Effects of Acute Alcohol Consumption on Food Intake and Pictorial Stroop Response to High Calorie Food Cues

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ABSTRACT

Aims: We examined (1) the effect of an acute dose of alcohol on the consumption of energy dense food and (2) on cognitive bias towards high-energy dense food cues and (3) whether the effect of an acute dose of alcohol on the consumption of energy dense food would be mediated by cognitive bias towards high-energy dense food cues.

Methods: Heavy social drinkers (n = 40) abstained from drinking for 12 hours prior to testing. On the test day, participants completed pre-challenge measures of alcohol and food craving, and cognitive bias towards alcohol in a placebo controlled, double blind design. Participants performed post-challenge measures of alcohol and food craving, ad lib energy dense food consumption and cognitive bias.

Results: We did not observe any of the hypothesised interactions between challenge condition, consumption of energy dense food and cognitive bias towards high-energy dense food cues.

Conclusions: Our data suggest that acute alcohol consumption does not influence consumption of energy dense food or cognitive bias towards high-energy dense food cues. These findings may reflect that alcohol does not increase the appetitive value of food and food-related cue, or that the measures used in this study were not sensitive to detect an effect. Further research is required to determine whether alcohol at higher doses and/or food cues that are frequently paired with alcohol intake stimulate changes in food intake and the reward value of food cues.

Keywords: Alcohol, food, cognitive bias, energy dense food

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INTRODUCTION

Heavy alcohol use and obesity are two of the biggest challenges to public health globally. Understanding the risk factors underlying both of these health behaviours is therefore a priority for research. Alcohol is one of the most important lifestyle factors impacting calorie intake (Chapman, Benedict et al. 2012), with growing evidence for alcohol as a contributory factor in weight gain and the development of obesity (for a review see Traversy and Chaput, 2015). However, the association between alcohol intake and weight gain is complex and research findings are inconsistent (Kwok, Dordevic et al. 2019). Further research is required to understand the mechanisms underlying the relationship between heavy alcohol use and overweight and obesity.

Several mechanisms have been proposed. Firstly, alcohol intake directly impacts weight gain due to its calorific value. Alcohol has a high-energy density, with a calorific value of 7.1 kcal/g (1 Unit of alcohol = 8g of alcohol and 56.8 kcal). The Royal Society for Public Health (2014) estimates that calories from alcohol account for around 10% of an adult drinkers' weekly calorie intake. Additionally, energy consumed from alcohol is additive to that gained from other nutritional sources (i.e. food) and is not accounted or compensated for in adult's diets (Yeomans 2010). Secondly, acute alcohol consumption stimulates food intake, via activation of neurochemical and peripheral systems involved in appetite control (Yeomans, Caton et al. 2003). Evidence from experimental studies have shown an effect of acute alcohol administration on food intake (Yeomans 2010). Several studies report greater food intake (9-26% increase) following an alcohol prime, across a range of doses (13g/ 1.6 Units -32 g/ 4 Units) compared to a no alcohol condition (Hetherington, Cameron et al, 2001, Caton, Ball et al, 2004, Caton, Marks et al, 2005, Caton, Bate et al, 2007). Studies which did not detect this effect (Poppitt, McCormack et al, 1998, Raben, Agerholm et al, 2005, have been suggested to be limited by sample size and delayed measurement of food intake following alcohol consumption (Yeomans 2010). However, no studies have demonstrated that alcohol consumption increases self-reported hunger (Hetherington, Cameron et al. 2001, Caton, Ball et al. 2004).

Finally, research points towards shared psychological processes as a mechanism underlying the association between acute alcohol use and increased food intake. Theoretical models (Goldstein and Volkow 2002, Volkow, Wang et al. 2008, Volkow, Wang et al. 2013) posit a role for impaired response inhibition and increased appetitive response to relevant stimuli (e.g. food and alcohol cues) in maladaptive eating and drinking behaviours. Studies of acute alcohol use consistently report that alcohol reduces inhibitory control of behaviour, which may lead to further alcohol consumption (Field, Wiers et al. 2010). Similarly, body mass index (Houben,

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Nederkoorn et al. 2014), unhealthy eating (Sims, Bennett et al. 2014), overeating (Guerrieri, Nederkoorn et al. 2008) and obesity (Nederkoorn, Braet et al. 2006) are associated with individual differences in inhibitory control. Impaired inhibitory control also mediates the effect of acute alcohol use on increased energy intake from food (Christiansen, Rose et al. 2016). However, the interplay between alcohol, food intake and disinhibition is complex, and may include influences from expectancy effects of alcohol and overeating (Caton, Nolan, 2015). This suggests that the disinhibing effects of acute alcohol consumption on increased food intake may be overplayed. An alternative psychological explanation is that under the influence of alcohol and in the presence of tempting food-related cues, cognitive control over food intake may give way to automatic, appetitive responding. To our knowledge no studies have explored alcohol's ability to enhance automatic, appetitive responses to food and food-related cues, in order to stimulate food intake.

Incentive Sensitization theory (Robinson and Berridge 2001) proposes that cues that are repeatedly paired with alcohol use (i.e. bar displays, wine bottles/glasses) gain strong motivational properties that are capable of driving alcohol-seeking behaviour and alcohol consumption. These alcohol-related cues "hijack" or grab the attention of drinkers, via a process of automatic detection when encountered in the environment. Several studies demonstrate that acute alcohol consumption increases incentive salience towards alcohol-related cues, via a mechanism of enhanced cognitive bias (Duka and Townshend 2004, Schoenmakers, Wiers et al. 2008, Adams, Ataya et al. 2012). Similar cognitive processes also play a role in uncontrolled food consumption (Graham, Hoover et al. 2011, Health 2014), where automatic detection and attention towards food-stimuli may contribute to overeating and obesity. Research indicates that overweight and obese individuals show an enhanced cognitive bias for food-related stimuli (Yokum, Ng et al. 2011, Hendrikse, Cachia et al. 2015). Given the frequent pairing of acute alcohol use and food intake (e.g. alcohol as an aperitif before meals, wine during mealtimes and post-drinking snacking), we aimed to examine the role of acute alcohol consumption in increasing consumption of food via a mechanism of enhanced cognitive bias towards high-energy dense food cues. The role of alcohol consumption in promoting food-related cognitive biases is supported by classical conditioning models of reward system function (Castellanos, Charboneau et al. 2009), where alcohol increases the incentive properties of food and associated cues. Recent evidence has indicated that olfactory alcohol cues increase attentional bias for food-cues (Karyadi and Cyders 2017). To our knowledge, no studies have examined whether acute alcohol use may increase energy-dense food intake via cognitive bias towards food-related cues.

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The aim of this study was to examine whether an acute dose of alcohol increases intake of high-energy dense food and enhances cognitive bias for high-energy dense food cues. This study also investigated whether the effects of acute alcohol on high-energy dense food are mediated by a cognitive bias for high-energy dense food cues. The comparison of cognitive bias towards low- versus high-energy dense food cues was chosen given the widespread use of this contrast across studies of cognitive bias for food cues (for a review see (Hendrikse, Cachia et al. 2015). We hypothesized that (1) Participants primed with an acute dose of alcohol would consume more energy dense food than participants given a non-alcoholic placebo drink, (2) Participants primed with an acute dose of alcohol would have a greater cognitive bias towards high energy dense food cues than participants given a non-alcohol placebo drink and (3) Effects of the alcohol prime on consumption of energy dense food would be mediated by cognitive bias towards high energy dense food cues.

METHODS

Design

Our study was a double-blind, placebo-controlled, between-subjects design, with one between-subjects factor of challenge condition (0.0 g/kg alcohol, 0.4 g/kg alcohol) and two within-subjects factors of time (pre-/post-challenge) and food cue type (high-energy, low-energy dense food cue).The study was approved by the University of Bath, Department of Psychology Ethics Committee (reference: 16-107).

Participants

Heavy social drinkers were recruited from students and staff at the University of Bath and members of the public. Heavy social drinkers were required to consume ≥ 14 units per week, above the UK government limits for low risk alcohol use. A unit is equivalent to 8g of alcohol. Heavy drinkers were selected to examine a sample that has been shown to exhibit attentional bias towards alcohol-related cues (Field, Mogg et al. 2004, Adams, Ataya et al. 2012). Prior to the study session participants were asked to abstain from alcohol for 12 hours (verified by exhaled breath alcohol). Participants received £10 each for participation.

Materials

Alcohol Administration. Participants were randomised to receive either an alcoholic or a placebo drink. For the alcohol condition participants received vodka at 37.5% alcohol by volume, at a dose of 0.40 g/kg alcohol with tonic water (up to a maximum of 125 ml vodka), or a placebo consisting of only tonic water. An alcohol dose

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of 0.40 g/kg was selected to administer alcohol at a level consistent with previous studies on the effect of acute alcohol use on food intake (Hetherington, Cameron et al. 2001, Caton, Ball et al. 2004, Christiansen, Rose et al. 2016). All drinks were flavoured with lime, sprayed with an alcohol mist and chilled.

Ad Lib Eating Task. Consumption of energy dense food was assessed using an ad-lib eating task, where participants were offered a bowl of ready salted crisps to consume for a period of 15 minutes. The amount of crisps consumed (weight in grams) was the primary outcome measure for this task.

Pictorial Stroop Task. Cognitive bias for food-related cues was assessed using a computerised pictorial Stroop task. This task has been shown to be sensitive to manipulation by acute alcohol consumption (Bruce, Jones et al. 2004). During the task, low- and high-energy dense food images were presented in the center of the screen, surrounded by a coloured border. Participants were instructed to respond by selecting the colour of border (yellow, red, green, blue) surrounding the image presented on the screen, using four colour coded buttons on the keyboard (yellow, red, green and blue). Participants were instructed to respond as quickly and accurately as possible. Pictorial stimuli consisted of 10 low- and 10 high-energy dense food images. Images were selected from a food image database (Blechert, Meule et al. 2014) based on their calorie content (Footnote [1]). A total of 10 high-energy dense (≥ 4 kcal/g) food pictures (including crisps, chocolate and popcorn) and 10 low-energy dense (≤ 1.6 kcal/g) food pictures (such as cherries, bananas and grapes) were selected based on definitions of high and low-energy dense foods by the British Nutrition Foundation (Foundation 2019). Table 1 presents the database image numbers and their energy density content (kcal per 100g/kcal per g) for all low and high energy dense food pictures. Each high energy dense image was matched with a low energy dense image based on colour, shape and size. All pictures had the same resolution, colour depth and were homogenous in regards to background colour and camera distance. Internal reliability for high-energy dense (α = 0.96), low-energy dense (α = 0.96) and all images was high (α = 0.98). Each trial began with a fixation cross presented for 500 ms, followed by a single food image presented until a response was made or 2500 ms had elapsed. Practice stimuli consisted of one block of 4 trials, comprising four neutral images (floral picture). Experimental stimuli consisted of two blocks of high-energy dense food cues (40 trials) and two blocks of low-energy dense food cues (40 trials). Blocks were presented in a sequential order (high, low, high, low). Low- and high-energy dense food cues were presented with equal frequency, each presented 4 times in each of the four colours; red, green, blue and yellow, to give 160 trials in total. The task lasted 10 minutes in total.

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Intraclass correlation estimates and their 95% confident intervals were calculated for RTs at pre- and post-challenge towards high and low-energy dense food cues for individuals randomised to both 0.0 g/kg and 0.4 g/kg alcohol challenge conditions, using an absolute-agreement, 2-way mixed-effects model. For the 0.0 g/kg condition ICC = 0.95, 95% CI [0.89, 0.98] and for the 0.4 g/kg ICC =0.92, 95% CI [0.54, 0.87] indicating good levels of test-retest reliability between challenge conditions.

Questionnaire Measures. Questionnaire measures included; Alcohol Use Disorder Identification Test (AUDIT) (Saunders, Aasland et al. 1993), (internal reliability α = 0.75), Alcohol Urges Questionnaire (AUQ) (Bohn, Krahn et al. 1995), (internal reliability α = 0.21), Dutch Eating Behaviour Questionnaire (DEBQ) (van Strien, Frijters et al. 1986), (internal reliability α = 0.90), Food Craving Questionnaire (FCQ) (Cepeda-Benito, Gleaves et al. 2000), (internal reliability α = 0.95), Behavioural Inhibition and Activation Scales BIS/BAS (Carver and White 1994), (internal reliability α = 0.66).

[Insert Table 1. About here]

Procedure

All participants were tested between 12 noon and 6pm in a laboratory in the Department of Psychology at the University of Bath. Participants provided informed consent and completed a screening process to confirm good physical and psychiatric health. Prior to the test session all participants were asked to abstain from drinking alcohol for 12 hours (verified by exhaled breath alcohol). Baseline questionnaire measures were completed (AUDIT, AUQ, DEBQ, BIS/BAS, FCQ-trait and state) followed by a pre-challenge measure of cognitive bias (Pictorial Stroop task). Participants were instructed “You will be randomised to receive either a non-alcoholic or alcoholic beverage” and were given 10 minutes to consume the drink. Following consumption participants were given 15 minutes to complete post-challenge measures (AUQ, FCQ- state, BIS/BAS) to allow the drink to be absorbed, in line with previous studies (Adams, Ataya et al, 2012, Adams, Ataya et al. 2013, Adams, Attwood et al, 2017). At the end of this period, participants completed the first awareness check to determine whether they were aware if they had received alcohol or placebo (i.e. Do you think your drink contained alcohol?). Following this participants completed the post-challenge measure of cognitive bias (approximately 15 minutes post-drink challenge). After completion of the cognitive bias task (approximately 25 minutes post-drink challenge) participants were offered a bowl of crisps and could eat ad libitum for a period of

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15 minutes. The period of time between drink and challenge and food intake was consistent with previous studies (Caton, Ball et al, 2004, Caton, Marks et al, 2005, Caton, Bate et al, 2006). Magazines were available to read during this period. Participants completed final questionnaire measures (AUQ, FCQ- state, BIS/BAS) and a second awareness check of the alcohol content of their drink. A final breath alcohol test was performed and participants were informed of their drink condition. At the end of the session participants received reimbursement and a full debrief. Participants who received the alcoholic drink were asked to sign a post session safety information sheet and were offered to wait in a quiet room for the effects of alcohol to wear off, participants who still felt intoxicated were offered a taxi home.

Data Analysis

All variables were screened for normality, where all analyses were ran with raw and transformed data. All analyses presented were conducted with raw data with the exception of alcohol craving (AUQ), with was transformed using Log10.

Consumption of energy dense food (amount of crisps consumed: grams) was analysed using a Welch's t-test, with a between-subjects factor of challenge condition (alcohol, placebo). Cognitive bias towards high-energy dense food cues (correct response: reaction times RT) was analysed using a 2 x 2 x 2 mixed-model ANOVA with a between-subjects factor of challenge condition (alcohol, placebo) and two within-subjects factors of time (pre-/post-challenge) and cue type (low-, high-energy dense food cue). For RT data, error trials and response times <200ms and >2000ms were removed. All analyses were initially conducted with covariates of "time since last meal" (minutes), baseline eating behaviour (DEBQ total score) and food (FCQ-Trait total score), alcohol craving (AUQ) and behavioural inhibition and activation (BIS/BAS scales). These covariates did not significantly interact with any other variables (with the exception of the BAS reward scale in the cognitive bias analysis) and are therefore not included in the reported analyses. For the cognitive bias analysis, data are presented for the model with and without BAS reward as a covariate. Mediation analysis and bootstrapping were performed to compare the direct effect of challenge condition (x) on consumption of energy dense food (y) and the indirect effect via cogitative bias towards high-energy dense food cues (m). Secondary analyses of BIS/BAS, FCQ- State and AUQ were conducted with a series of 2 x 3 mixed-model ANOVAs with a between-subjects factor of challenge condition (alcohol, placebo) and a within-subjects factor of time (baseline, post-challenge time 1, post-challenge time 2). All analyses were conducted using IBM SPSS Statistics 22 and PROCESS for SPSS 2.16.3.

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RESULTS

Characteristics of Participants

Participants (n = 40, 50% female) were, on average, aged 26 years (SD = 5, range 18–43), weighed 76 kg (SD = 15, range 54–123) and drank 20 alcohol units per week (SD = 8, range 14–55). Participants had an average AUDIT score of 13 (SD = 6, range 4–29), with 80% of the sample scoring above the cut-off of 8, indicative of hazardous alcohol consumption. Participants average time since last meal was 3 hours (SD = 2, range 0.5–13). Table 2 shows characteristics of participants by alcohol challenge condition.

[Insert Table 2. About here]

Consumption of Energy Dense Food

A Welch's t-test indicated that the amount of crisps consumed did not differ according to challenge condition (F [1, 38] = 0.008, p = 0.93, η^2 <0.001). Means, standard deviations and confidence intervals are presented in Table 3. Graphical representation of individual data points by challenge condition are presented in Figure 1.

[Insert Figure 1. About here]

Cognitive Bias towards High Energy Dense Food Cues

ANOVA of correct response RTs, indicated a main effect of time (F [1, 38] = 33.88, p < 0.001, η^2 = 0.47), such that correct response RTs decreased from pre-challenge (M =658.93 SD =89.19) to post-challenge (M = 618.48 SD =85.99). There were no further main effects or interactions (ps > 0.19). Graphical representation of individual data points by challenge condition are presented in Figure 1. Graphical representation of mean correct response RTs by time and challenge condition are presented in Figure 2.

ANOVA of correct response RTs, with BAS reward as a covariate indicated a main effect of time (F [1, 37] = 33.09, p = <0.001, η^2 = 0.47), such that correct response RTs decreased from pre-challenge (M =658.93 SD =89.19) to post-challenge (M = 618.48 SD =85.99). A main effect of challenge condition was also indicated (F

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[1, 37] = 5.23, $p = 0.028$ $\eta^2 = 0.12$), such that correct response RTs were slower for participants in the placebo condition (M =666.50 SD =98.76) compared to those in the alcohol condition (M =610.92 SD =73.20). A main effect of BAS reward was also indicated F [1, 37] = 9.98, $p = 0.003$ $\eta^2 = 0.21$). However, this was not further explored, given that a main effect of a covariate on correct response RT was not interpretable. There were no further main effects or interactions (ps > 0.12).

[Insert Table 3. About here]

[Insert Figure 2. About here]

Mediation Analysis

This analysis was not conducted, as there was no effect of drink condition on either consumption of energy dense food or cognitive bias.

Secondary Analyses

Alcohol Craving. ANOVA of mean AUQ scores indicated a main effect of time (F [2, 76] = 5.80, p = 0.007, $\eta^2 = 0.13$), such that there was a decrease in alcohol craving from baseline (M = 1.3 SD = 0.1) to post-challenge time 2 (M = 1.2 SD = 0.2). This was qualified by evidence of a weak interaction between time x challenge condition (F [2, 76] = 2.92, $p = 0.068$, $\eta^2 = 0.07$). There was no evidence for any further main effects or interactions (ps > 0.30). For the interaction between time x challenge condition, simple effects analyses were conducted for participants allocated to the alcohol (0.4 g/kg) and placebo (0.0 g/kg) conditions separately. For participants receiving alcohol there was no main effect of time (F [2, 38] = 0.23, $p = 0.75$, $\eta^2 = 0.01$). For participants receiving placebo there was a main effect of time (F [2, 38] = 9.01, $p = 0.001$, $\eta^2 = 0.32$), such that there was a decrease in alcohol craving from baseline (M = 1.3 SD = 0.1) to post-challenge time 2 (M = 1.2 SD = 0.2).

Behavioural avoidance/inhibition. ANOVA of mean BIS and BAS (Drive, Fun, and Reward) scores indicated no evidence of any main effects or interactions (ps > 0.19).

Food Craving. ANOVA of mean FCQ-State desire sub-scores indicated a main effect of time (F [2, 76] = 4.90 p = 0.010, $\eta^2 = 0.11$), such that there was an increase in desire to eat from baseline (M = 6.18 SD = 2.97) to post-challenge time 2 (M = 7.35 SD = 3.15). There was no evidence of any further main effects or interactions (ps > 0.36).

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ANOVA of mean FCQ-State negative reinforcement sub-scores indicated weak evidence of a main effect of challenge condition (F [1, 38] = 4.20, p = 0.047 η^2 = 0.10), such that relief from negative states by eating was higher in participants receiving alcohol compared (M = 7.42 SD = 3.05) to those receiving placebo (M = 5.70 SD = 2.59). There was no evidence of any further main effects or interactions (ps > 0.15).

ANOVA of mean FCQ-State lack of control sub-scores indicated evidence of a main effect of time (F [1, 76] = 3.80, p = 0.032, η2 = 0.09), however pairwise comparisons did not indicate evidence of a mean difference between any timepoints. There was no evidence of any further main effects or interactions (ps > 0.68).

ANOVA of mean FCQ-State craving (i.e. hunger) sub-scores indicated evidence of a main effect of time (F [1, 76] = 4.26, p = 0.030, η^2 = 0.10), however pairwise comparisons did not indicate evidence of a mean difference between any timepoints. There was no evidence of any further main effects or interactions (ps > 0.28).

ANOVA of mean FCQ-State positive reinforcement sub-scores indicated no evidence of any main effects or interactions (ps > 0.16).

DISCUSSION

Results did not support our hypotheses that alcohol would increase consumption of energy dense food, or cognitive bias towards high-energy dense food cues. Additionally, findings did not support our hypothesis that cognitive bias towards high-energy dense food cues would mediate alcohol's effects on consumption of energy dense food.

Our results add to an inconsistent body of research examining the effects of an alcohol priming dose on food intake. Consistent with several studies (Foltin, Kelly et al. 1993, Poppitt, Eckhardt et al. 1996, Ouwens, van Strien et al. 2003, Rose, Hardman et al. 2015) we did not observe that acute alcohol consumption increased voluntary food intake. However, this finding is in contrast to a recent growing number of studies (Hetherington, Cameron et al. 2001, Caton, Ball et al. 2004, Caton, Bate et al. 2007, Yeomans 2010, Christiansen, Rose et al. 2016) and a meta-analysis (Kwok, Dordevic et al. 2019) reporting that an alcohol prime increases the consumption of food. Whilst previous studies (Hetherington, Cameron et al, 2001, Caton, Ball et al, 2004, Caton, Marks et al, 2005, Caton, Bate et al, 2007) have reported a 9-26% increase in food intake following acute alcohol consumption, compared to a placebo drink condition, our study found a 3% increase in food intake following the consumption of a placebo drink, relative to alcohol.

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This inconsistency may be explained by the failure of the present study to detect an effect of acute alcohol use on food intake using the design and methods described. Additionally, this discrepancy in findings may reflect differences in methodology (i.e. amount of alcohol administered, type of alcoholic beverage, fixed and adjusted alcohol dosing and types of food served). However, our study was closely modelled after (Christiansen, Rose et al. 2016), using a placebo-controlled, between-subjects design, with alcohol administered as a spirit and mixer (adjusted for weight) and energy dense food available ad lib for 15 minutes. In terms of alcohol administration, the current study, used a dose of 0.4 g/kg at servings ranging from 3.05 units (24.4 grams of alcohol) to 4.05 units (36 grams alcohol). This was comparable with previous studies which have used adjusted doses of 0.6 g/kg (Christiansen, Rose et al. 2016) and fixed doses of 3-4 alcohol units (24-32 g ethanol), (Hetherington, Cameron et al. 2001, Caton, Ball et al. 2004). It is evident that further research is required to explore dose-dependent effects and the impact of different types of alcohol and food on alcohol's priming effect on food intake. For instance, there may be a pharmacological threshold of alcohol below which calorie intake will not be stimulated (Yeomans and Phillips 2002, Caton, Ball et al. 2004). Blood Alcohol Concentration (BAC) has also been shown to be influenced by type of alcoholic beverage, where spirits yield a greater BAC than beer or wine ingested at the same dose (Mitchell, Teigen et al. 2014). Furthermore, different food types may be associated with alcohol intake i.e. sweet, salty etc. and the effects of acute alcohol intake on different food should be further explored.

Previous evidence indicates that alcohol olfactory cues do not increase "cognitive" attentional biases for food-related cues (Karyadi and Cyders 2017). Similarly, in the present study we did not observe that acute alcohol consumption increases cognitive bias for high-energy food cues. This finding adds to an inconsistent body of work exploring the similarity between cognitive function and task performance between addictive behaviours and obesity (Vainik, Dagher et al. 2013). However, given that ours is the first study to explore the effects of acute alcohol consumption on cognitive bias towards food-related cues, findings will be discussed in light of the following methodological considerations. Whilst, studies including ours have examined cognitive bias towards low versus high calorie food cues (Forestell, Lau et al. 2012, Hendrikse, Cachia et al. 2015) others (Brignell, Griffiths et al. 2009, Castellanos, Charboneau et al. 2009, Calitri, Pothos et al. 2010) have included a "control" condition, with images of household items (i.e. shampoo bottle, sewing kit). The absence of a control condition in the present study may have contributed to the failure to detect cognitive bias towards food-related cues, where following alcohol consumption, participants may have demonstrated a general bias of all appetitive

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food cues. Our findings should also be considered in light of methodological concerns regarding cognitive bias measurement, which can be unreliable and produce noisy reaction time data (Ataya, Adams et al. 2012). Additionally, the reliability of established tasks such as the Stroop task in assessing individual differences has been called into question (Hedge, Powell et al. 2018). In the present study we selected a modified Stroop task, with pictorial cues and a large number of experimental trials to improve reliability in capturing cognitive bias (Ataya, Adams et al. 2012). We also assessed Stroop task reaction times at pre- and post-challenge for test-retest reliability between challenge conditions, demonstrating good levels of reliability. Consistent with Karyadi and Cyders (2017), we did not observe an effect of alcohol exposure (olfactory cue, alcohol prime) on cognitive bias as measured by reaction times on a computerised task. However, Karyadi and Cyders (2017) did observe an effect of an alcohol cue on food-related cognitive bias using eye-tracking methodology. These inconsistent findings may reflect differences in the mechanisms of cognitive bias captured by different measures (i.e. reaction time tasks, eye-tracking), (Schoenmakers, Wiers et al. 2008). Future studies are required to clarify the optimal experimental and control cues and measurement tool for exploring the effects of alcohol on cognitive bias towards food-related cues.

. Secondary data revealed that for participants receiving placebo, alcohol craving decreased from baseline to post-challenge. For those receiving placebo, the expectancy of receiving alcohol without the delivery of alcohol, may have decreased alcohol craving. This is in contrast to our previous findings, where an alcohol dose of 0.4 g/kg, compared with placebo decreased alcohol craving (Adams, Ataya et al. 2012). However, there are well-documented inconsistencies regarding the effects of acute alcohol consumption on alcohol craving across alcohol priming studies (For a review (Field, Schoenmakers et al. 2008). We also observed that all participants experienced an increased desire to eat from baseline to post-challenge, suggesting that appetite was stimulated across both alcohol and placebo conditions. Our finding is also consistent with previous studies that have failed to demonstrate a specific effect of alcohol on appetite stimulation (Hetherington, Cameron et al. 2001, Caton, Ball et al. 2004, Caton, Marks et al. 2005, Caton, Bate et al. 2007).

Potential limitations of our research include the measurement of cognitive bias at both baseline and post-challenge, which may have produced practice-related improvement on the modified Stroop Task (Davidson, Zacks et al. 2003). However, the inclusion of a baseline measure did enable us to control for individual differences in task performance prior to challenge administration. This study is also the first to use a combination of the pictorial Stroop task and the comparison of low versus high calorie food cues, thus the

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sensitivity of this measure to acute alcohol use to not yet known. However, all scales and images used in the Stroop task had high levels of internal reliability, with the exception of alcohol craving as measured by the Alcohol Craving Questionnaire. Findings on alcohol craving should therefore be interpreted with caution. We did not perform an a priori power calculation to determine sample size, therefore our study may be underpowered to detect an effect of acute alcohol use on either food intake or cognitive bias towards high-calorie food cues. Finally, we did not control for participant weight, BMI or metabolic state in the present study, exclude participants who were potentially overweight or underweight or restrict food intake prior to the experiment. These aspects of our study design may have inflated the variance between individual participants, making it more difficult to detect an effects of an acute alcohol dose. Therefore, further research is required to examine the influence of acute alcohol consumption on food intake and cognitive bias towards food-related cues in healthy, under and overweight individuals and under different conditions of metabolic state.

In conclusion, our study did not detect an effect of a moderate alcohol prime on the intake of energy dense food, but our design only provides sufficient power to study large effects. Therefore, this finding adds to an inconsistent body of research examining the pharmacological action of alcohol on short-term food consumption. Further research is required to determine whether alcohol increases intake of food in a dose-dependent fashion. Additionally, future studies should consider the contribution of different beverage types and individual differences in eating and drinking behaviours on alcohol's effects on food intake. Our study also did not detect an effect of a priming dose of alcohol dose on cognitive bias towards appetitive food-related cues. Given that this is the first study to examine this effect, future research is essential for exploring the relationship between alcohol consumption and food intake and appetitive motivational responses to food cues.

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