level of detail to provide a useful information base from which to make effective decisions.

Summary of actions

Assemble a team with appropriate knowledge and expertise.

Construct a flow diagram of the water supply system from catchment to consumer.

Assemble pertinent information and document key characteristics of the water supply system to be considered (see Table A1).

Periodically review the water supply system analysis.

Characterisation of the water supply system should be fully documented and should be a collaborative effort between relevant agencies. Characterisations will be specific for each system but should include, where appropriate, consideration of the catchment area, source water, groundwater system, reservoirs and raw water transport, treatment systems, distribution system and consumers.

Table A1.1 provides examples of some key characteristics to be considered in assessing drinking water supply systems from catchment to consumer. Seasonal characteristics, as well as extreme and infrequent events such as droughts or floods, should also be considered.

Much of the necessary information may be available in existing documentation from studies carried out previously or from external agencies. Sources of useful information can include:

land use surveys and catchment maps

sanitary surveys

surveys of major streams and rivers

research and investigative monitoring

inspections and field audits

employee knowledge

records from local authorities (eg locations of septic tanks, animal feedlots, sewage treatment plants)

community surveys

public and consumer complaints.

Geographic information systems (GIS) can provide a useful means of displaying, cataloguing and interpreting data.

Table A1.1 *Key characteristics of the drinking water supply system*

Catchments

- Geology and soils
- Topography and drainage patterns (hydrology)
- Streams and rivers
- Meteorology and weather patterns (climatic and seasonal variations)
- Riparian conditions
- Vegetative cover
- General catchment and river health
- Wildlife (eg native and feral animals)
- Historical contaminated sites
- Competing water uses
- Land irrigation practices
- Nature and intensity of development and land-use activities:
  - agricultural, dairy and animal husbandry
  - land clearing
  - forestry
  - mining
  - industrial
  - rural and urban development / residential
  - sewage treatment works and septic tanks
  - recreational activity
- Intermittent or seasonal use practices
- Future planning activities
- Development and planning restrictions

Source water

- Surface water (river, reservoir, dam)
- Groundwater
- Flow and reliability of source water
- Seasonal and event changes (including infrequent events such as droughts or floods)
- Spatial variations
- General and unique constituents (physical, chemical, microbial):
  - major ions and pH
  - salinity, hardness
  - turbidity
  - bacteria, viruses and protozoa
  - naturally occurring organics
  - volatile and nonvolatile synthetic organics
  - metals and radionuclides

Groundwater systems

- Geology, homogeneity
- Confined or unconfined aquifer
- Depth to water table
- Flow rate and direction
- Dilution characteristics
- Recharge area
- Well-head protection
- Depth of casing

Storage reservoirs and intakes

- Detention times
- Reservoir design:
  - size
  - materials
  - storage capacity
  - depth of storage
- Seasonal variations:
  - stratification
  - algal blooms
- Treatment efficiencies (microbial removal)
- Protection (eg covers, enclosures, access)
- Recreational or human activity
- Intake location and operation
- Bulk transport:
  - pipeline material
  - length
  - flow rate and changes in flow rate
  - cleaning systems

Treatment systems

- Treatment processes (including optional processes)
- Treatment configuration
- Equipment design:
  - size
  - materials
  - peak flow rates
  - process change control
  - backup systems
- Monitoring equipment and automation
- Water treatment chemicals used:
  - coagulant
  - filtration aids
  - fluoride
  - powdered activated carbon
  - disinfectant
- Treatment efficiencies
- Disinfection log removals of pathogens
- Disinfection residual and contact period

Table A1.1 *Key characteristics of the drinking water supply system (continued)*

Service reservoirs and distribution systems	
• Reservoir design: – size – materials – storage capacity – depth of storage • Detention times • Seasonal variations: – stratification • Protection (eg covers, enclosures, access)	• Distribution system design: – size – network – pipe materials – pipe age • Hydraulic conditions (eg detention times, flows) • Backflow protection • Secondary disinfection practices • Disinfectant residuals • Disinfection byproducts
Consumers	
• Consumer distribution (industry, bodies corporate, general community) • Vulnerable groups (hospitals, nursing homes)	• Water demand and patterns of drinking water consumption (diurnal and seasonal variations) • Internal plumbing

A1.3 Assessment of water quality data

A review of historical data from source waters, treatment plants and finished water supplied to consumers can assist in understanding drinking water system characteristics and the identification of hazards.

Summary of actions

Assemble historical data from source waters, treatment plants and finished water supplied to consumers (over time and following specific events).

Assess data using tools such as control charts and trends analysis to identify trends and potential problems.

Water quality data should be reviewed both over time and following specific events (eg heavy rainfall) to identify those aspects of the system that require improvement. Water quality parameters that can provide useful information include:

turbidity or particle counts

microbial quality

chemical quality

algal counts

naturally occurring organic matter

colour

pH

disinfectant residuals

disinfection byproducts.

Tools that may be useful in assessing data include control charts and modelling methods (eg using temporal overlays of water quality records and climatic information). In some cases, awareness of potential problems or hazards can be difficult because events occur gradually or result from cumulative effects. Trends analysis can be a valuable tool for recognising the accumulation of gradual changes and for predicting where things may be going wrong.

A1.4 Hazard identification

Adoption of a risk-based approach that includes the identification of hazards from catchment to consumer and the assessment of the potential impact on drinking water quality and human health (ie risk) is essential to effective system management. Hazard identification and risk assessment are useful for understanding the vulnerability of a drinking water supply and planning effective risk management strategies to assure drinking water quality and safety.

The purpose of this element is to identify and document all potential hazards and the hazardous events and sources that might give rise to the presence of these hazards.

Summary of actions

Define the approach and methodology to be used for hazard identification. Devise an evaluation team with appropriate representatives.

Review hazardous agents in drinking water and ensure that their link to public health is understood (see Section V — Fact Sheets).

Identify and document hazards, sources and hazardous events for each component of the water supply system (see Tables A1.2 and A1.3).

Periodically review and update the hazard identification to incorporate any new hazards.

A structured approach is important to ensure that significant issues are not overlooked and that areas of greatest risk are identified. There is no single right way to perform these activities; however, the process should involve a structured and comprehensive evaluation of the water supply system.

For each component of the water supply system, all hazards and hazardous events and sources that might affect drinking water quality and safety (what can happen and how) should be identified and documented. Table A1.2 provides examples of various pollution sources and the potential hazards they produce.

All potential hazards, hazardous events and sources should be included in the assessment, regardless of whether or not they are under the direct control of the drinking water supplier. Continuous, intermittent or seasonal pollution patterns should also be considered as well as extreme and infrequent events such as droughts or floods. Table A1.3 provides examples of potential sources and hazardous events, from catchment to consumer, to be considered.

Table A1.2 *Examples of sources and potential hazards*[a]

Potential sources	Potential hazard
Septic tanks	Pathogens,[b] nitrates/nitrites
Sewage treatment plants	Pathogens, nutrients
Animal husbandry	Pathogens, nutrients, turbidity, colour
Horticulture	Pesticides, fertiliser nutrients, turbidity, colour
Rural stormwater	Pathogens, turbidity, colour
Forestry	Pesticides, turbidity, colour
Industry	Heavy metals, organic chemicals including halogenated organics; specific industries can be associated with specific types of contaminants (eg arsenic and copper associated with wood preserving, cadmium and chromium with electroplating and chromium with leather tanning)
Mining	Acid mine wastes from pyrites tailings can release and transport metals such as aluminium, iron and manganese; other naturally occurring metals such as cadmium and copper can also be leached; arsenic can be associated with old goldfield areas
Urban stormwater	Lead and zinc from roads, turbidity, colour, petrol/oil products, microorganisms from pets (lower range of pathogens than from humans or livestock waste)
Stormwater/sewer overflows	Pathogens, nutrients, turbidity, colour

a Human and animal waste represent the largest sources of potential hazards in drinking water. Both can include high numbers of enteric pathogens and large amounts of nutrients. Due to the scale of primary production in Australia, the total amount of livestock waste would greatly exceed the amount of human waste.

b The potential range of pathogens present will vary according to the type of waste involved. Many enteric pathogens, and in particular viruses and protozoa, infect only one species. In general, human enteric viruses are only carried and excreted by humans. Human infectious *Cryptosporidium parvum* can be carried by humans and livestock, but the current state of knowledge suggests that the species of *Cryptosporidium* that infect birds do not infect humans.

Table A1.3 *Examples of hazardous events and their potential sources*

Catchments and groundwater systems	
• Rapid variations in raw water quality	• Poorly vegetated riparian zones, failure of sediment traps and soil erosion
• Sewage and septic system discharges	• Stormwater flows and discharges
• Industrial discharges	• Existing or historical waste-disposal or mining sites/ contaminated sites and hazardous wastes
• Chemical use in catchment areas (eg use of fertilisers and agricultural pesticides)	• Unconfined and shallow aquifers
• Major spills and accidental spillage	• Groundwater under direct influence of surface water
• Public roads	• Inadequate well-head protection and unhygienic practices
• Human access (recreational activity)	• Uncased or inadequately cased bores
• Wildlife (native and feral)	• Saline intrusion of coastal aquifers
• Unrestricted livestock	• Contaminated aquifers
• Inadequate buffer zones	• Climatic and seasonal variations (eg heavy rainfalls, droughts)
• Surrounding land use (eg animal husbandry, agriculture, forestry, industrial area, waste disposal, mining)	• Bushfires, natural disasters, sabotage
• Changes in surrounding land use	

Table A1.3 *Examples of hazardous events and their potential sources (continued)*

Storage reservoirs and intakes	
• Open reservoirs and aqueducts, uncovered storages	• Stratification
• Human access/absence of exclusion areas around shorelines	• Soil erosion
• Animal access including birds and vermin	• Inadequate buffer zones and vegetation
• Short-circuiting of reservoir	• Climatic and seasonal variations (eg heavy rainfalls, droughts)
• Depletion of reservoir storage	• Public roads / accidental spillage
• No selective withdrawal	• Failure of alarms and monitoring equipment
• No alternative water sources	• Bushfires and natural disasters
• Unsuitable intake location	• Sabotage
• Cyanobacterial blooms	
Treatment systems	
• Significant flow variations through water treatment system	• Failure of dosing equipment
• Incapable equipment or unit processes	• Inadequate filter operation and backwash recycling
• Inadequate backup	• Ineffective disinfection
• Inappropriate treatment processes	• Equipment malfunctions
• Process control incapability or operational inflexibility	• Poor reliability of processes
• Use of unapproved or contaminated water treatment chemicals and materials	• Failure of alarms and monitoring equipment
	• Power failures
• Chemical dosing failures	• Sabotage and natural disasters
• Inadequate mixing	• Formation of disinfection byproducts
Service reservoirs and distribution systems	
• Open reservoirs and aqueducts / uncovered storages and unprotected pipe system	• Biofilms, sloughing and resuspension, regrowth
	• Pipe bursts or leaks
• Human access, absence of exclusion areas around shorelines	• Inadequate repair and maintenance, inadequate system flushing and reservoir cleaning
• Animal access including birds and vermin	
• Short-circuiting of reservoir, stagnation zones	• Commissioning new mains
• Buildup of sediments and slimes	• Inadequate disinfection after construction, repairs
• Inappropriate materials and coatings or material failure	• Flow variability, inadequate pressures
• Aged pipes, infrastructure	• Treatment dosing failure
• Corrosion of reservoirs and pipe system	• Inadequate maintenance of chlorine residual
• Mixing of different source waters	• Formation of disinfection byproducts
• Infiltration and ingress of contamination from cross-connections, backflow (soil and groundwater)	• Failure of alarms and monitoring equipment
	• Sabotage and natural disasters
Consumers	
• Potential consumer misuse	• Inappropriate plumbing and construction materials
• Leaching of metals	

A1.5 Risk assessment

The objective of risk assessment is to distinguish between very high and low risks so that priorities for risk management can be established.

Once potential hazards and their sources and events have been identified, the level of risk associated with each hazard or event needs to be estimated. Not all hazards will require the same degree of attention, and risk estimation assists in directing attention and resources to those hazards that are most threatening.

In some instances, an initial screening-level risk assessment may be useful to identify broad issues and show where to focus efforts for a more detailed assessment.

Summary of actions

- Define a consistent approach to be used for risk assessment.
- Evaluate the major sources of uncertainty associated with each hazard and hazardous event and consider actions to reduce uncertainty.
- Determine significant risks and establish and document priorities for risk management (based on assessment of maximum and residual risk).
- Periodically review and update the risk assessment.

An example of an approach to estimating the level of risk is provided in Tables A1.4, A1.5 and A1.6. These tables have been adapted from AS/NZS 4360:1999 ***Risk Management*** and can be modified to meet the needs of an organisation.

Using these tables to guide a risk assessment will quickly reveal the need to define the level of detail required and format to be used for classifying events. Events may arise along a continuum from commonly recurring incidents of minor consequence to rarer incidents with more serious consequences. In some cases, variations of the same type of event can appear at both ends of the spectrum. For example, 'loss of disinfectant residual in the distribution system' can have distinctly different meanings. A slight reduction or a loss in parts of a system may be fairly common and have limited health consequences; a total loss of disinfection should be rare but could have potentially severe consequences. There is no set of rules to be followed in using these tables; rather, they are offered as a general guide for the development of a consistent methodology that will be relevant for the water system under study.

Table A1.4 *Qualitative measures of likelihood*

Level	Descriptor	Example description
A	Almost certain	Is expected to occur in most circumstances
B	Likely	Will probably occur in most circumstances
C	Possible	Might occur or should occur at some time
D	Unlikely	Could occur at some time
E	Rare	May occur only in exceptional circumstances

Table A1.5 *Qualitative measures of consequence or impact*

Level	Descriptor	Example description
1	Insignificant	Insignificant impact, little disruption to normal operation, low increase in normal operation costs
2	Minor	Minor impact for small population, some manageable operation disruption, some increase in operating costs
3	Moderate	Minor impact for large population, significant modification to normal operation but manageable, operation costs increased, increased monitoring
4	Major	Major impact for small population, systems significantly compromised and abnormal operation if at all, high level of monitoring required
5	Catastrophic	Major impact for large population, complete failure of systems

Table A1.6 *Qualitative risk analysis matrix — level of risk*

Likelihood	Consequences				
	1 Insignificant	**2 Minor**	**3 Moderate**	**4 Major**	**5 Catastrophic**
A (almost certain)	Moderate	High	Very high	Very high	Very high
B (likely)	Moderate	High	High	Very high	Very high
C (possible)	Low	Moderate	High	Very high	Very high
D (unlikely)	Low	Low	Moderate	High	Very high
E (rare)	Low	Low	Moderate	High	High

Based on the assessment of risk, priorities for risk management should be determined. Maximum risk in the absence of preventive measures should first be determined to identify high-priority risks and provide an indication of worst-case scenarios in the event of failures. Residual risk, determined in conjunction with evaluation of existing preventive measures, should also be assessed to provide information on the effectiveness of existing strategies and the need for improvements.

Uncertainty

The outcome of hazard identification and risk assessment will depend on the level of uncertainty associated with each parameter. Evaluating the major sources and types of uncertainty associated with the hazards can assist in understanding the limitations of the hazard identification and risk assessment as well as how these limitations can be reduced.

Hazard identification and risk assessment need to explicitly consider the sources and types of uncertainty.

Uncertainty can be broadly classified into two types: *variability* and *knowledge uncertainty*. By documenting the major sources of variability and knowledge uncertainty that arise for all risks, insights can be gained into the appropriate actions for reducing the role of uncertainty.

Variability represents the true differences that can occur in the specific values of parameters that contribute to a risk — for example, contaminant concentrations over time and space, flows and number of people exposed. Variability contributes to uncertainty because it usually cannot be described completely, due to incomplete or insufficient monitoring data, and there is no single correct answer that will cover all circumstances. For example, the mean temperature over a defined period of time will not represent the high and low extremes and these may be more important depending on what we are seeking to know. Because there is variability in temperature, a decision will need to be made on which value or values to use from the available data, and this choice will carry with it some uncertainty.

Knowledge uncertainty represents an inadequate state of knowledge that exists in the values of parameters measured. Knowledge uncertainty may be reflected in a lack of assurance that methods are accurately measuring what is intended or in a lack of understanding of how a process works. For example, in using methods to count *Cryptosporidium* oocysts, there may be a degree of uncertainty that the particles being counted are truly *Cryptosporidium* oocysts. Alternatively, while there may be confidence that the method for counting oocysts is accurate, further uncertainty exists about what the measurement means because it is not known if the oocysts are viable and, if viable, whether they are infective.

There is value in being able to distinguish the relative impacts of variability and knowledge uncertainty. Variability cannot be reduced by more accurate measurement. However, by characterising variability more fully, the nature of a hazard (and thereby the dimensions of the risk) can be better understood. Understanding how variability contributes to uncertainty may lead to actions to change a system to reduce its variability (eg increasing reservoir storage times to minimise fluctuations in water quality).

In contrast to variability, knowledge uncertainty can be reduced by better measurement and research. The increased understanding from reducing knowledge uncertainty can provide greater assurance that the preventive measures being considered will achieve their intended purpose. This requirement supports the need for a research capability within the water industry.

A1.6 Preventive measures and multiple barriers

The identification, evaluation and planning of preventive measures should always be based on system-specific hazard identification and risk assessment. The level of protection used to control a hazard should be proportional to the associated risk.

The multiple barrier principle should be employed and preventive measures should be comprehensive from catchment to consumer. Wherever possible, the focus of these measures should be to prevent contamination in the catchment rather than to rely on downstream control. Box A1.1 (below) provides further information on catchment management and source water protection.

Summary of actions

Identify existing preventive measures from catchment to consumer for each significant hazard and event.

Determine the residual risk.

Evaluate alternative and additional preventive measures where improvement is required.

Document the preventive measures and strategies addressing each significant risk into a plan.

Establish mechanisms to ensure cooperation and development of action plans with external agencies.

Examples of preventive measures and management strategies from catchment to consumer are provided in Table A1.7. An indication of removals of enteric pathogens using the multiple barrier approach is provided in Table A1.8. Table A1.9, in the following section, also provides examples of preventive measures for Giardia from catchment to consumer for a river system.

Once preventive measures addressing each significant risk have been identified, the strategies should be documented into a plan. Any new preventive measures to be implemented over the longer term, such as covering water storages or the introduction of filtration, should be incorporated into an improvement plan (see Section 3.12.2 ***Drinking water quality management improvement plan***).

Where responsibility for preventive measures lies outside the direct control of the drinking water supplier (ie with external agencies), mechanisms for communication to ensure cooperation and development of action plans should be established (see Section 3.1.3 ***Engaging stakeholders***).

Box A1.1 *Catchment management and source water protection*

Catchment management and source water protection provide the first barrier for the protection of water quality. Catchment management usually involves a coordinated approach to develop short-term and long-term plans to enhance water quality and eliminate or control any potential sources of pollution.

Whether water is drawn from surface catchments or underground sources, it is important that the local catchment or aquifer is understood, and that the activities that could lead to water pollution are identified and managed. Effective catchment management and source water protection include development of a catchment management plan with the commitment of land use planning authorities to prevent inappropriate development and to enforce relevant planning regulations.

Catchment management plans

A comprehensive catchment management plan should be developed and implemented to mitigate any existing and potential future risks, and where practical, aim to improve the quality of water harvested over time. The plan should include, where appropriate, the following elements:

- a policy statement identifying the protection of water quality as an explicit objective of local legislation
- preparation and review of land use planning controls jointly with the planning authority
- establishment of agreed processes and criteria for managing development applications
- a clear statement of responsibilities of different agencies and agreed coordination processes
- identification of water quality hazards, estimation of risks and planning of relevant management strategies
- a monitoring program to identify pollution sources, maintain quality control, and collect long-term data to determine trends
- regular documented inspections to monitor catchment conditions and land use changes
- a community awareness program, including strategies for working with landowners to support the catchment management plan
- agreed and tested emergency response plans with relevant emergency services for responding to major pollution events such as spillages or contamination.

The extent to which catchment pollution can be controlled or remediated is often limited in practical terms wherever there are competing water uses and pressure for increased development in the catchment. In devising catchment management plans, it may be necessary or useful to divide large catchments into smaller, more manageable units (eg subcatchments). Where this is done, it is important to ensure that, in combination, the various plans provide an integrated approach across the entire catchment. For large river systems protection may be possible only over limited reaches in the vicinity of the raw water off-take or reservoir inlet.

Planning controls

Well-designed planning regulations are a critical component of sound catchment management and protection of water quality. Where possible, protection of water resources should be included as a principal objective in planning policies.

Planning regulations should address management and control of high-risk development in catchments and aquifer intake areas (eg intensive animal feedlots) and should also address the issue of long-term incremental development. Urban development, agroindustry and general industry should be carefully scrutinised to ensure that they will not impact on water resources. On site waste treatment and disposal systems should be permitted only where sites are suitable and there is minimal risk to the water supply. Such systems should be designed, installed and maintained correctly, and inspected regularly. Defects should be reported and rectified.

Responsibility for the development and implementation of planning strategies and regulations is generally shared between state and local government agencies. It is important that drinking water suppliers and environment and health authorities establish strong links with planning agencies and take an active role in:

- the development or amendment of these planning strategies and regulations
- the evaluation of individual development proposals with respect to potential impacts on water quality or quantity.

Where appropriate, formal agreements should be required to ensure approval conditions are complied with and recorded on land titles to alert potential purchasers of the obligations associated with the property.

Box A1.1 *Catchment management and source water protection (continued)*

Community awareness

Community awareness programs should be developed to promote the protection of water quality. Support for local landcare and watercare groups is a relatively low-cost opportunity to develop community awareness and reduce pollution risks.

Diffuse sources of pollution arising from agricultural and animal husbandry activities are difficult to manage but their effect on water quality can be minimised by the use of best practice management such as fencing of streams, management of riparian zones and off-stream watering of stock. Landowners can be encouraged to protect stream banks and provide buffer strips through community awareness programs and by subsidising tree planting and fencing works.

Cooperation with landowners and close collaboration with agricultural agencies are essential for the management of point sources such as dairy effluent and stockyard runoff. Demonstration projects that aim to show the benefits of collecting and using this material are useful.

Table A1.7 *Examples of preventive measures from catchment to consumer*

Source water and catchments	
• Use of an appropriate source water	• Regular inspections of catchment areas
• Ownership and control of catchment area	• Protection of waterways (fencing out livestock, buffer zones, management of riparian zones)
• Designated and limited uses	• Runoff interception
• Registration of chemicals used in catchments	• Use of planning and environmental regulations to regulate potential water polluting developments
• Control of human activities within catchment boundaries	• Use of industry codes of practice and best practice management
• Control of wastewater effluents	
• Involvement in land use planning procedures	
• Participation of community and landowners within the catchment area	
Water extraction and storage systems	
• Control of water extraction	• Prevention of unauthorised access
• Alternate selection of water source	• Destratification of water storage
• Use of available water storage for periods of heavy rainfall	• Diversion of stormwater downstream from intake
• Appropriate location and protection of intake	• Roofed storages and reservoirs with appropriate stormwater collection and drainage
• Proper well construction including casing, sealing and well-head security	• Securing tanks from access by animals
• Proper location of wells in aquifer	• System maintenance
• Water storage systems to maximise detention times	– reservoir cleaning or scouring
• Infiltration wells	– pipeline flushing
• Enclosed water storages	– fittings maintenance
Water treatment system	
• Coagulation or flocculation and sedimentation	• Availability of backup systems
• Alternative treatment	• Water treatment process optimisation, including
• Use of approved water treatment chemicals and materials	– chemical dosing
• Control of water treatment chemicals	– filter backwashing
• Regular assessment of hazards and risks	– flow rate
• Use of skilled and trained operators	– minor infrastructure modifications
• Process controllability of equipment	• Use of tank storage in periods of poor-quality raw water

Table A1.7 *Examples of preventive measures from catchment to consumer (continued)*

Distribution systems	
• Distribution system maintenance • Availability of backup systems (power supply) • Maintaining an adequate disinfectant residual • Cross-connection and backflow prevention devices implemented	• Fully enclosed distribution system and storages • Secondary disinfection • Appropriate repair procedures, including subsequent disinfection of water mains • Maintaining adequate system pressure
Monitoring	
• Quality assurance and validation procedures for sampling and testing	• Calibration and maintenance of equipment
Consumers	
• Information dissemination: – responsibilities relating to drinking water quality – plumbing and appliances – backflow prevention – point of use devices	

Table A1.8 *Estimated removals of enteric pathogens using multiple barriers*

Enteric organisms	Estimated reduction in numbers of enteric pathogens				Estimated overall removal[b]
	Watershed protection	**Reservoir detention**	**Filtration**	**Disinfection[a]**	
Bacteria	0.5–1 log removal	~ 1 log removal per 10 days storage Retention for over 60 days will provide almost complete removal.	0.5–1 log removal	Complete inactivation can be achieved by a range of disinfectants including chlorine, chloramines and UV, provided doses and contact times are sufficient.	Complete removal achievable
Viruses	Complete removal of human enteric viruses if human waste excluded.	1–2 log removal Long-term detention (1–6 months)	Conventional: 2 log removal Direct: 1 log removal Membrane: > 4 log removal	Chlorine, UV light, ozone and chlorine dioxide: 3 log removal	Removal of 5 log achievable
Giardia	0.5–1 log removal	1.5–2.5 log removal Long-term detention (1–6 months)	Conventional: 2.5 log removal Direct: 2 log removal Membrane: > 4 log removal	Chlorine: 1–2 log removal Ozone and chlorine dioxide: 2 log removal	Removal of 5.5–8 log achievable
Cryptosporidium	0.5–1 log removal	1–2 log removal Long-term detention (1–6 months)	Conventional: 2 log removal DAFF: 2 log removal Direct filtration: 2 log removal Membrane: > 4 log removal	Ozone: 0.5–2 log removal Chlorine dioxide: 0.5–1 log removal UV light: 3 log removal Chlorine and chloramines: ineffective	Removal of 3.5–7 log achievable

DAFF = dissolved air flotation and filtration

[a] Log removals based on standard doses and minimum contact times of 30 minutes

[b] Using standard technology (catchment control, detention, conventional filtration, chlorination)

[c] Depending on pore size

A1.7 Critical control points

Appropriate selection of critical control points is an important consideration, as increased focus in process control (monitoring and documentation) for a water supply system will be directed toward these activities and processes. The identity and number of critical control points is system specific and will be determined by the range and magnitude of potential hazards and associated risks. Identification of critical control points may be aided by the use of a decision tree, as shown in Figure A1.2.

Critical control points have several operational requirements, including establishing an appropriate monitoring regime specifying specific parameters and critical limits to ensure the process or activity operates effectively. Failure to meet a critical limit represents loss of control of the process and an unacceptable health risk, either directly, through the supply of unsafe water, or indirectly, where multiple critical control points exist, by exceeding the capacity of subsequent processes. Corrective actions must also be available to re-establish process control when criteria have not been met.

If there is a deviation from a critical limit corrective actions also must be available to reduce the health risk from hazards present in the system.

Summary of actions

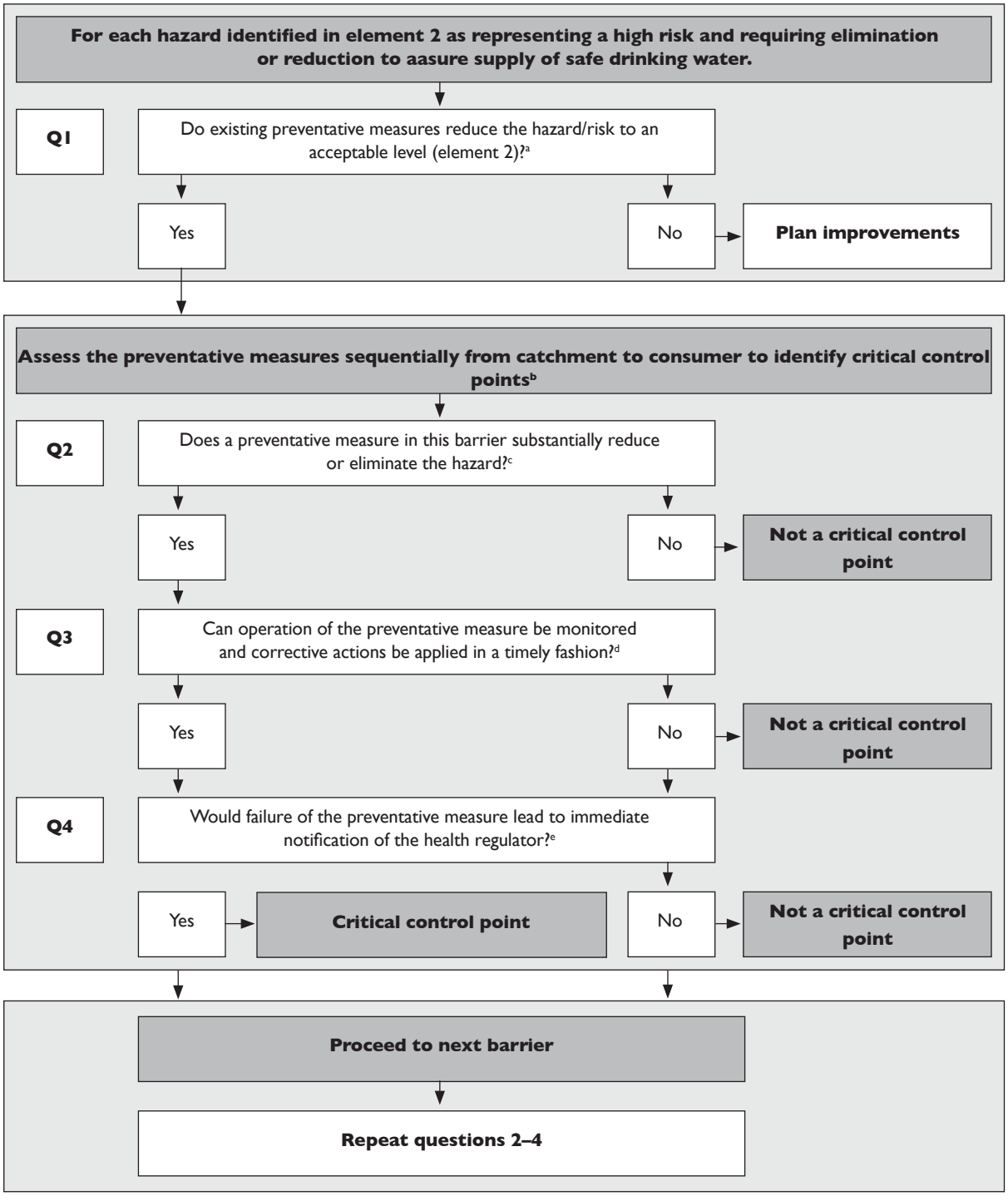
Assess preventive measures from catchment to consumer to identify critical control points.

Establish mechanisms for operational control (see Section 3.4 *Operational procedures and process control*).

Document the critical control points and criteria.

Table A1.9 provides examples of potential sources of Giardia, preventive measures and potential critical control points from catchment to consumer for a river system. Table A1.10 provides further detail on potential critical control points and operational criteria.

Figure A1.2 *Critical control point decision tree*



Notes

a Preventive measures should be applied from catchment to consumer in accordance with the multiple barrier approach. Overall, when considered together, these preventive measures should prevent or reduce the hazard to an acceptable level.

b Drinking water systems generally include numerous preventive measures and strategies that all contribute to assuring the safety of water supplied to consumers; however, only a limited number are amenable to selection as critical control points. The identification of critical control points is system specific and involves making judgments based on knowledge of the potential hazards and associated risks, and the preventive measures. Each significant hazard identified should have a critical control point. However, there may be more than one critical control point to address the same hazard, and more than one hazard may be prevented or reduced by a specific critical control point. Appropriate selection of critical control points is an important consideration because the focus in process control (monitoring and documentation) will increasingly be directed toward these processes and activities. Too many critical control points may make the system unwieldy and too few may fail to provide adequate assurance of drinking water quality.

c Important considerations are that the preventive measure is essential for significantly reducing the given hazard and that the effectiveness of the measure has been validated.

d Operational control must be provided for the preventive measure to assure its ongoing effectiveness. This includes establishing a monitoring regime to ensure the process or activity operates to requirements. Practicalities of monitoring should be considered. Operational parameters and criteria must be monitored with sufficient frequency to guarantee the critical control point is providing protection against targeted hazards and to reveal any failures in a timely fashion. Corrective actions must be available to regain control immediately when criteria have been exceeded or deviated from.

e Failure means deviation from a critical limit not from a target criterion (see Section 3..3.2 and 3.4.2). The significance of critical control points is that failure of the process or activity represents an out-of-control hazard. A practical consideration is whether failure of the process or activity (ie loss of control) will result in a (potentially) unacceptable health risk and require immediate notification of the health regulator.

Table A1.9 *Example preventive measures and potential critical control points for* ***Giardia*** *— river system*

	Potential sources of *Giardia* →	Preventive measures →	Potential critical control points[a]
River/weir	Septic tank effluent	Installation, design and maintenance standards Setback distances	
	Livestock waste	Riparian zones Stocking rate controls Stream fences Flow diversion from reservoir of highly contaminated first-flush water following heavy rainfall	Flow diversion
Resevoir	Human or livestock access	Restrict access Fencing Interception drains Detention	
Treatment plant		Coagulation Sedimentation Filtration	Filtration
Treatment plant		Disinfection, automatic dosing and monitoring	Disinfection
Distribution system	Cross-connections / backflows Mains breaks / new mains	Booster chlorination Cross-connection control Positive pressure Maintenance and repair protocols and procedures	(one or more of the preventive measures)

a Determined using the critical control point decision tree

Table A1.10 *Example — potential critical control points and operational criteria*

Activity/process	Hazard(s)	Critical limit	Monitoring	Corrective action
Groundwater abstraction	Enteric bacteria, viruses and protozoa from septic and livestock waste; nitrates	Physical surety of bore and radius of protection zone. Absence of *E. coli*. NO_3 50 mg/L (as nitrate)	Weekly inspection and testing for presence of *E. coli*. 6-monthly monitoring of nitrate	Repair fault in bore Enforce protection zone
Catchment water reception at take off weir prior to reservoir	Higher levels of enteric bacteria, viruses and protozoa from septic and livestock waste following heavy rainfall	Set flow rate and turbidity limits at location upstream of weir	Continuous stream monitoring station	Divert flow away from reservoir intake
Reservoir mixing/ destratification	Cyanotoxins	6500 cells/mL *Target value: 1000 cells/mL*	Continuous monitoring of temperature and dissolved oxygen through the water column Regular sampling; increase frequency following detection of cyanobacteria or in summer	Dose reservoir with copper sulfate Improve efficiency of mixing Take reservoir out of service
Filtration	Enteric bacteria, viruses and protozoa	Combined filtered water turbidity < 0.5 NTU 95% of time. Maximum 5 NTU *Target value: < 0.3 NTU at all times*	Continuous online monitoring	Identify problem and take action eg repair faulty operation Increase coagulant dose Filter backwash
Primary disinfection and storage	Enteric bacteria, viruses and *Giardia*	Free chlorine residual > 1 mg/L Detention >x (to set minimum C.*t*)[a]	Continuous online monitoring and alarms with automatic feedback to chlorine dosing Flow not to exceed x ML per hr	Increase chlorine dose Decrease flows to increase detention time Stop supply
Secondary disinfection	Enteric and free-living bacteria	Free chlorine residual >1 mg/L	Continuous online monitoring and alarms with automatic feedback to chlorine dosing	Increase chlorine dose Stop supply
Distribution of treated water	Enteric bacteria, viruses and protozoa Chemical contaminants	Minimum free chlorine residual of 0.2 mg/L at specified locations Positive pressure at specified locations	Continuous online monitoring Hydraulic pressure	Increase chlorine dose Identify and repair source of pressure loss

C.*t* = contact time; *E. coli* = *Escherichia coli*; NTU = nephelometric turbidity unit

a see Section A8 *Chlorination as an example of a critical control point*

A1.8 Chlorination as an example of a critical control point

Disinfection is designed to kill pathogenic microorganisms, thereby preventing waterborne diseases. Chlorination is the most commonly used process for disinfection; it is effective in killing bacteria and can be reasonably effective in inactivating viruses (depending on type) and most protozoa, including *Giardia*. *Cryptosporidium* is not inactivated by the concentrations of chlorine that can be safely used in drinking water.

Although the microbial quality of drinking water is of primary importance and must never be compromised, chlorine levels and the formation of chlorination byproducts should be controlled to prevent any adverse health effects that may eventually be found to be attributable to disinfection byproducts.

The effectiveness of chlorination depends on several factors, including:

chlorine dose

contact time between chlorine and the water

chlorine demand

pH

temperature

turbidity.

Chlorine demand is important because it is the chlorine residual in the water and not the chlorine dose that determines the efficacy of chlorination. Natural water contains inorganic and organic compounds that react with chlorine. Reactions with naturally occurring organic matter produce chlorination byproducts, the most well known being the trihalomethanes. Chlorine may also react with compounds such as phenols to impart a taste and odour to water.

A sufficient chlorine dose must therefore be added to the water to allow for the chlorine demand reactions to occur, and to ensure that there is an adequate free chlorine residual available to disinfect the water effectively. Turbidity should be reduced as much as possible before the addition of the disinfectant in order to decrease the chlorine demand, limit shielding of microorganisms in particles and reduce the formation potential of chlorination byproducts.

Chlorination fulfils the requirements of a critical control point. The effectiveness of eliminating potentially harmful microorganisms is validated by extensive research and technical literature (eg see USEPA 1999). In addition, process control measures are readily available. Chlorination must be functional and effective at all times, as even short periods of suboptimal performance can represent a serious risk to public health.

Table A1.11 and the following text provide a summary of the chlorination process as a critical control point.

Table A1.11 *Chlorination as a critical control point*

Hazards

Enteric bacteria, viruses and *Giardia*

Process controls

- Chlorine dosing system
- Plant flow rate / operation of clear well storage
- pH adjustment
- Chlorine cylinder changeover
- Backup power / duplicate facilities

Operational monitoring

Parameter	*Target criteria*	*Critical limits*	*Monitoring methods*
Chlorine residual	> 0.5 mg/L	Specific low chlorine residual set to achieve a minimum C.*t* requirement based on maximum flow and minimum storage times. Time is an important factor in determining the critical limit eg if there is a filtered water storage prior to supply to customers an interruption to chlorination of up to several hours may not result in the C.*t* value falling below the minimum limit.	Online, continuous chlorine residual analyser, flow and pH 24-hour monitored alarms on residual monitoring, pH and chlorine dosing equipment Regular turbidity and temperature monitoring, and chlorine demand calculations. Increase frequency on changing water quality Appropriate electronic or hard copy monitoring records
pH	pH 6.5–7.5		
Flow rate	Set to achieve minimum contact time		
Chlorine dose	Set points ± x%		
Turbidity	< 1.0 NTU		
Temperature			

Corrective action

Any breach in critical limits or target criteria should result in any of the following operating procedures as necessary:

- inspect and calibrate equipment
- adjust flow rate
- adjust chlorine dose or feed point
- carry out additional monitoring, increase sampling and testing
- recalculate C.*t* values
- implement unplanned maintenance procedure
- secondary or booster disinfection
- use alternative supply or divert water
- engage backup equipment
- plant automatic shutdown
- implement emergency response
- record actions to be taken and report (internally or externally as required).

Verification

- Calibration and maintenance of equipment
- Drinking water quality monitoring
- Consumer satisfaction
- Evaluation and audit

PROCESS CONTROLS

Effective operation of chlorination requires consideration of several associated process control measures. These include:

Chlorine dosing system, ideally with flow-proportional automatic dosing and feedback loops to achieve target chlorine residual and provide rapid responses to any changes in flow and water quality. Flow meters and alarms should be provided on the chlorine feed system to warn of disinfectant loss.

Plant flow-rate control and the design and operation of the clear-well or post-treatment reservoir (whichever is used to provide an adequate contact time). The infrastructure for chlorination should be of sufficient capacity to handle maximum flow rates and should not be hydraulically overloaded or subjected to rapid changes in hydraulic loading, as these conditions will compromise its effectiveness.

pH adjustment for supplies where sudden large changes of pH are known to occur (eg due to problems arising from chemical dosing with lime, permanganate, caustic soda etc).

Provision of an alarm system on the chlorine supply, to indicate when the supply is running low, and of a spare or surplus chlorine supply. Chemical suppliers should be evaluated and selected on their ability to supply product in accordance with required specifications.

Inspection, calibration and maintenance of equipment to ensure continuing process capability and accuracy of monitoring results.

Emergency measures such as backup generators, alarms and duplicate facilities (eg chlorinator, disinfectant feed system, pumps, monitoring equipment etc) to avoid loss of disinfection if failure occurs.

OPERATIONAL MONITORING

Operational parameters

It is essential to monitor residual chlorine concentration, flow rate (contact time), chlorine dose, pH, temperature and turbidity to determine whether water is being disinfected properly. Total coliforms and heterotrophic bacteria can also be used.

For processes such as disinfection, where failure can result in a rapid change in water quality and pose a significant health risk, monitoring should be online and continuous to provide an immediate indication of performance. Flow measurement and chlorine residual can be monitored online and continuously with feedback loops to ensure correct conditions are met. For supplies where sudden changes of pH are known to occur, continuous monitoring of this parameter should also be considered. Alarm systems that are monitored 24 hours a day should be installed to indicate when operational criteria have not been met.

Critical limits and target criteria

Operational criteria for chlorination are normally determined by calculating the C.*t* values required to attain target levels of pathogen inactivation at specified temperatures and pH. C.*t* is the product of residual chlorine concentration in mg/L and the contact time in minutes.

Free chlorine residuals and C.*t* values should be validated for individual water supplies.Tables of C.*t* values for various temperatures and pHs for the inactivation of *Giardia* and viruses by free chlorine and other disinfectants have been published by the United States Environmental Protection Agency (eg see Table A1.12 and USEPA 1999).

Ongoing compliance with minimum C.*t* values should be confirmed.

***Table A1.12** C.t values for inactivation by free chlorine (mg.min/L)*

		99% (2 log) inactivation		99.9% (3 log) inactivation	
	pH	**10°C**	**20°C**	**10°C**	**20°C**
Giardia[a]	7.0	75	37	112	56
	8.0	108	41	162	81
Viruses	6.0–9.0	3	1	4	2

Source: USEPA (1999). Disinfection Profiling and Benchmarking Guidance Manual, EPA 815-R-99-013.
a At a free chlorine residual of 1 mg/L

Corrective action

Corrective action taken in response to target criteria or critical limits not being met could include:

examination of the chlorination process (investigate equipment)

adjustment of flow rate to increase detention time

adjustment of pH

recalculation of C.*t* values

adjustment of disinfectant dose rates

variation of the disinfection application point

verification of chlorine dose solution

increased sampling, verification of operational monitoring

inspection and calibration of equipment

engagement of backup chlorination equipment

secondary disinfection, spot dose or booster disinfection

water diversion or reliance on alternate supply (storage)

shutdown of plant, automatic immediate shutdown

implementation of an emergency response plan (eg issuing advice to boil water).

VERIFICATION

The chlorination process should be verified by supplementing with:

regular calibration and maintenance of the chlorine dose and monitoring equipment to ensure continuing process capability and accuracy of monitoring results. Procedures, schedules, responsibilities and records (maintenance logs) for the calibration and maintenance of equipment should be documented

routine sampling and testing of *E. coli* (or thermotolerant coliforms) in the distribution system and as supplied to consumers

monitoring of consumer comments and complaints regarding chlorine taste and odour

performance evaluation and operational audit to confirm that objectives are being met. This entails the periodic review of operational monitoring, drinking water quality monitoring data and consumer satisfaction, logbook records of planned and unplanned maintenance and calibration, and operating procedures.

Appendix 2: Further sources of information on drinking water quality management

A2.1 Drinking water quality management — general

Agriculture and Resource Management Council of Australia and New Zealand/Australian and New Zealand Environment and Conservation Council (1994). *National water quality management strategy: water quality management — an outline of policies.* Available at: www.affa.gov.au/docs/nrm/water/water_reform/nwqms/publist.html.

Agriculture and Resource Management Council of Australia and New Zealand/Australian and New Zealand Environment and Conservation Council (1994). *National water quality management strategy: policies and principles — a reference document.*

Agriculture and Resource Management Council of Australia and New Zealand/Australian and New Zealand Environment and Conservation Council (1998). *National water quality management strategy: implementation guidelines.* Available at: www.affa.gov.au/docs/nrm/water/water_reform/nwqms/publist.html.

American Water Works Association (1999). *Water quality and treatment: A handbook of community water supplies.* 5th edition. Ed. Letterman RD. McGraw-Hill.

American Water Works Association Research Foundation/International Water Supply Association (1998). *Treatment process selection for particle removal.* Ed. Brock McEwen J. AWWARF/IWSA.

Bannister R, O'Connor N and Stivers C (2000). Managing water quality risks from catchment to tap. *Water,* March/April: 46–50.

Clancy JL (2000). Sydney's 1998 water quality crisis. *Journal of the American Water Works Association,* 92(3): 55–66.

Clancy JL (2001). Lessons from the 1998 Sydney water crisis. *Water,* 28(1): 33–36.

Davison A and Deere D (1999). Safety on tap. *Microbiology Australia,*May: 28–31

Deere D and Davison A (1998). Safe drinking water: are food guidelines the answer. *Water,* November/December: 21–24.

Deere D and Davison A (1999). Assuring Water Quality. *Water,* July/August:8–9.

Government of British Columbia. *Drinking water protection plan — a discussion document.* Available at: www.env.gov.bc.ca/wat/wq/dw/.

Government of Western Australia (2000). *State water quality management strategy.* Available at: www.wrc.wa.gov.au/protect/SWQMS_final.pdf.

Hrudey SE (2001). Drinking water quality: a risk management approach. *Water, 28(1)*: 29–32.

MacKenzie WR, Hoxie NJ, Proctor ME et al. (1994). A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *New England Journal of Medicine,* 331(3): 161–167.

Nadebaum PR, Adlem LM, Baker AJ, Chapman MR and Rizak S. (2000). Improved management of drinking water quality. *Water,* July/August: 12–16

National Health and Medical Research Council/Agriculture and Resource Management Council of Australia and New Zealand. (1996). *Australian Drinking Water Guidelines*. NHMRC/ARMCANZ. Available at: www.nhmrc.health.gov.au/publicat/synopses/eh19syn.htm.

National Water Quality Management Strategy. Information available at: www.affa.gov.au/docs/nrm/water/water_reform/nwqms/nwqms_toc.html.

New Zealand Ministry of Health (2000). *Drinking water standards for New Zealand 2000*. Available at: www.moh.govt.nz/moh.nsf/wpg_Index/Publications-Index.

New Zealand Ministry of Health (1995). *Guidelines for drinking water quality management for New Zealand*.

Taylor MEU (2000). Drinking water management in New Zealand. *Water,* 27(6): 36–40.

Walkerton, Canada inquiry, available at: www.walkertoninquiry.com

World Health Organization (1993). *Guidelines for Drinking water Quality*. WHO.

World Health Organization (1999). *Toxic cyanobacteria in water: a guide to their public health consequences, monitoring and management*. Eds. Chorus I, Bartram E and Spon FN.

A2.2 Catchment management and source water protection

Australian Water and Wastewater Association (1999). Catchment Management Workshop Proceedings. Adelaide.

Source water protection. Further references available at: www.awwa.org/unitdocs/referenc.htm#ground and www.epa.gov/safewater/protect.html.

United States Environmental Protection Authority (1999). *Protecting sources of drinking water: selected case studies in watershed management*. USEPA, Washington DC. Available at: www.epa.gov/safewater.

United States Environmental Protection Authority. Source Water Assessment Program. Information available at: www.epa.gov/safewater/protect/swap.html.

United States Environmental Protection Authority (1999). *Guidance manual for conducting sanitary surveys of public water supply systems; surface water and groundwater under the direct influence (GWUDI)*. Available at: www.epa.gov/safewater/mdbp/pdf/sansurv/sansurv.pdf.

A2.3 Groundwater protection

Agriculture and Resource Management Council of Australia and New Zealand/Australian and New Zealand Environment and Conservation Council (1995). *National Water Quality Management Strategy: Guidelines for Groundwater Protection in Australia*.

A2.4 Risk assessment and management

AS/NZS 3931 (Australian Standard/New Zealand Standard) (1998). *Risk analysis of technological systems — application guide*. Standards Australia/Standards New Zealand.

AS/NZS 4360(Australian Standard / New Zealand Standard) (1999). *Risk management*. Standards Australia/Standards New Zealand.

AS/NZS ISO 14001 (Australian Standard/New Zealand Standard, International Organization for Standardization) (1996). *Environmental management systems — specification with guidance for use*. Standards Australia/Standards New Zealand.

AS/NZS ISO 14004 (Australian Standard/New Zealand Standard, International Organization for Standardization) (1996). *Environmental management systems — general guidelines on principles, systems and supporting techniques*. Standards Australia/Standards New Zealand.

Bannister R, O'Connor N and Stivers C (2000). Managing water quality risks from catchment to tap. *Water,* March/April: 46–50.

enHealth Council (2000). *Environmental health risk assessment*. Public consultation draft. Available at: www.health.gov.au/pubhlth/strateg/envhlth/risk/risk_draft.pdf.

Hrudey S (2001). Drinking water quality: a risk management approach. *Water, 28(1)*: 29–32.

New Zealand Ministry of Health (2000). *Draft model public health risk management plans for drinking water supplies*. Available at: www.moh.govt.nz/moh.nsf/wpg_Index/Publications-Index.

United States Environmental Protection Authority (1999). *Guidance manual for conducting sanitary surveys of public water supply systems; surface water and groundwater under the direct influence (GWUDI)*. Available at: www.epa.gov/safewater/mdbp/pdf/sansurv/sansurv.pdf.

A2.5 System analysis and management/process control and optimisation

American Water Works Association (1999). *Water quality and treatment: a handbook of community water supplies*. 5th edition. Ed. Letterman RD. McGraw-Hill.

American Water Works Association Research Foundation/International Water Supply Association (1998). *Treatment process selection for particle removal*. Ed. Brock McEwen J. American Water Works Association Research Foundation /IWSA.

Clancy J, Hargy T, Marshall M and Dyksen J (1998). UV light inactivation of *Cryptosporidium* oocysts. *Journal of the American Water Works Association,* September: 92–102

Cleasby JL, Dharmarajah AH, Sindt GL and Baumann ER (1989). Design and operation guidelines for optimization of the high-rate filtration process: plant survey results. American Water Works Association Research Foundation/American Water Works Association.

Cleasby JL, Sindt GL, Watson DA and Baumann ER (1992). Design and operation guidelines for optimization of the high-rate filtration process: plant demonstration studies. American Water Works Association Research Foundation/American Water Works Association.

Cleasby JL, Williamson MM and Baumann ER (1963). Effect of filtration rate changes on quality. *Journal of the American Water Works Association,* 55(7): 869–878

Cohen YK. (1999). Water quality program defines operations. *Opflow,* 25(10): 1,4–5. American Water Works Association.

Colwell M and Mosse PRL (2000). Optimisation of filter function using particle counting. *Water,* November/December: 33–35.

Cornwell DA and Lee RG (1993). *Recycle stream effects on water treatment*. American Water Works Association Research Foundation/American Water Works Association.

Cromwell J, Owen D, Dysken J and Means E (1997). *Managerial assessment of water quality and system reliability*. American Water Works Association Research Foundation/American Water Works Association.

Department of Land and Water Conservation, New South Wales (1999). *The management of Giardia and Cryptosporidium in town water supplies: protocols for local government councils*.

Health and Welfare Canada (1993). *Water treatment principles and applications: a manual for the production of drinking water*. (Guidelines for Canadian Drinking Water Quality).

Hudson HE and Singley JE (1974). Jar testing and utilization of jar testing data. In *Upgrading existing*

water treatment plant. American Water Works Association Seminar Proceedings. AWWA.

Murray BA (1995). Particle counting in water treatment. *Water,* 22(2).

Partnership for Safe Water Program. American Water Works Association. Information available at: www.awwa.org/partner/partner1.htm and www.epa.gov/ogwdw000/psw/psw.html.

Peterson T (2000). Process optimization and the OandM Manual. *Opflow,* June: 17–19.

Pizzi N (1996). Optimizing your plant's filter performance. *Opflow,* 22(5): 1,4,5.

Renner RC and Hegg BA (1997). *Self-assessment guide for surface water treatment plant optimization.* American Water Works Association Research Foundation/American Water Works Association.

Renner RC, Hegg BA, Bender JH and Bissonette EM (1993). Composite correction program optimizes performance at water plants. *Journal of the American Water Works Association,* June: 67–74.

Trussell RR (1999). Safeguarding distribution system integrity. *Journal of the American Water Works Association,* January: 46–54.

United States Environmental Protection Authority (1989). *Guidance manual for compliance with the filtration and disinfection requirements for public water systems using surface water sources.* USEPA, Washington DC.

United States Environmental Protection Authority (1999). *Guidance manual for conducting sanitary surveys of public water supply systems; surface water and groundwater under the direct influence (GWUDI).* Available at: www.epa.gov/safewater/mdbp/pdf/sansurv/sansurv.pdf.

United States Environmental Protection Authority (1999). *Alternative disinfectants and oxidants guidance manual.* Available at: www.epa.gov/safewater/mdbp/pdf/alter/cover_al.pdf.

World Health Organization (1999). *Toxic Cyanobacteria in water: a guide to their public health consequences, monitoring and management.* Eds. Chorus I, Bartram E and Spon FN.

A2.6 Monitoring and verification

Allen MJ, Clancy JL and Rice EW (2000). The plain hard truth about pathogen monitoring. *Journal of the American Water Works Association,* 92(9): 64–76.

Clancy JL (2000). Sydney's 1998 water quality crisis. *Journal of the American Water Works Association,* 92(3): 55–66.

Clancy JL (2001). Lessons from the 1998 Sydney water crisis. *Water,* 28(1): 33–36.

Davison A and Deere D (1999). Safety on tap. *Microbiology Australia,* May: 28–31

Deere D and Davison A (1998). Safe drinking water: are food guidelines the answer. *Water,* November/December: 21–24.

Deere D and Davison A (1999). Assuring water quality. *Water 21,* July/August: 8–9.

Fairley CK, Sinclair MI and Rizak S (1999). Monitoring drinking water: the receding zero. *Medical Journal of Australia,* 171: 397–398.

Fairley CK, Sinclair MI and Rizak S (1999). Monitoring not the answer to cryptosporidium in water. *The Lancet,* 354(9183): 967–969.

Murray BA (1995). Particle counting in water treatment. *Water,* 22(2).

New Zealand Ministry of Health (2000). *Drinking water standards for New Zealand 2000.* Available at: www.moh.govt.nz/moh.nsf/wpg_Index/Publications-Index.

A2.7 Materials and chemicals

AS/NZS 4020 ((Australian Standard/New Zealand Standard) (1999). *Products for use in contact with drinking water.* Standards Australia/Standards New Zealand.

Mosse PRL and Colwell M (2001). Chemical quality assurance — Gippsland water's journey: getting the right chemical in the right place at the right time. *Water,* 28(3): 45–46.

SAA HB131 (1999). *Guidelines for applying and interpreting AS/NZS 4020: Products for use in contact with drinking water.* Standards Australia.

A2.8 Incident and emergency management

American Water Works Association (2001). *Emergency planning for water utilities. manual of water supply practices.* M19. 4th Edition. American Water Works Association, Denver.

Clancy JL (2000). Sydney's 1998 water quality crisis. *Journal of the American Water Works Association,* 92(3): 55–66.

Clancy JL (2001). Lessons from the 1998 Sydney water crisis. *Water,* 28(1): 33–36.

A2.9 Employee training and awareness

DeNileon GP (2000). Certification ensures system integrity, process control. *Opflow,* 26(3): 7,8,14.

Truss Jr. CL (2000). The future of operator training. *Journal of the American Water Works Association,* 92(1): 80–81.

A2.10 Research and development

Hudson HE and Singley JE (1974). Jar testing and utilization of jar testing data. In *Upgrading existing water treatment plant.* American Water Works Association Seminar Proceedings. AWWA.

A2.11 Documentation and reporting

AS/NZS ISO 10013 (Australian Standard/New Zealand Standard, International Organization for Standardization) (1996). *Guidelines for developing quality manuals.* Standards Australia/Standards New Zealand.

Cohen YK (1999). Water quality program defines operations. *Opflow,* 25(10): 1,4–5. American Water Works Association.

Jordan CG (1998). Water quality reports boost consumer confidence. *Journal of the American Water Works Association,* 90(1): 40–43.

Peterson T (2000). Process optimization and the OandM Manual. *Opflow,* June: 17–19.

A2.12 Community consultation and communication

Jordan CG (1998). Water quality reports boost consumer confidence. *Journal of the American Water Works Association,* 90(1): 40–43.

Krestensen C (1990). *Common Pitfalls and Oversights in Government Consultation with Consumer Groups*. Consumers' Health Forum of Australia, Canberra.

Maywald S (1989). *Consulting with your Community: A guide to effective and equitable community consultation techniques for local government and associated organisations*. Local Government Community Services Association.

South Australian Health and Social Welfare Councils (1991). *Consultation principles and consultation protocol*.

Whately R. *Community communication programs — a seven step guide*. Major Urban Water Authorities of Australia.

A2.13 Hazard analysis and critical control point (HACCP)

Codex Alimentarius Commission (CAC) (1997). Hazard analysis and critical control point (HACCP) systems and guidelines for its application. Annex to CAC/RCP 1-1969, Rev 3. Available at: ftp://ftp.fao.org/codex/standard/fh_basic.pdf.

Davison A and Deere D (1999). Safety on tap. *Microbiology Australia,* May: 28–31.

Deere D and Davison A (1998). Safe drinking water: are food guidelines the answer. *Water,* November/December: 21–24.

Deere D and Davison A (1999). Assuring water quality. *Water 21,* July/August: 8–9.

Dickinson G (1997). *HACCP (Hazard Analysis Critical Control Point): A Food Safety Plan*. Royal Melbourne Institute of Technology, Department of Applied and Environmental Sciences.

Gray R and Morain M (2000). HACCP application to Brisbane Water. *Water,* January/February: 41–42.

Havelaar AH (1994). Application of HACCP to drinking water. *Food Control,* 5(3): 145–152.

HB 90.5 (2000). *Correlation Between ISO 9001:2000 and the HACCP Principles*. Standards Australia.

Jay JM (2000). The HACCP system and food safety. In *Modern Food Microbiology,* 6th edition, chapter 21: 407–422. Aspen Publishers.

Nadebaum P, Chapman M, Baker A and Rizak, S (2001). Application of HACCP to drinking water supply systems. Paper presented at the Annual Conference of the Australian Water Association, Canberra, 1–5 April.

National Advisory Committee on Microbiological Criteria for Foods (1992). *Hazard analysis and critical control point system*. NACMCF, Washington DC:

National Advisory Committee on Microbiological Criteria for Foods (1997). *Hazard analysis and critical control point principles and application guidelines*. Washington DC.

Tompkin RB (1992). Corrective action procedures for deviations from the critical control point critical limits. In *HACCP — principles and applications*, eds. Pierson MD and Corlett Jr DA: 72–82. Chapman and Hall.

World Health Organization (1997). HACCP — Introducing the hazard analysis and critical control point system (WHO/FSF/FOS/97.2). Available at: www.who.int/fsf/Codex/IntroducingHACCP.pdf.

A2.14 Quality management/continuous improvement

Anon (1999). Getting it together: integrated management systems. *Waste Management and Environment,* 10(5): 19–20.

AS/NZS 4581 (1999). *Management system integration — guidance to business, government and community organizations*. Standards Australia/Standards New Zealand.

AS/NZS 8402 (1994). *Quality management and quality assurance — vocabulary*. Standards Australia/Standards New Zealand.

AS/NZS ISO 9000.2 (1998). *Quality management and quality assurance standards. Part 2: Generic Guidelines for the Application of ISO 9001, ISO 9002 and ISO 9003*. Standards Australia/Standards New Zealand.

AS/NZS ISO 9001 (2000). *Quality management systems — requirements*. Standards Australia/Standards New Zealand.

AS/NZS ISO 9004 (2000). *Quality management systems — guidelines for performance improvement*. Standards Australia/Standards New Zealand.

AS/NZS ISO 10013 (1996). *Guidelines for developing quality manuals*. Standards Australia/Standards New Zealand.

AS/NZS ISO 14001 (1996). *Environmental management systems — specification with guidance for use*. Standards Australia/Standards New Zealand.

AS/NZS ISO 14004 (1996). *Environmental management systems — general guidelines on principles, systems and supporting techniques*. Standards Australia/Standards New Zealand.

HB 90.5-2000. *Correlation between ISO 9001:2000 and the HACCP principles*. Standards Australia.

Hill M, Deere D and Hansen G (2000). Excellence through continuous improvement: connectaflow. *Water,* January/February: 43–44.

Nestel G, et al. (1997). *Guidance for self-assessment and peer review for the AWWA Qualserve program*. American Water Works Association Research Foundation/American Water Works Association.

Pearson TR (2000). Embracing total quality management. *Journal of the American Water Works Association,* 92(1): 52–53.

Responsible Care report. Available at: www.ccpa.ca/english/.

A2.15 Reference web sites

American Water Works Association	www.awwa.org
Australian Water Association	www.awa.asn.au
AWWA Research Foundation	www.awwarf.com
International Water Association	www.iawq.org.uk
National Health and Medical Research Council	www.nhmrc.gov.au
New Zealand Ministry of Health	www.moh.govt.nz
United States Environmental Protection Agency	www.epa.gov
USEPA Office of Groundwater and Drinking Water	www.epa.gov/safewater
Water and Rivers Commission (Western Australia)	www.wrc.wa.gov.au
Water Environment Federation	www.wef.org
World Health Organization	www.who.int/home-page

GLOSSARY

Glossary

acute reference dose (ARfD):	an estimate of the amount a substance in food or drinking water, normally expressed on a body-weight basis, that can be ingested in a period of 24 hours or less without appreciable health risks to the consumer, on the basis of all known facts at the time of the evaluation.
ADWG:	***Australian Drinking Water Guidelines***, published by the National Health and Medical Research Council (NHMRC).
biofilm:	microbial populations that grow on the inside of pipes and other surfaces.
benchmark dose (BMD)	an exposure due to a dose of a substance associated with a specified low incidence of risk, generally in the range of 1% to 10%, of a health effect; or the dose associated with a specified measure or change of a biological effect.
***Campylobacter*:**	a group of bacteria that is a major cause of diarrhoeal illness.
catchment:	area of land that collects rainfall and contributes to surface water (streams, rivers, wetlands) or to groundwater.
chlorination:	use of chlorine as a means of disinfection.
chloramination:	use of chloramines (compounds formed by the reaction of hypochlorous acid or aqueous chlorine with ammonia) as a means of disinfection.
chlorine demand:	the difference between the amount of chlorine added to water and the amount of residual chlorine remaining after a given contact time. Chlorine demand may change with dosage, time, temperature, pH, and the nature and amount of any impurities in the water.
coagulation:	clumping together of very fine particles into larger particles using chemicals (coagulants) that neutralise the electrical charges of the fine particles and destabilise the particles.
Codex Alimentarius:	a food quality and safety code developed by the Codex Alimentarius Commission of the Food and Agriculture Organization of the United Nations and the World Health Organization.
coliform bacteria:	group of bacteria whose presence in drinking water can be used as an indicator for operational monitoring.
consumer:	an individual or organisation that uses drinking water.
corrective action:	procedures to be followed when monitoring results indicate a deviation occurs from acceptable criteria (adapted from Codex Alimentarius).
critical control point:	a point, step or procedure at which control can be applied and which is essential to prevent or eliminate a hazard or reduce it to an acceptable level (adapted from Codex Alimentarius).
critical limit:	a prescribed tolerance that must be met to ensure that a critical control point effectively controls a potential health hazard; a criterion that separates acceptability from unacceptability (adapted from Codex Alimentarius).
***Cryptosporidium*:**	microorganism commonly found in lakes and rivers that is highly resistant to disinfection. Cryptosporidium has caused several large outbreaks of gastrointestinal illness, with symptoms that include diarrhoea, nausea and stomach cramps. People with severely weakened immune systems (ie severely immunocompromised people) are likely to have more severe and more persistent symptoms than healthy individuals (adapted from United States Environmental Protection Agency).

C.*t*:	the product of residual disinfectant concentration (C) in milligrams per litre determined before or at taps providing water for human consumption, and the corresponding disinfectant contact time (*t*) in minutes.
cyanobacteria:	bacteria containing chlorophyll and phycobilins, commonly known as 'blue-green algae'.
destratification:	agitation of water body to break up and mix otherwise stable layers of water.
disinfectant:	an oxidising agent (eg chlorine, chlorine dioxide, chloramines and ozone) that is added to water in any part of the treatment or distribution process and is intended to kill or inactivate pathogenic (disease-causing) microorganisms.
disinfectant residual:	the amount of free and/or available disinfectant remaining after a given contact time under specified conditions.
disinfection:	the process designed to kill most microorganisms in water, including essentially all pathogenic (disease-causing) bacteria. There are several ways to disinfect, with chlorine being most frequently used in water treatment.
disinfection byproduct:	products of reactions between disinfectants, particularly chlorine, and naturally occurring organic material.
distribution system:	a network of pipes leading from a treatment plant to customers' plumbing systems.
dose–response:	the quantitative relationship between the dose of an agent and an effect caused by the agent.
drinking water:	water intended primarily for human consumption (but excluding bottled water, for the purposes of these guidelines).
drinking water quality management audit:	the systematic and documented evaluation of activities and processes to confirm that objectives are being met, and which includes an assessment of management system implementation and capability.
drinking water quality monitoring:	the wide-ranging assessment of the quality of water in the distribution system and as supplied to the consumer, which includes the regular sampling and testing performed for assessing conformance with guideline values and compliance with regulatory requirements and agreed levels of service.
drinking water supplier:	an organisation, agency or company that has responsibility and authority for treating and/or supplying drinking water.
drinking water supply system (water supply system):	all aspects from the point of collection of water to the consumer (can include catchments, groundwater systems, source waters, storage reservoirs and intakes, treatment systems, service reservoirs and distribution systems, and consumers).
enteric pathogen:	pathogen found in the gut.
epidemiology:	the study of the distribution and determinants of health/disease states in human populations.
***Escherichia coli*:**	bacterium found in the gut, used as an indicator of faecal contamination of water.
eucaryote:	organism with a defined nucleus (animals, plants and fungi, but not bacteria or cyanobacteria).
eutrophication:	degradation of water quality due to enrichment by nutrients such as nitrogen and phosphorus, resulting in excessive algal growth and decay and often low dissolved oxygen in the water.

exposure:	contact of a chemical, physical or biological agent with the outer boundary of an organism (eg through inhalation, ingestion or dermal contact).
exposure assessment:	the estimation (qualitative or quantitative) of the magnitude, frequency, duration, route and extent of exposure to one or more contaminated media.
filtration:	process in which particulate matter in water is removed by passage through porous media.
flocculation:	process in which small particles are agglomerated into larger particles (which can settle more easily) through gentle stirring by hydraulic or mechanical means.
***Giardia lamblia*:**	A protozoan frequently found in rivers and lakes. If water containing infectious cysts of ***Giardia*** is ingested, the protozoan can cause a severe gastrointestinal disease called giardiasis.
grab sample:	single sample collected at a particular time and place that represents the composition of the water only at that time and place.
groundwater:	water contained in rocks or subsoil.
guideline value:	the concentration or measure of a water quality characteristic that, based on present knowledge, either does not result in any significant risk to the health of the consumer (health-related guideline value), or is associated with good quality water (aesthetic guideline value).
hazard:	a biological, chemical, physical or radiological agent that has the potential to cause harm.
hazard analysis critical control point (HACCP) system:	a systematic methodology to control safety hazards in a process by applying a two-part technique: first, an analysis that identifies hazards and their severity and likelihood of occurrence; and second, identification of critical control points and their monitoring criteria to establish controls that will reduce, prevent, or eliminate the identified hazards.
hazard control:	the application or implementation of preventive measures that can be used to control identified hazards.
hazard identification:	the process of recognising that a hazard exists and defining its characteristics (AS/NZS 3931:1998).
hazardous event:	an incident or situation that can lead to the presence of a hazard (what can happen and how).
helminth:	a worm-like invertebrate of the order Helminthes.
heterotrophic bacteria:	bacteria that use organic matter synthesised by other organisms for energy and growth.
heterotrophic plate count (HPC):	the number of colonies of heterotrophic bacteria grown on selected solid media at a given temperature and incubation period, usually expressed in number of bacteria per millilitre of sample.
integrated catchment management:	the coordinated planning, use and management of water, land, vegetation and other natural resources on a river or groundwater catchment, based on cooperation between community groups and government agencies to consider all aspects of catchment management.

ISO 9001:2000 (Quality Management):	an international accredited standard that provides a generic framework for quality management systems. Designed to assure conformance to specified requirements by a supplier at all stages during the design, development, production, installation, and servicing of a product, it sets out the requirements needed to achieve an organisation's aims with respect to guaranteeing a consistent end product.
ISO 14001:1996 (Environmental Management Systems):	an international accredited standard that provides a generic framework for guidance on the development and implementation of an environmental management system to minimise the impacts of business operations on the environment and to foster environmental sustainability.
indicator:	a specific contaminant, group of contaminants or constituent that signals the presence of something else (eg ***Escherichia coli*** indicate the presence of pathogenic bacteria).
indicator organisms:	microorganisms whose presence is indicative of pollution or of more harmful microorganisms.
jar test:	a laboratory procedure used to estimate the minimum or ideal coagulant dose required to achieve certain water quality goals. A jar test simulates a water treatment plant's coagulation and flocculation units with differing chemical doses, and mixing and settling times
limit of detection (LOD):	is normally used to indicate the lowest level that can be reliably detected
limit of quantitation (LOQ):	the minimum concentration of a substance that can be accurately quantified within a specified degree of confidence; often somewhat higher than the limit of detection
limit of reporting (LOR):	the minimum concentration of a chemical used for reporting purposes. Results of analyses lower than the LOR are considered to be of lesser reliability and thus may be omitted from reported data
log removal:	used in reference to the physical–chemical treatment of water to remove, kill, or inactivate microorganisms such as bacteria, protozoa and viruses (1-log removal = 90 per cent reduction in density of the target organism, 2-log removal = 99 per cent reduction, 3-log removal = 99.9 per cent reduction, etc)
lowest-observed-adverse-effect level (LOAEL)	the lowest exposure level of a chemical substance that causes statistically and biologically significant adverse differences in test samples as compared to other samples not subjected to the chemical substance
lowest-observed-effect level (LOEL)	the lowest exposure level of a chemical substance that causes statistically and biologically significant differences (adverse or otherwise) in test samples as compared to other samples not subjected to the chemical substance
maximum risk:	risk in the absence of preventive measures.
microorganism:	organism too small to be visible to the naked eye. Bacteria, viruses, protozoa, and some fungi and algae are microorganisms.
multiple barriers:	use of more than one preventive measure as a barrier against hazards.
***Naegleria fowleri*:**	an amoeba that causes a form of meningitis.
nephelometric turbidity unit (NTU):	a measure of turbidity.

no-observed-adverse-effect level (NOAEL)	an exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed and control populations
no-observed-effect level (NOEL)	an exposure level at which there are no statistically or biological significant differences in the frequency or severity of any effect (adverse or otherwise) between in the exposed and control populations
operational monitoring:	the planned sequence of measurements and observations used to assess and confirm that individual barriers and preventive strategies for controlling hazards are functioning properly and effectively.
particle count:	the results of a microscopic examination of treated water with a 'particle counter' — an instrument that classifies suspended particles by number and size.
pathogen:	a disease-causing organism (eg bacteria, viruses and protozoa).
pH:	an expression of the intensity of the basic or acid condition of a liquid. Natural waters usually have a pH between 6.5 and 8.5.
point-of-use treatment device:	a treatment device applied to a single tap used for the purpose of reducing contaminants in drinking water at that one tap.
preventive measure:	any planned action, activity or process that is used to prevent hazards from occurring or reduce them to acceptable levels.
procaryote:	organism whose nucleus is not clearly defined (bacteria and cyanobacteria but not animals, plants or fungi).
Protozoa:	a phylum of single-celled animals.
quality:	the totality of characteristics of an entity that bear on its ability to satisfy stated and implied needs; the term 'quality' should not be used to express a degree of excellence (AS/NZS ISO 8402:1994).
quality assurance:	all the planned and systematic activities implemented within the quality system, and demonstrated as needed, to provide adequate confidence that an entity will fulfil requirements for quality (AS/NZS ISO 8402:1994).
quality control:	operational techniques and activities that are used to fulfil requirements for quality (AS/NZS ISO 8402:1994).
quality management:	includes both quality control and quality assurance, as well as additional concepts of quality policy, quality planning and quality improvement. Quality management operates throughout the quality system (AS/NZS ISO 8402:1994).
quality system:	organisational structure, procedures, processes and resources needed to implement quality management (AS/NZS ISO 8402:1994).
radionuclide:	an isotope of an element that is unstable and undergoes radioactive decay.
raw water:	water in its natural state, prior to any treatment; or the water entering the first treatment process of a water treatment plant.
representative sample:	a portion of material or water that is as nearly identical in content and consistency as possible to that in the larger body of material or water being sampled.
reservoir:	any natural or artificial holding area used to store, regulate or control water.
residual risk:	the risk remaining after consideration of existing preventive measures.

risk:	the likelihood of a hazard causing harm in exposed populations in a specified time frame, including the magnitude of that harm.
risk assessment:	the overall process of using available information to predict how often hazards or specified events may occur (likelihood) and the magnitude of their consequences (adapted from AS/NZS 4360:1999).
risk management:	the systematic evaluation of the water supply system, the identification of hazards and hazardous events, the assessment of risks, and the development and implementation of preventive strategies to manage the risks.
sanitary survey:	a review of the water sources, facilities, equipment, operation and maintenance of a public water system to evaluate its adequacy for producing and distributing safe drinking water.
service reservoir/tank:	a storage for drinking water, generally within the distribution system, used to meet fluctuating demands, accommodate emergency requirements and/or equalise operating pressures.
source water:	water in its natural state, before any treatment to make it suitable for drinking.
storage reservoir:	a natural or artificial impoundment used to hold water before its treatment and/or distribution.
stratification:	the formation of separate layers (of temperature, plant or animal life) in a lake or reservoir. Each layer has similar characteristics (eg all water in the layer has the same temperature).
surface water:	all water naturally open to the atmosphere (eg rivers, streams, lakes and reservoirs).
surrogate:	see indicator.
symbiont:	an organism that lives in a mutually beneficial close association with another organism.
target criteria:	quantitative or qualitative parameters established for preventive measures to indicate performance; performance goals.
thermotolerant coliforms:	see coliform bacteria.
total coliforms:	see coliform bacteria.
total quality management:	adds to the concepts of quality management a long-term global management strategy and the participation of all members of the organisation for the benefit of the organisation itself, its members, its customers and society as a whole (AS/NZS ISO 8402:1994).
toxicology:	study of poisons, their effects, antidotes and detection.
turbidity:	the cloudiness of water caused by the presence of fine suspended matter.
validation of processes:	the substantiation by scientific evidence (investigative or experimental studies) of existing or new processes and the operational criteria to ensure capability to effectively control hazards.
verification of drinking water quality:	an assessment of the overall performance of the water supply system and the ultimate quality of drinking water being supplied to consumers; incorporates both drinking water quality monitoring and monitoring of consumer satisfaction.
virus:	molecules of nucleic acid (RNA or DNA) that can enter cells and replicate in them.