

The effect of alcohol on food-related attentional bias, food reward and intake: two experimental studies.

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Abstract

Acute alcohol consumption has been shown to increase food intake, and long-term alcohol consumption may be a risk for weight gain. A potential, but under-studied, mechanism for this effect is alcohol's ability to enhance food reward. In two studies, participants consumed an alcoholic drink (Study 1: 0.3 grams of alcohol per kilogram of bodyweight (g/kg); Study 2: 0.6 g/kg) and a placebo-alcohol drink in a within-subjects design. In both studies, food-related appetitive and motivational states, and attentional bias (AB) towards food-related cues were measured. In Study 1 (N = 44), participants completed a visual probe task with concurrent recording of eye-movements which measured AB towards images of palatable foods, unpalatable foods, and non-food control items. Participants also completed measures of appetite and snack urge ratings, salivary response towards palatable foods and an *ad libitum* food taste test. In Study 2 (N = 84), participants completed a similar procedure, but completed a modified Stroop task which measured differences in food-related and alcohol-related AB across the two drink conditions. In Study 1, there was no difference in food-related AB between drink conditions, and no differences in snack urge, appetite ratings, salivary response, or food intake. In contrast, Study 2 showed an alcohol-induced increase in AB towards food, but not alcohol. Snack urge, alcohol urge ratings and *ad libitum* food intake were also higher after alcohol consumption, relative to the placebo. Collectively, these findings suggest that alcohol can increase food reward and food intake, but these effects may only occur at a higher dose.

Keywords: alcohol; attentional bias; food reward; appetite; food intake; alcohol

List of abbreviations: AUDIT (Alcohol Use Disorders Identification Test, BMI (body mass index), BIS (Barratt Impulsiveness Scale), BrAC (breath alcohol concentration), DEBQ (Dutch Eating Behaviour Questionnaire), TLFB (Timeline Follow-back).

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1. Introduction

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Obesity and over-consumption of alcohol are two major global health concerns. These may be related, as excessive drinking has been implicated as having a causal role in the etiology of over-eating and obesity (Chapman et al., 2012; Sayon-Orea et al., 2011). This link between alcohol consumption and obesity is unsurprising given the high caloric density of alcohol at 7.1 kcal/g. Experimental evidence shows that not only are these calories poorly compensated for, but acute alcohol consumption can increase food intake relative to consumption of an alcohol-free drink (Kwok et al., 2019).

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One proposed mechanism for this alcohol-induced increase in food intake is the ability of alcohol to enhance the rewarding properties of food (Yeomans, 2010a). In humans, food reward (defined as the momentary value of food; Rogers & Hardman, 2015) can be measured using explicit measures, such as self-report scales which measure appetite, liking of food and desire to consume food (Rogers & Hardman, 2015; Ruddock, Field, & Hardman, 2017), but can also be measured using tasks which capture implicit biases to food cues, such as measures of attentional bias. In the case of self-report measures, indices of food reward (i.e., appetite and snack urge ratings) have been shown to increase after alcohol consumption (Caton, Bale, & Hetherington, 2007; Rose, Hardman, & Christiansen, 2015; Schrieke et al., 2015).

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Attentional bias (defined as the ability for certain stimuli to capture one's attention; Field et al., 2016) has been implicated as an index of food reward, because attentional biases are thought to indicate underlying appetitive motivational processes. When an object (such as food) is craved or desired, a greater level of attention is allocated towards cues related to this object (for review, see Field et al., 2016). In support of this theory, several studies have demonstrated that attentional bias (AB) towards food cues is positively associated with motivational states relating to food, such as hunger and food craving (Castellanos et al., 2009; Gearhardt, Treat, Hollingworth & Corbin, 2012; Graham, Hoover, Ceballos, & Komogortsev, 2011; Mogg, Bradley, Hyares, & Lee, 1998; Nijs,

Franken, & Muris, 2010; Nijs, Muris, Euser, & Franken, 2010; Schmitz, Naumann, Trentowska, & Svaldi, 2014; Tapper, Pothos, & Lawrence, 2010; Werthmann, Roefs, Nederkoorn, & Jansen, 2013; Werthmann et al., 2011). Furthermore, a recent meta-analysis by Hardman et al. (2020) found a significant correlation of $r = 0.13$ between food craving and food-related AB.

To date, little research has focused on how alcohol intoxication can alter food-related AB. One study found that AB towards food cues was increased by smelling alcohol odours, in the absence of alcohol consumption (Karyadi & Cyders, 2019). However, another study showed that the magnitude of food-related AB did not differ between consumption of a placebo-alcohol, and alcoholic doses of 0.3 g/kg or 0.65 g/kg (Monem & Fillmore, 2019). However, this study was powered to detect only a medium-to-large effect size, which may explain why no difference was found, as evidence suggests that the relationship between food craving and food-related AB is small (Hardman et al., 2020). Given these discrepant findings, the present research aimed to further investigate whether acute alcohol consumption can increase AB towards food cues.

The extent to which alcohol increases food-related AB may also depend on how rewarding the food cues are. Energy-dense, highly palatable foods (often high in fat and sugar) are more rewarding than low-calorie foods (Rogers & Brunstrom, 2016). Initial evidence suggests that alcohol can increase the desire to consume foods with low levels of palatability (Schrieke et al., 2015). However, there have been no studies to date which have systematically compared the effects of alcohol intoxication on AB towards high- and low-palatable foods. Alcohol intoxication may also produce changes in physiological responses to palatable foods. This is because cephalic phase responses (such as salivary response to food) have been shown to correlate with hunger (Wooley & Wooley, 1981) and desire to consume food (Keesman, Aarts, Vermeent, Häfner, & Papies, 2016). Through alcohol's enhancement of food reward, salivary response to food cues may therefore increase after acute alcohol consumption, however this remains untested.

It has been suggested that acute alcohol consumption produces greater levels of food intake among individuals high in dietary restraint – those who restrict energy intake to avoid weight gain. This may occur due to a reduction in the ability to maintain restrained eating behaviours, resulting in a

temporary change to dietary intentions (Caton, Nolan, & Hetherington, 2015). This effect was first studied by Polivy and Herman (1976a; 1976b) who found that when restrained eaters were aware of the presence of alcohol, their eating behaviour became disinhibited. Whereas, when restrained eaters were unaware of the presence of alcohol, food intake was suppressed (relative to unrestrained individuals), suggesting that alcohol-related expectancy effects may contribute towards disinhibited eating in restrained individuals. However, subsequent research has been unable to demonstrate that restrained eaters are more susceptible to alcohol-induced increases in food intake (Christiansen et al., 2016a; Poppitt et al., 1996; Yeomans, 2010b; Yeomans, Hails, & Nesic, 1999), even when they are made aware of the presence of alcohol (Ouwens et al., 2003). Taken together, restraint is an important variable to take into consideration when conducting research on alcohol and food intake.

2. Study 1

Overview

Study 1 investigated whether food reward (measured using self-report appetite, snack urge ratings, salivary response to food, and AB towards food-cues) and *ad libitum* food intake would differ between administration of a placebo-alcohol and an alcoholic drink (dose = 0.3 g/kg). This dose was chosen because although Monem and Fillmore (2019) were unable to show an enhanced food AB at 0.3 g/kg, this same dose has been shown to enhance AB towards other appetitive stimuli (i.e., alcohol) relative to a placebo-alcohol (Duka & Townshend, 2004; Schoenmakers et al., 2008).

The AB measure was a visual probe task with concurrent eye-tracking, with comparisons of three image pairs: palatable food and unpalatable food images, palatable food and non-food images, unpalatable food and non-food images. Fixation duration from concurrent eye-tracking was the outcome measure, as this has greater internal reliability as compared with reaction time assessments when measuring food-related AB using the visual probe task (van Ens, Schmidt, Campbell, Roefs, & Werthmann, 2019). It was predicted that all measures of food reward and food intake would increase

after consumption of an alcoholic drink, relative to a placebo-alcohol. In secondary analyses, we tested whether dietary restraint moderates the effect of drink condition on food intake.

2.1 Method

2.1.1 Participants

At the time of data collection, no previous published studies had investigated the difference in food-related AB between consumption of an alcoholic drink and placebo, therefore the study was powered to detect a small-to-medium effect size ($d_z = 0.39$) for differences in food-related AB between drink conditions. Using G*Power 3.1 (Faul, Erdfelder, Buchner & Lang, 2009) and based on 80% power and an alpha level of 5%, 43 participants were required. Forty-four participants (men = 22) aged between 18 and 54 y (Mean = 25.55, standard deviation = 8.22), were recruited in order to achieve full counterbalancing of drink order. Participants were recruited through online and email advertisement, and word-of-mouth and were eligible to take part if they were aged 18 – 65 y, had no history of food allergies or intolerances, were regular consumers of alcohol (consuming alcohol at least once a week and drinking at least 10 UK alcohol units per week), and enjoyed consuming cookies and tortilla chips, as these were used as test foods. Participants were excluded if: they wore glasses to correct their vision (due to interference with the eye-tracking camera); had a current or past alcohol use or eating disorder; had a current or recent illness that may increase sensitivity to alcohol (e.g., cold and flu); were taking medication that may be affected by alcohol; were currently breastfeeding or pregnant. Participants were also required to consume a light meal, low in fat, one hour prior to the test session. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee. Participants were reimbursed through either course credits or a £10 shopping voucher.

2.1.2 Design

The study used a single-blind randomised within-subjects design with drink type (alcoholic drink, placebo-alcohol) as the independent variable. Each participant completed both conditions in

two separate sessions separated by at least one week. The order of conditions was randomised and counterbalanced across participants.

2.1.3 Measures

Beverage Preparation and Administration.

The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV) at a dose of 0.3 g of alcohol per kg of body weight (2.68 UK units of alcohol for a participant weighing 70 kg), up to a maximum of 200 ml of vodka (1 g of vodka = 2.08 kcal). The drink was mixed with chilled diet lemonade in the ratio one-part vodka to three parts diet lemonade. The placebo drink consisted of diet lemonade only (beverage volume was matched within participants across conditions); a vodka mist was sprayed on the surface of the drink to create the impression that it contained alcohol.

Pictorial Stimuli

The Visual Probe Task (VPT) consisted of three image types (with two subtypes within each image type, presented on an equal amount of trials) – palatable foods (tortilla chips and chocolate chip cookies), unpalatable foods (boiled potatoes and wholemeal bread), and non-food controls (leaves and drink coasters). This generated three types of image pairs – palatable and unpalatable, palatable and control, unpalatable and control (each with eight image pairs). To ensure that images were well matched on visual characteristics, tortilla chips, boiled potatoes and leaves were only ever presented with each other, and chocolate chip cookies, wholemeal bread and drink coaster were presented with each other. Images were sourced from a web browser (<https://www.google.com/imghp?hl=EN>) and selected if they had appropriate visual characteristics. Images can be found in the supplementary materials. All images were 400 x 300 pixels and were displayed on a plain black background.

Visual Probe Task (VPT)

The VPT was programmed in Inquisit version 4 (Millisecond software, 2016). Each trial began with a white fixation cross presented in the centre of the screen for 500 ms. Immediately afterwards, a pair of pictures were presented for 2000 ms, one picture on the left of the screen and the

other on the right, 60 mm apart. After this, the pictures disappeared, and a probe – an ‘X’ – appeared in the position of one of the images. Participants were required to respond to whether the probe appeared in the position of the left or right image, by pressing the ‘E’ or ‘I’ key, respectively. The inter-trial interval was 500 ms.

The task consisted of 108 trials. Participants first completed ten practice trials in which neutral picture pairs (images of office supplies) were presented. The main task consisted of two buffer trials (neutral picture pairs) followed by 96 critical trials. Each of the 24 picture pairs were presented four times, both images in each pair were presented twice on the left and twice on the right side of the screen, with the probe appearing an equal number of times behind each image. The visual probe replaced both images in the pair with equal frequency. Trials were presented in a random order for each participant. Eye-movements were recorded during the 2000 ms of stimulus presentation using an eye-tracker (Applied Science Laboratories Eye-Trac D6, Bedford MA) at a sampling rate of 120 Hz. The outcome measure was fixation duration (in milliseconds). Gaze direction bias and reaction time to probes were also measured on each trial and are reported in the supplementary materials.

Salivation:

Consistent with previous studies (Brunstrom, Yates & Witcomb, 2004; Hardman, Scott, Field & Jones, 2014), volume of salivation was measured by participants placing a 3.5 cm dental roll under their tongue for 30 seconds. The dental roll was weighed before and afterwards. This difference in weight (g) was recorded as the amount of salivation.

Bogus taste-test.

The taste-test consisted of a 200 g serving of Maryland chocolate chip cookies (487 kcal/100 g) and 200 g serving of plain tortilla chips (499 kcal/100 g), which were served with 400 grams of water. The foods were served in two identical white bowls. Tortilla chips and cookies were broken into smaller pieces so that participants could not easily monitor the amount consumed (Higgs & Woodward, 2009). Participants were asked to taste each of the foods and to rate them on a series of sensory properties (anchors; ‘Not at all’ – ‘Extremely’) (data not analysed). Participants were given

15 minutes to complete this task and were told to consume as much of the foods as they liked if they finished before the 15-minute period. Taste-test consumption was calculated by subtracting the post taste-test weight from the pre-taste-test weight. Grams consumed was converted to kilocalories. The bogus taste-test has been shown to be a valid measure of food intake (Robinson et al., 2017).

Dutch Eating Behavior Questionnaire.

The Dutch Eating Behaviour Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares, 1986) is a 33-item questionnaire measuring eating styles associated with being overweight. The three subscales are restraint ($\omega = .92$), emotional eating ($\omega = .95$), and external eating ($\omega = .86$).

Timeline Follow Back.

In the Timeline Follow Back (TLFB; Sobell & Sobell, 1990), participants estimated the number of alcohol UK units (one UK unit 8 g of alcohol) consumed over the past seven days.

Alcohol Use Disorders Identification Test.

The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, de la Fuente, & Grant, 1993) is a 10-items questionnaire assessing hazardous drinking ($\omega = .85$). Scores range between 0 and 40, with scores of ≥ 8 indicating hazardous alcohol use.

Snack Urge Scale.

The Snack Urge Scale (Hardman et al., 2015) measured expected liking, desire to consume, craving, and difficulty to resist each of the snack foods in the present moment. This was measured using a 100-mm Visual Analogue Scale (VAS: ‘Not at all’ – ‘Extremely’). A composite snack urge score was calculated by adding scores from the four scales together, which was then summed across the two snack foods.

Appetite Ratings.

Appetite Ratings (Blundell et al., 2010) of hunger (I feel hungry) and fullness (My stomach feels full) were measured using a 100-mm VAS ('Not at all' – 'Extremely'). These scores were combined (hunger added to the inverse score of fullness) and reported as a single appetite rating.

2.1.4 Procedure

Test sessions took place between 12:00 and 18:00 on weekdays in the Department of Psychology on the University of Liverpool campus. Each session lasted no longer than 90 minutes. All participants completed both sessions at least one week apart from each other. Participants were told that the present study was investigating how different doses of alcohol can affect reaction times towards and taste perception of food. Participants were told that across both sessions, they would consume two alcoholic drinks: one 'low' and one 'high' in alcohol. This was done in an attempt to match the anticipated effects of alcohol across conditions, such that participants expected to consume alcohol in both sessions, as has been done in previous research (e.g., Baines et al., 2019). Upon arrival, participants gave written informed consent. As participants were required to consume a light meal an hour before the beginning of the sessions, they next reported when they had last eaten and what they had consumed to ensure they had complied with this instruction. Participants were then breathalysed (all had a BrAC of 0.00) and completed a medical history questionnaire to check for food allergies. Height and weight measurements were then taken in person (using a stadiometer and weighing scale, respectively) in order to calculate the alcohol dosage. Next, baseline salivation was measured, followed by completion of baseline appetite, snack urge ratings, the DEBQ, AUDIT, and TLFB. Participants then consumed the test drink within ten minutes. This was immediately followed by a ten-minute absorption period where participants sat quietly. Next, the second set of breathalyser, salivation, appetite, and snack urge measures were taken. Participants then completed the VPT. Immediately afterwards, a third salivation measure was taken, which was measured when the taste-test foods were placed in front of the participant (this was the food-exposure measure). The third set of appetite, and snack urge ratings were taken also in the presence of the test foods. Participants then completed the bogus taste-test. Afterwards, a third breathalyser measure was taken, followed by the fourth set of appetite and snack urge ratings. For session 2 only, participants then completed an

awareness check, whereby participants were asked to state what they believed to be the true aims of the experiment. Participants were then fully debriefed and reimbursed for their time.

2.1.5 Data reduction and analysis

For the eye-tracking data, valid fixations were defined as a stable eye-movement within one degree of a visual angle for 100 ms or longer, as defined in previous research (Jones et al., 2012). Mean bias scores were the primary outcome measure of the eye-tracking data. To calculate mean bias scores, mean fixation duration on control images was subtracted from mean fixation duration on target images; positive scores were indicative of an AB towards target images. Target images were palatable foods in the palatable vs. unpalatable and palatable vs. control trials, and unpalatable foods in the unpalatable vs. control trials. Internal reliability (calculated using McDonalds ω) was calculated for each pair of images (the target image and its matched control image). This was done by calculating the mean fixation duration for each target stimuli and its matched control. The control fixation duration was subtracted from the target fixation duration. As there were eight image pairs within each pair type, McDonalds ω reflects internal consistency across eight AB scores for each pair type – see Table S1 of the supplementary materials for these internal reliability scores. Eye-tracking data from four participants were removed from all eye-tracking analyses due to insufficient calibration quality of the eye-tracker, leaving 40 participants for the analysis.

For mean bias scores, a 2 (drink; alcohol, placebo) x 3 (pair; palatable vs control, unpalatable vs. control, palatable vs unpalatable) repeated measures ANOVA was conducted. One-sample t-tests were also conducted to see whether mean bias scores significantly differed from zero (indicative of bias towards a stimulus type). In order to test whether AB performance was related to appetitive motivational states, we tested whether average food-related AB (on palatable vs control trials) across the two drink conditions correlated with average post-drink snack urge ratings across the two conditions.

To test whether the drink type affected self-report measures of food reward, 2 (drink; alcoholic drink, placebo-alcohol) x 4 (baseline, post-drink, food-exposure, post-taste-test) ANOVAs were conducted on snack urge and appetite ratings. Similarly, a 2 (drink; alcoholic drink, placebo-alcohol) x 3 (baseline, post-drink, food-exposure) ANOVA was conducted on the measure of salivary response. Pairwise comparisons using Bonferroni correction was conducted when breaking down significant main effects. Greenhouse-Geisser corrected tests are reported where sphericity is violated.

Paired sample t-tests were conducted to determine whether food intake significantly differed across conditions, and also whether total calories consumed (food intake and drink calories combined) significantly differed across conditions. Finally, using the MEMORE macro for SPSS (Montoya & Hayes, 2017), a moderation analysis was performed to see whether DEBQ restraint scores moderated the effect of drink type on food intake.

2.2 Results

2.2.1 Participant characteristics

Participant characteristics are shown in Table 1.

Table 1. Mean (\pm SD) for participant characteristics.

Measure	Total sample (N = 44)
Age (years)	25.55 \pm 8.22
BMI (kg/m ²)	25.98 \pm 5.73
DEBQ Restraint	2.55 \pm 0.67
DEBQ Emotional	2.46 \pm 0.81
DEBQ External	3.29 \pm 0.55
AUDIT (out of 40)	10.89 \pm 4.81
7-Day TLFB (in units)	19.68 \pm 13.37

2.2.2 Mean attentional bias scores (Figure 1)

As shown in Figure 1, there were no significant main effects of drink $F(1, 39) = 0.36, p = .551, \eta_p^2 = .01$, or pair type $F(1.24, 48.41) = 1.42, p = .246, \eta_p^2 = .04$, and no significant drink x pair interaction $F(1.24, 48.41) = 0.80, p = .400, \eta_p^2 = .02$. One-sample t-tests revealed that mean bias scores were significantly greater than zero on the palatable vs. control trials in both the alcohol $t(39) =$

3.14, $p = .003$, $d = 0.50$ and placebo condition $t(39) = 3.14$, $p = .003$, $d = 0.50$, and for unpalatable/control trials in the placebo condition $t(39) = 2.41$, $p = .021$, $d = 0.38$, but not for any other trial type on for either drink condition. There was no significant correlation between average post-drink snack urge ratings and average mean bias scores for palatable vs control trials $r < -.01$, $p = .993$.

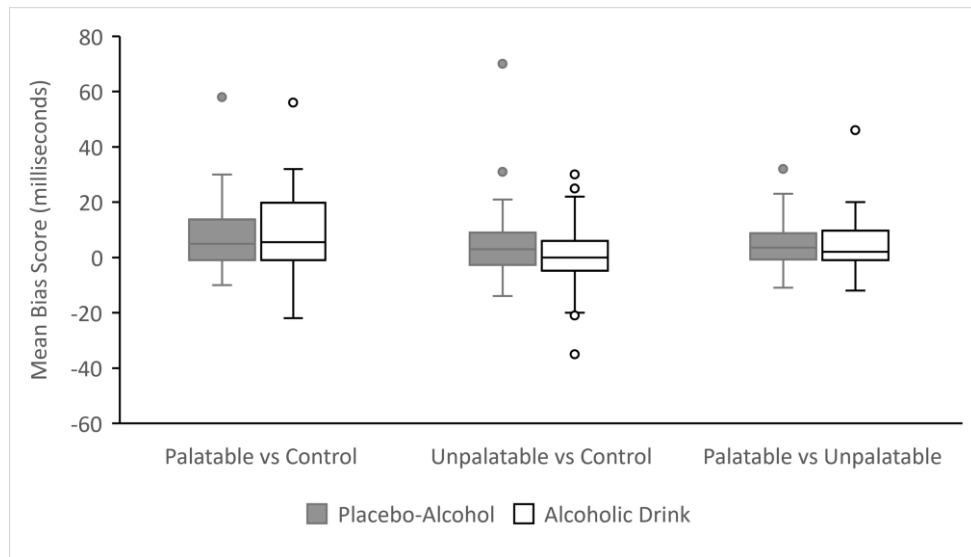


Figure 1. Boxplot displaying mean attentional bias scores split by pair type and drink condition. Positive scores indicate greater fixation duration towards palatable images for palatable vs control trials and palatable vs unpalatable trials. Positive scores indicate greater fixation duration towards unpalatable images for unpalatable vs control trials. Dots indicate outliers

2.2.3 Appetite Ratings (Figure 2a)

There was a significant main effect of time on appetite ratings $F(2.15, 90.36) = 47.25$, $p < .001$, $\eta_p^2 = .53$ (see Figure 2a for comparisons across time points). There was no main effect of condition $F(1, 42) = 1.20$, $p = .279$, $\eta_p^2 = .03$ or interaction between time and condition $F(2.45, 103.09) = 1.22$, $p = .305$, $\eta_p^2 = .03$.

2.2.4 Snack Urge Ratings (Figure 2b).

308 The analysis revealed a main effect of time $F(2.03, 87.26) = 23.34, p < .001, \eta_p^2 = .35$ (see
 309 Figure 2b for comparisons across time points). However the main effect of condition $F(1,43) = 0.31, p$
 310 $= .583, \eta_p^2 = .01$, and interaction between time and condition $F(2.49, 107.24) = 1.50, p = .224, \eta_p^2 =$
 311 $.03$ were both non-significant.

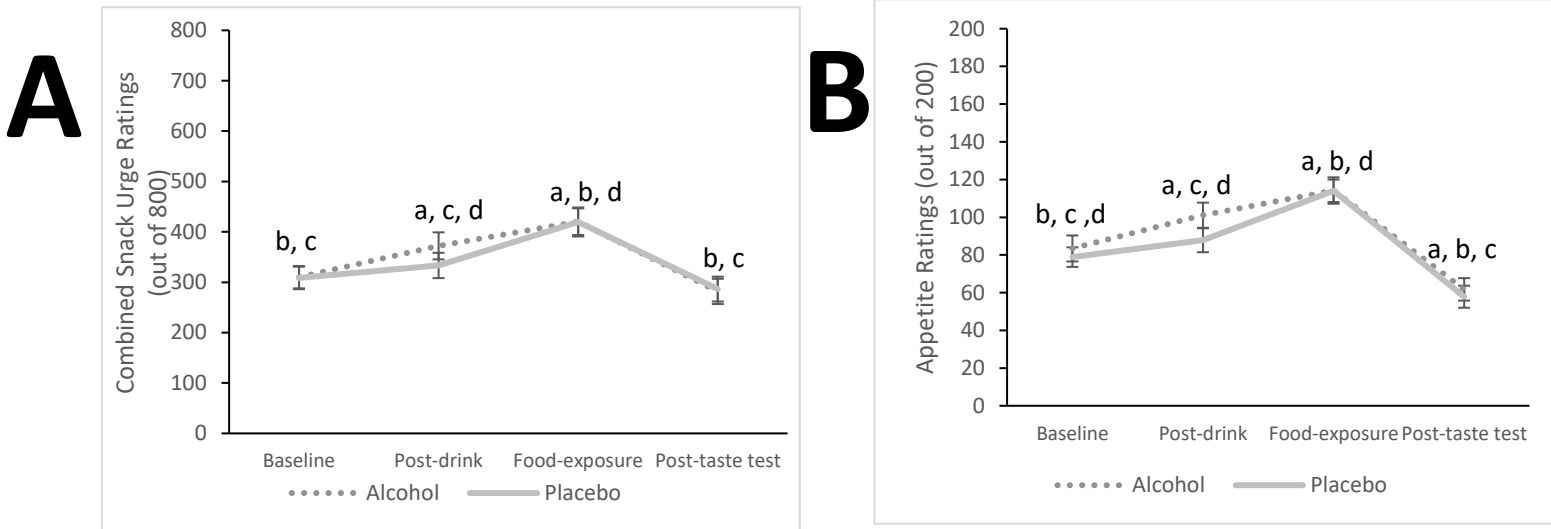


Figure 2. Snack Urge (2a) and Appetite ratings (2b) over time, by condition (Mean \pm SEM). Letters refer to Bonferroni corrected pairwise comparisons breaking down significant differences ($p < .05$) between time points (collapsed across drink conditions): a = difference from baseline; b = difference from post-drink; c = difference from food exposure, d = difference from post-taste test.

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313 2.2.5 Salivation Measure

314 There was a significant main effect of time $F(2, 86) = 6.56, p = .002, \eta_p^2 = .13$. Pairwise
 315 comparisons revealed that the amount of salivation was lower at baseline than at post-drink ($p = .018$;
 316 mean difference = 0.05; 95% CI [-0.09, -0.01]) and at food exposure ($p = .005$; mean difference =
 317 0.07; 95% CI [-0.12, -0.02]). However, there was no significant difference between post-drink and
 318 food exposure ($p = 1.00$; mean difference = 0.02; 95% CI [-0.03, 0.07]). The main effect of condition,
 319 $F(1, 43) = 0.54, p = .468, \eta_p^2 = .01$, and the time by condition interaction $F(1.72, 74.14) = 0.38, p =$
 320 $.655, \eta_p^2 = .01$ were both non-significant.

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322 2.2.6 Calorie Measures (Figure 3)

Paired sample t-tests revealed no significant difference between conditions for the amount of food calories consumed during the taste test, $t(43) = -0.92$, $p = .361$, $d = 0.14$. However there was a significant difference in total calories consumed (drink calories combined with food calories) $t(43) = 3.37$, $p = .002$, $d = 0.51$, with participants in the alcohol condition consuming significantly more calories overall relative to the placebo condition. The moderation analysis revealed that DEBQ restraint scores did not moderate the effect of drink type on food intake $b = 87.23$ $[-15.45, 189.91]$, $SE = 50.88$, $t(42) = 1.71$, $p = .094$.

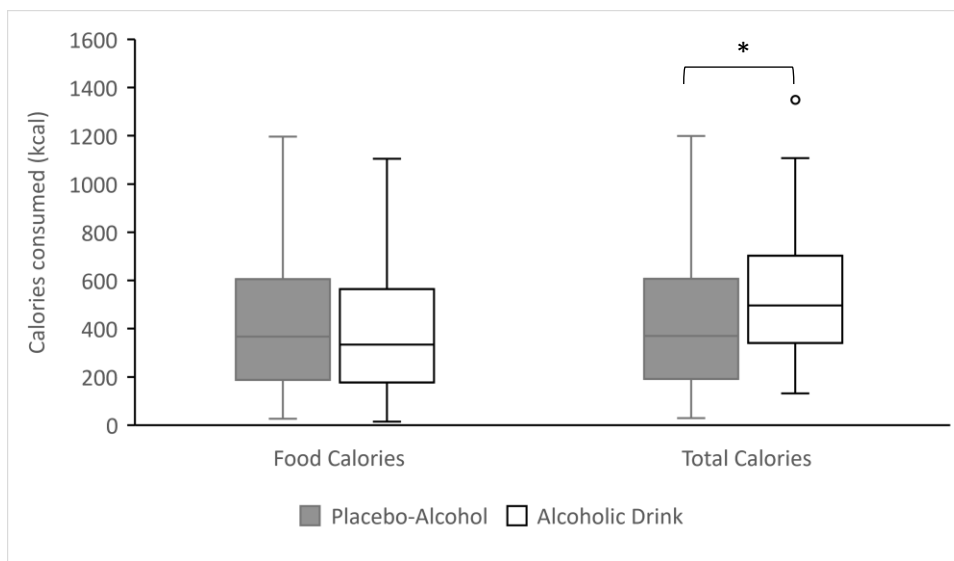


Figure 3. Boxplot displaying number of calories consumed during the *ad libitum* taste test (food calories) and combined with calories consumed from the test drink (total calories), split by condition. Note. $*p = .002$. Dots indicate outliers

2.3. Interim Discussion

Study 1 investigated whether an alcohol dose of 0.3 g/kg could alter food-related attentional biases, self-report appetitive motivational states, salivation response, and food intake relative to a placebo-alcohol. The results showed that alcohol consumption did not produce greater attentional biases towards food-related stimuli, nor was there evidence of alcohol-induced changes in appetitive motivational states or increase in salivation towards palatable foods. Although null, these results are in line with the notion that changes in AB are, in part, the result of changes in motivational states

(Field et al., 2016). Furthermore, Study 1 showed no change in *ad libitum* food intake. However, total caloric intake was significantly greater in the alcohol condition relative to placebo. This latter finding is in line with previous research which has consistently shown that the calories within an alcoholic beverage appears to be additive and are not compensated for at a later eating episode (Caton, Ball, Ahern, & Hetherington, 2004; Christiansen et al., 2016a; Mattes, 1996; Rose, Hardman, & Christiansen, 2015; Yeomans, Hails, & Nesic, 1999).

These null findings may be explained by the dose of alcohol being too low to produce meaningful changes in appetitive motivational states and food intake. Previous research has found that a dosage of 0.6 g/kg produces significant changes in snack urge ratings and food intake (Christiansen et al., 2016a; Rose, Hardman, & Christiansen, 2015). Furthermore, Rose and Duka (2006) found that self-report appetitive motivation towards alcohol increased after a dose of 0.6 g/kg but not 0.3 g/kg, relative to placebo. A higher alcohol dosage was not used in Study 1 because other research has found an AB towards other types of appetitive stimuli at a dose of 0.3 g/kg (Schoenmakers et al., 2008). Furthermore, higher doses of alcohol have consistently failed to enhance alcohol-related AB (0.6 g/kg - Duka & Townshend, 2004; 0.65 g/kg - Monem & Fillmore, 2019), despite evidence showing increases in alcohol craving at similar doses (Duka, Jackson, Smith & Stephens, 1999; Rose & Duka, 2006). These null findings may be because higher doses of alcohol are problematic for measuring AB due to oculomotor impairments following alcohol consumption (Abroms, Gottlob, & Fillmore, 2006; Moser, Heide, & Kömpf, 1998; Rohrbaugh et al., 1988). Therefore, an AB task which uses ocular behaviour (i.e., eye movements) as its outcome measure, may mask an effect of AB when using higher doses of alcohol despite enhancements in food-related appetitive motivational states.

3. Study 2

Overview

Study 2 investigated whether consumption of a 0.6 g/kg dose of alcohol can enhance AB towards images of food, increase self-report measures of food reward (appetite and snack urge ratings) and increase food intake, relative to a placebo-alcohol. Additionally, in order to provide a

further manipulation check between drink conditions, the study tested whether consumption of the alcoholic drink could produce an increase in alcohol-related motivational states (alcohol urge ratings) and AB towards alcohol cues, as this dose has previously been shown to increase motivation for alcohol (Duka, Jackson, Smith & Stephens, 1999; Rose & Duka, 2006).

In order to mitigate the issue of impairments in ocular behaviours at higher doses, Study 2 measured both food and alcohol-related AB with a pictorial modified Stroop task, which captures AB using manual response latencies rather than ocular fixation behaviour. The pictorial form of the Stroop task has been shown to produce acceptable levels of internal reliability (Ataya et al., 2012).

An additional aim was to examine the role of top-down and bottom-up processes in driving alcohol-induced increases in food intake. Dual-process models argue that eating behaviour is determined by an interaction of bottom-up drives relating to motivational orientation and food reward, and top-down cognitive control (Appelhans, 2009). Several studies have demonstrated the combined effect of food reward and impulsivity (indicating weaker top-down control) in predicting eating behaviour and weight change (Appelhans et al., 2011; Kakoschke et al., 2015; Nederkoorn et al., 2009; Nederkoorn et al., 2010; Price, Higgs, & Lee, 2015; Rollins, Dearing, & Epstein, 2010). For example, Nederkoorn et al. (2009) showed that poor response inhibition (a type of impulsivity) was related to overeating only when desire to eat was also high. It is therefore possible that top-down control and bottom-up reward processes interact to facilitate alcohol-induced overeating. To test this, Study 2 investigated whether trait impulsivity (specifically motor impulsivity) and alcohol-induced changes in food-related AB (using the pictorial modified Stroop task) could interactively predict changes in food intake across drink conditions. We chose motor impulsivity as our measure of (weaker) top-down control because previous research has shown this to be positively associated with disinhibited eating and BMI (Price et al., 2015; Van Koningsbruggen et al., 2013) and, most relevantly, to interact with food-related AB to predict weight gain (Meule & Platte, 2016).

We predicted there would be an enhanced food and alcohol-related AB after consumption of the alcoholic drink compared with a placebo-alcohol. We also predicted that participants would consume more calories in an *ad libitum* taste test after consumption of the alcoholic drink, and that

appetite, snack urge and alcohol urge ratings would increase to a greater extent after alcohol consumption compared with the placebo. We predicted a positive correlation between post-drink snack urge ratings and food-related AB, and between post-drink alcohol urge ratings and alcohol-related AB. Lastly, we predicted that the interaction term of motor impulsivity and change in food-related AB between conditions would significantly predict change in food intake between conditions.

3.1 Method

3.1.1 Participants

The study was powered based on an earlier version of Hardman et al.'s (2020) meta-analysis (Hardman et al., 2018) which found a correlation of $r = 0.14$ between food-related AB and food craving. Based on 80% power and an alpha level of 5%, 81 participants would be needed in order to detect the same effect size between drink conditions. 84 participants (men = 13) aged between 18 and 26 y ($M = 18.75$; $SD = 1.13$) completed both sessions in order to counterbalance the order of drink condition and the order of target and neutral blocks in the Stroop task (see measures section for further details). Six additional participants completed session one, but did not return for session 2, and were therefore excluded from all analyses. Participants were recruited through the university undergraduate credit scheme. Inclusion criteria was the same as in Study 1 with the following changes: participants were able to take part if they wore glasses to correct their vision, but participants were excluded if they were colour-blind. All participants provided written informed consent to participate in the experiment, which was approved by the University of Liverpool Health and Life Sciences Research Ethics Committee. Participants were reimbursed through course credits. The method and analysis strategy for this study were pre-registered on the Open Science Framework (<https://osf.io/cnaxr/>).

3.1.2 Design

The study used a single-blind randomised within-subjects design with drink type (alcoholic drink, placebo-alcohol) as the independent variable. Each participant completed both conditions in

two separate sessions separated by at least one week. The order of drink condition was randomised and counterbalanced across participants.

3.1.3 Measures

Modified Stroop Task

Participants completed four blocks of a pictorial modified Stroop task on PsychoPy2 (Peirce et al., 2019). Each block consisted of 40 trials: ten different images, presented four times, each time with a different coloured border surrounding the image (either blue, red, yellow, or green). The four blocks consisted of: food images (five images of cookies and five of tortilla chips), food control images (five of drink coasters and five of leaves), alcohol images (alcoholic drinks), and alcohol control images (office stationary). The food and food control images were the same as in Study 1 with the addition of two extra pairs (sourced from the same website as in Study 1). Alcohol and alcohol control images were taken from a previous study (Field et al., 2011) and are included in the supplementary materials. Four alcohol/alcohol-control picture pairs were removed due to these images containing an identifiable person.

Each image was 351 x 259 pixels and was surrounded by a 10-pixel coloured border. Images were matched on visual properties such as colour and brightness. For each trial, participants were required to respond to the colour of the border surrounding the image as quickly and as accurately as possible, participants did so by providing a key response (using D, F, J, and K). The keys were marked with coloured stickers that matched the corresponding colours for responses. The same colours were matched with the same key for every participant. Participants were instructed to place the index and middle finger of the left hand on the 'D' and 'F' key respectively, and the same fingers of the right hand on the 'J' and 'K' key.

In both sessions, participants completed a block of 40 practice trials using filler images (a plain image surrounded by each border colour 10 times) before the main task in order to become familiar with the location of each key response. Participants were required to repeat the practice block until they provided correct responses on at least 95% of trials within this block. The main task

consisted of four blocks, this was completed in blocked presentation in order to avoid any interference carry over effects (Waters, Sayette, & Wertz, 2003). The order of blocks was counterbalanced such that for the first session, half of the participants saw the presentation in the following order: food images, food control images, alcohol images, alcohol control images. The other half of participants saw the presentation in the following order: food control images, food images, alcohol control images, alcohol images. All participants saw the other presentation order in their second session.

For the main task each trial began with a fixation cross, presented in the middle of the screen for 500 ms. Following this, the image was presented in the middle of the screen until a response was made. The inter-trial interval was 500 ms. There was also a 5-second inter-block break.

Before AB scores were calculated, all responses quicker than 200 ms, slower than 2000 ms responses, three standard deviations above the individual mean response and incorrect responses were removed. This resulted in the removal of 4.98% of trials. After data reduction, mean reaction time on control trials was subtracted from mean reaction time on target trials; positive scores were indicative of an AB towards the target stimuli (food images and alcohol images). Internal reliability (calculated using McDonalds ω) was calculated for each pair of stimuli (the target image and its matched control image). This was done by calculating the mean reaction time across all coloured borders for each target stimuli and its matched control. The control reaction time was subtracted from the target reaction time. As there were 10 image pairs for both the food-related and alcohol-related AB, McDonalds ω for each AB type reflects internal consistency across 10 AB scores. Internal reliability scores are presented in Table S3 of the supplementary materials.

Barratt Impulsivity Scale (BIS [v11]; Patton, Stanford, & Barratt, 1995):

Trait impulsivity was assessed across three dimensions; attentional ($\omega = .77$), motor ($\omega = .71$), and non-planning ($\omega = .80$). The BIS consists of 30 items (score Rarely/Never – Almost always/Always) with higher scores indicating greater impulsivity. The motor dimension (motor impulsivity) of the scale (which captures acting without thinking) was measured to see whether it

predicts alcohol-induced change in food intake. Data on the other dimensions of the BIS were recorded to characterise the sample.

Alcohol Urge Questionnaire (AUQ; Bohn, Krahn, & Staehler, 1995):

Participants were asked to provide current alcohol urge ratings across three domains: desire for alcohol; expectation of positive effect from drinking; and inability to avoid drinking if alcohol was available. Items were responded to on a scale from 1 to 7 with high scores being indicative of greater alcohol urge.

Subjective intoxication scales (SIS; Duka et al., 1998):

Participants were asked to provide subjective feelings of being ‘lightheaded’, ‘irritable’, ‘stimulated’, ‘alert’, ‘relaxed’, and ‘contented’ before and after consumption of the test drink. These data are presented in the supplementary materials. Participants were also asked how many units of alcohol they believed they had consumed at the end of each session.

Beverage Preparation and Administration.

This was the same as in Study 1 with the following changes: the alcohol dose was at 0.6 g/kg (5.35 UK units of alcohol for a participant weighing 70 kg); participants consumed the test drink in three separate portions, each served in set 5-minute intervals, meaning that participants consumed the test drink in 15 minutes.

The following measures were used as in Study 1: DEBQ (restraint $\omega = .96$; emotional eating $\omega = .96$; external eating $\omega = .92$); TLFB; AUDIT ($\omega = .74$), Snack Urge Scale, Appetite Ratings, bogus taste-test.

3.1.4 Procedure

The procedure was similar to that of Study 1 with the following changes: the cover story was changed such that participants in Study 2 were told that the aims were to see how different doses of

alcohol can affect visual and taste perception of food. At the beginning of the session, participants completed baseline alcohol urge and subjective intoxication scale ratings and completed the BIS; participants completed a 20-minute absorption period after consumption of their test drink; participants completed post-drink alcohol urge and subjective intoxication scale ratings; after completing the taste test, participants provided a set of alcohol urge ratings and were asked how many units of alcohol they believed the test drink contained. For the second session, the procedure was identical to session 1, apart from participants consumed the other drink type, and also completed the modified Stroop task in the other block order, and did not complete height and weight measures, the DEBQ, BIS, TLFB, or AUDIT. Lastly, at the end of the second session, participants completed the aims awareness question and were fully debriefed and reimbursed.

3.1.5 Data Analysis

A 2 (drink; alcoholic-drink, placebo-alcohol) x 2 (task; food AB, alcohol AB) repeated measures ANOVA was conducted on AB scores. Follow-up paired samples t-tests were conducted in order to investigate the effect of the drink condition on AB score, separately for the two types of target stimuli. One-sample t-tests were conducted for both AB tasks, split by drink, in order to test whether mean bias scores significantly differed from zero. In order to test whether AB performance was related to appetitive motivational states, two correlations were conducted to test whether average food-related AB across the two drink conditions correlated with average post-drink snack urge ratings across the two conditions, and to test whether average alcohol-related AB correlated with average alcohol urge ratings at post-drink, between the two drink conditions.

Two paired samples t-tests were conducted to examine whether food intake and total caloric intake (food and drink calories combined) differed between drink conditions. A moderation analysis was performed to see whether DEBQ restraint scores moderated the effect of drink type on food intake. Separate 2 (drink; alcohol, placebo) x 3 (baseline, post-drink, post-taste test) repeated measure ANOVAs were conducted for appetite ratings, total snack urge ratings, and total alcohol urge ratings

(Greenhouse-Geisser corrected tests are reported where sphericity is violated). A one-sample t-test was conducted to see whether the estimated number of units consumed in the placebo condition significantly differed from zero to confirm whether participants believed there to be alcohol in this condition. A paired samples t-test was conducted on unit estimation scores between drink conditions. A 2 (drink; alcohol, placebo) x 2 (baseline, post-drink) repeated measures ANOVA was conducted for subjective intoxication scale scores (see supplementary materials for analysis subjective intoxication scale scores).

A hierarchical regression was conducted with restraint scores, BMI and food AB scores in the placebo condition entered in step 1 as control variables. Change in food-related AB between conditions (positive scores indicating a greater AB in the alcohol condition relative to placebo) and trait motor impulsivity and the interaction between change in food-related AB x trait motor impulsivity were entered as predictor variables at step 2. Change in food intake between conditions (positive scores indicative of greater food intake in the alcohol condition relative to placebo) was the dependent variable. Due to high VIF scores (> 10), the predictor variables were mean centred. This reduced VIF scores to an acceptable level.

3.2 Results

3.2.1 Participant characteristics are shown in Table 2.

Table 2. Sample characteristics (mean \pm SD).

Measure	Total sample (N = 84)
Age (years)	18.75 \pm 1.13
BMI (kg/m ²)	22.41 \pm 3.54
DEBQ Restraint	2.25 \pm 0.95
DEBQ Emotional	2.80 \pm 0.89
DEBQ External	3.34 \pm 0.66
AUDIT (out of 40)	13.27 \pm 4.31
7-Day TLFB (in alcohol units)	18.24 \pm 4.31
BIS (attentional)	17.79 \pm 3.85
BIS (motor)	22.70 \pm 4.11
BIS (non-planning)	24.69 \pm 4.61
BIS (total)	65.18 \pm 9.81

3.2.2 Modified Stroop (Figure 4)

There was a nonsignificant main effect of task on mean bias scores $F(1, 83) = .46, p = .501, \eta_p^2 = .01$, a nonsignificant main effect of drink on mean bias scores $F(1, 83) = .44, p = .437, \eta_p^2 = .01$, but a significant task by drink interaction $F(1, 83) = 4.62, p = .034, \eta_p^2 = .05$. Follow-up paired samples t-tests revealed that mean bias scores for the alcohol AB measure did not differ between drink conditions $t(83) = 1.05, p = .297, d = 0.08$. However, mean bias scores on the food AB task were significantly greater in the alcohol condition relative to the placebo condition $t(83) = 2.28, p = .025, d = 0.18$. One-sample t-tests revealed that mean bias scores in the food AB task in the alcohol condition were significantly greater than zero $t(83) = 3.33, p < .001, d = 0.36$, but scores in the placebo-alcohol condition did not differ from zero $t(83) = 0.54, p = .593, d = 0.06$. For the alcohol AB task, mean bias scores did not differ from zero in the alcohol condition $t(83) = 0.42, p = .679, d = 0.05$, but were significantly greater than zero in the placebo condition $t(83) = 2.01, p = .047, d = 0.22$. There were no significant correlations between average post-drink alcohol urge scores and average alcohol-related AB ($r = .13, p = .229$) or between average post-drink snack urge scores and average food-related AB scores ($r = -.07, p = .558$).

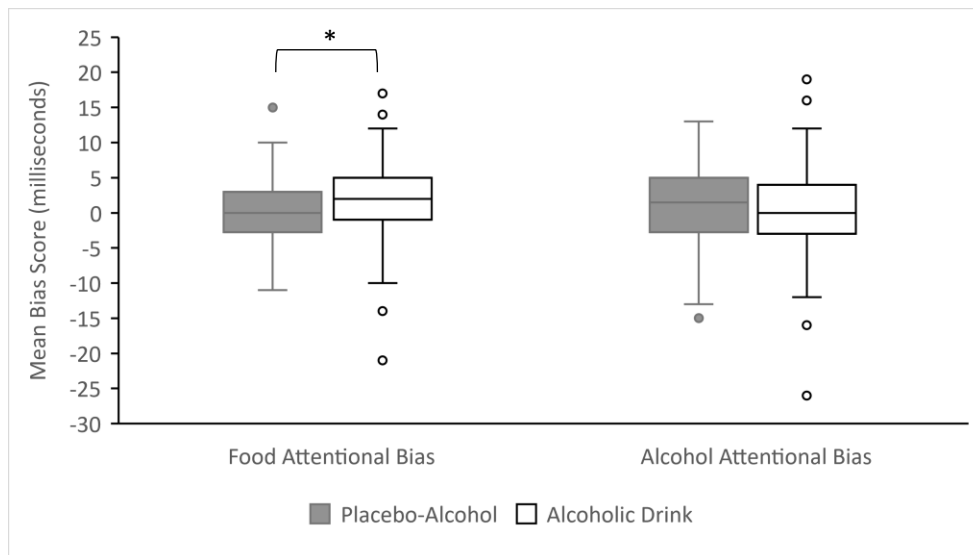


Figure 4. Boxplot displaying mean bias scores split by AB task and drink condition. Note: $* p = .025$. For food attentional bias, positive scores indicate greater fixation duration towards food images relative to control images. For alcohol attentional bias, positive scores indicate greater reaction time towards alcohol images relative to control images. Dots indicate outliers.

3.2.3 Caloric Intake (Figure 5)

There was a greater number of calories consumed from the taste test in the alcohol condition compared with the placebo condition $t(83) = 4.67, p < .001, d = 0.36$. Similarly, there was greater total caloric intake in the alcohol condition compared with the placebo condition $t(83) = 15.11, p < .001, d = 1.17$. The moderation analysis revealed that DEBQ restraint scores did not moderate the effect of drink type on food intake $b = -15.03 [-57.85, 27.80], SE = 21.53, t(82) = 0.70, p = .487$.

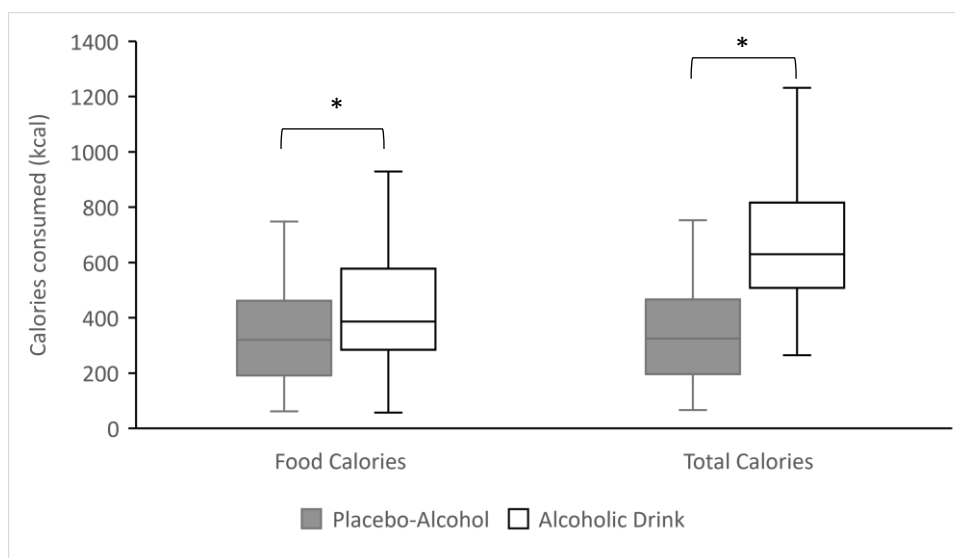


Figure 5. Boxplot displaying number of calories consumed during the *ad libitum* taste test (food calories) and combined with calories consumed from the test drink (total calories), split by condition Note: * $p < .001$. Dots indicate outliers

3.2.4 Appetite Ratings (Figure 6a)

There was a significant main effect of drink on appetite ratings $F(1, 83) = 5.67, p = .019, \eta_p^2 = .06$, with consumption of the alcoholic drink producing greater appetite ratings. There was also a significant main effect of time $F(1.79, 148.80) = 45.50, p < .001, \eta_p^2 = .35$. Pairwise comparisons revealed that baseline appetite ratings were significantly lower than post-drink ratings ($p < .001$; mean difference = 23.67; 95% CI [-32.04, -15.31]) but were significantly greater than post-taste test ratings

($p = .013$; mean difference = 13.21; 95% CI [2.17, 24.25]). Post-drink ratings were significantly greater than post-taste test ratings ($p < .001$; mean difference = 36.89; 95% CI [27.77, 46.01]). Lastly, there was no significant drink by time interaction $F(2,166) = 0.75, p = .474, \eta_p^2 = .01$.

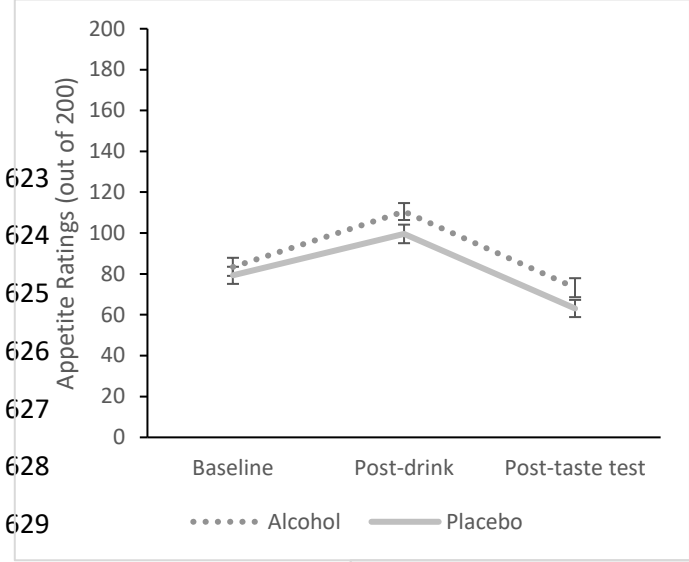
3.2.5 Snack Urge Ratings (Figure 6b)

There was a main effect of drink $F(1,83) = 10.54, p = .002, \eta_p^2 = .11$, with those in the alcohol condition reporting greater snack urge. There was also a significant main effect of time $F(1.46, 121.54) = 13.13, p < .001, \eta_p^2 = .14$, and a significant drink by time interaction $F(1.85, 153.49) = 7.08, p = .002, \eta_p^2 = .08$. See Figure 6b for comparisons of drink condition differences between time points.

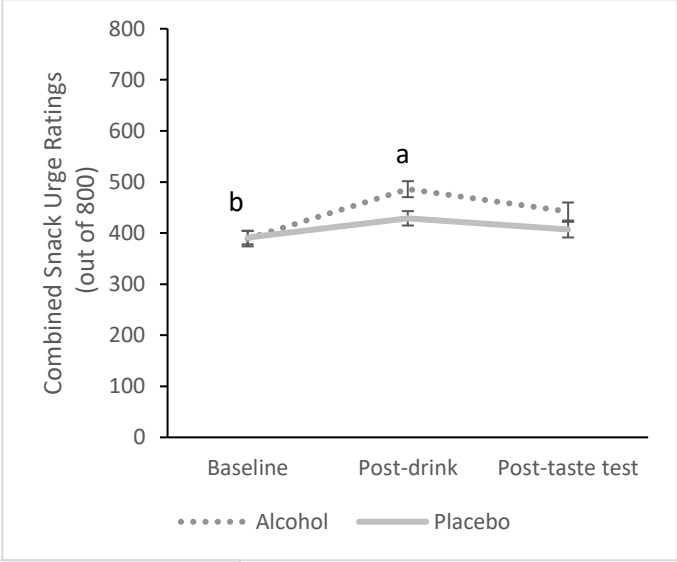
3.2.6 Alcohol Urge Ratings (Figure 6c)

There was a significant main effect of drink $F(1,83) = 31.63, p < .001, \eta_p^2 = .28$, with significantly greater alcohol urge ratings in the alcohol condition. There was also a significant main effect of time $F(1.82, 32.79) = 30.85, p < .001, \eta_p^2 = .27$ and a significant drink by time interaction $F(2,166) = 21.00, p < .001, \eta_p^2 = .20$. See Figure 6c for comparisons of drink condition differences between time points.

A



B



C

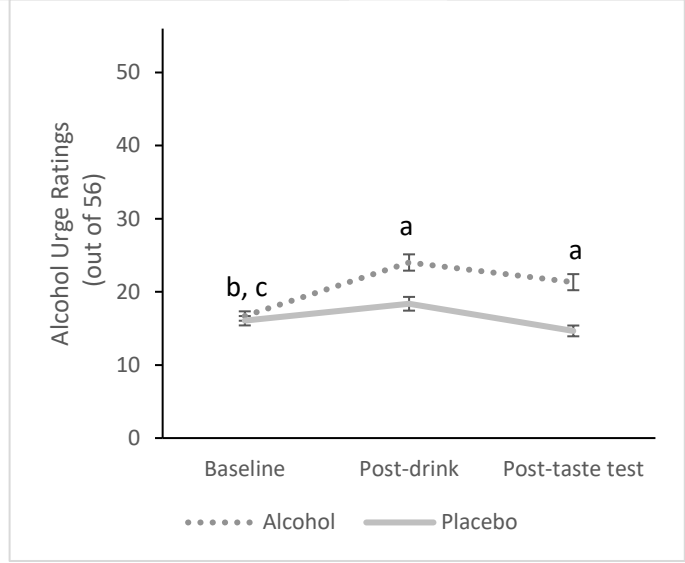


Figure 6 Appetite (6a), Snack urge (6b) and Alcohol urge ratings (6c) split by condition and across each time point. (Mean \pm SEM) Note: Letters refer to Bonferroni corrected pairwise comparisons which compare difference scores between drink conditions across each time point ($p < .05$): a = different from baseline difference scores; b = different from post-drink difference scores; c = different from post-taste test difference scores.

3.2.7 Unit Estimation

A one-sample t-test revealed that the number of units estimated to be in the placebo drink was significantly greater than zero $t(83) = 11.55, p < .001, d = 1.26$. A paired samples t-test showed that the number of units estimated to be in the alcoholic drink (4.70 ± 2.44) was significantly greater than the number of units estimated to be in the placebo drink (1.30 ± 1.03) $t(83) = 12.38, p < .001, d = 1.35$.

3.2.8 Predictors of change in food intake

The regression analysis was performed to test whether motor impulsivity, differences in food-related AB between conditions, and the interaction between them, predicted change in food intake across the two conditions. The regression model predicted 13.9% of variance in change in food intake, adjusted $R^2 = .14$, $F(6, 77) = 2.08$, $p = .065$. Dietary restraint ($\beta = -.06$, $p = .635$), BMI ($\beta = .31$, $p = .014$) and food-related AB in the placebo condition ($\beta = -.20$, $p = .168$) predicted 9.6% of variance. After controlling for these variables, step 2 predicted 4.3% of variance, with no significant predictor variables: change in food-related AB ($\beta = -.14$, $p = .314$); motor impulsivity ($\beta = -.18$, $p = .101$); change in food-related AB x motor impulsivity ($\beta < .01$, $p = .992$).

4. General Discussion

Collectively, findings from Studies 1 and 2 substantially differ. Study 1 failed to show any alcohol-induced increases relating to both implicit and explicit measures of food reward and food intake. Conversely, in Study 2, alcohol consumption enhanced snack urge ratings, food-related AB and food intake, along with increases in alcohol urge ratings. Taken together, findings from both studies suggests that alcohol intoxication increases appetitive motivational states, food-related AB and food intake, but only when administered above a certain dose (in this case 0.6 g/kg). This seemingly dose-dependent response is in line with previous research by Caton et al. (2004), who demonstrated that food intake was significantly greater after consumption of 4 UK units of alcohol compared with consumption of 1 UK unit. Results from the explicit measures of food reward are consistent with other studies which have shown that an alcohol dose of 0.6 g/kg is sufficient to increase snack urge ratings (Rose et al., 2015). Food intake also significantly increased after alcohol consumption, which has been demonstrated in several studies (see Kwok et al., 2019 for review). Dietary restraint did not moderate this effect, suggesting that those with higher levels of dietary restraint (when measured using the DEBQ) are not more susceptible to alcohol-induced increase in food intake. This is in line with previous research which has also failed to demonstrate that restrained individuals are more susceptible to alcohol-induced overeating (Christiansen et al., 2016a; Poppitt et al., 1996; Ouwens et al., 2003). However, as this was a secondary analysis, our study was not specifically powered to test for moderation by dietary restraint. Therefore these findings need to be treated with caution.

The food-related AB findings in Study 2 reveal that in contrast to previous research (Monem & Fillmore, 2019), alcohol intoxication can increase the magnitude of food-related AB. This discrepancy in findings may be explained by the use of a different AB task. As mentioned, the null finding of Monem and Fillmore (2019) may have been due to alcohol-induced impairments to visual performance, as their measure of AB used concurrent eye-tracking. Impairments to the ocular system are more pronounced at higher doses of alcohol (Abroms, Gottlob, & Fillmore, 2006; Rohrbaugh et al., 1988). The Stroop task used in the current study did not use ocular behaviour as its outcome measure and may therefore have been better suited to the current dose and allowed an AB effect to be detected. However, this suggestion remains speculative and further research should elucidate whether such an effect is dependent on the type of AB measure used.

Study 2 failed to show an alcohol-induced increase in alcohol-related AB. Although unexpected, this finding is in line with previous studies which have shown that alcohol consumption fails to enhance AB towards alcohol cues at doses of 0.6 g/kg (Duka & Townshend, 2004) or 0.65 g/kg (Monem & Fillmore, 2019), but does increase self-reported urge to drink (e.g. Rose & Duka, 2006 – 0.6 g/kg). Overall, this suggests that alcohol consumption increases appetitive motivation for alcohol, but that different assessment procedures may be focusing on different aspects of motivation and/or value towards certain stimuli.

Relatedly, the present findings raise questions regarding the construct validity of AB in the context of food reward. Theories suggest that AB is, in part, indicative of appetitive motivational states (Field et al., 2016). However, there was no significant correlation between measures of food motivational state and AB in either study. This null finding is likely due to insufficient statistical power, as we were not powered to detect a small correlational effect – findings from a recent meta-analysis has shown the association between food cravings and food-related AB to be $r = 0.13$ (Hardman et al., 2020). Nevertheless, the present findings suggest that AB should not be used as an index of food reward in isolation. Future research which aims to measure changes in AB should do so alongside other measures of food-related motivational states.

Contrary to our prediction, in Study 2 there was no interaction between motor impulsivity and change in food-related AB as a predictor of change in food intake. This finding does not support a dual-process model of eating behaviour within the context of acute alcohol consumption, which predicts that overeating is determined by an interaction of bottom-up (food reward responsivity) and top-down (impulsivity) processes. One explanation for this null finding could be due to alcohol intoxication in itself impairing state components of impulsivity at similar doses to those used in the present study (Christiansen et al., 2016a; Fillmore & Vogel-Sprott, 1999; Mulvihill, Skilling, & Vogel-Sprott, 1997). Therefore, the predictive power of trait motor impulsivity may have been masked by alcohol-induced changes in state behaviours (i.e., after alcohol consumption, impulsive behaviours may have increased and therefore may have become level across all participants within this condition). Future studies may wish to investigate if alcohol-induced changes in state impulsivity interact with food reward to predict changes in food intake.

There were some limitations with the current studies. Firstly, the two studies were not perfectly matched on all methodological components. For example, Study 2 implemented an absorption period double the length to Study 1. This was done to avoid participants feeling satiated after consumption of the test drink, as the volume of liquid consumed in Study 2 was greater due to the implementation of a larger alcohol dose. Another methodological difference was the type of AB measure used. This was changed because, as previously mentioned, it was more appropriate to use response latency rather than ocular attention as the outcome measure, when implementing a higher alcohol dose. A second limitation is that Study 2 did not test an equal number of men and women. This may be problematic if alcohol affects food intake differently in men and women, however a recent meta-analysis has shown that alcohol-induced increases in eating occurs in both men and women (Kwok et al., 2019). Thirdly, we did not measure how much participants liked each test drink. It is possible that a difference in liking of the drinks may have affected subsequent food intake. Fourthly, expectancy effects were not consistent across drink conditions. In Study 2, participants correctly believed that they had consumed more units in the alcoholic drink condition relative to the placebo condition. As changes to appetite-related behaviours can be affected by expectancy effects

alone (Christiansen et al., 2013; Christiansen et al., 2016b; Polivy & Herman, 1976a, 1976b; Yeomans & Phillips, 2002), the significant increase in snack urge ratings, AB and food intake may result from a combination of expectancy and pharmacological effects. However, the estimated number of units in the placebo condition was significantly greater than zero. Therefore, believing that both drinks contained some amount of alcohol may have limited differences in expectancy effects. Future research should systematically manipulate expectancy effects by comparing an alcohol-free placebo with a control drink in order to isolate alcohol-related expectancy effects on eating behaviours, in the absence of actual alcohol consumption. A fifth limitation is that both studies implemented a single-blind design, meaning that the experimenter knew which drink participants would receive, therefore failing to minimise the risk of experimenter bias. Finally, the alcoholic and caloric content of the test drinks were not matched across participants. It could be argued that because the caloric content in the alcoholic drink was greater than in the placebo, appetite levels across conditions may have differed. However, data from both studies show that appetite ratings were not suppressed by greater caloric intake from the test drink, suggesting that this difference in caloric intake did not affect findings. Instead, the alcohol dose was adjusted by bodyweight in order to achieve a better matched breath alcohol concentration across participants. This is important because evidence from the present studies and previous research (e.g., Caton et al., 2004) suggest that an alcohol-induced effect on eating behaviour is dependent on the dosage of alcohol. Therefore, it was essential that participants received a dose which produced a more consistent breath alcohol concentration across participants. If the alcohol dose was unadjusted, some participants may not have received a dosage high enough to produce changes in behaviour.

In summary, the two studies suggest that alcohol's ability to affect indices of food reward may be dose-dependent - at lower doses of alcohol consumption, changes to appetitive motivational states appear to be minimal. However, a direct comparison between different alcohol doses is needed to confirm this. Importantly, both Studies 1 and 2 found an alcohol-induced increase in total caloric intake, which may increase the risk of weight gain if these calories are not compensated for. A higher dose of alcohol consumption significantly increased food-related AB, motivational states, and food

intake. This adds to the continually growing body of evidence which demonstrates that acute alcohol consumption alters behavioural states relevant to eating behaviour, and further implicates drinking behaviour as an important risk factor for weight gain through a lack of caloric compensation after alcohol has been consumed.

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Consent for publication: Not applicable.

Availability of data and material: The datasets generated and analysed for Study 1 and Study 2 are available on the Open Science Framework repository (<https://osf.io/cnaxr/>).

References

Abrams, B. D., Gottlob, L. R., & Fillmore, M. T. (2006). Alcohol effects on inhibitory control of attention: distinguishing between intentional and automatic mechanisms. *Psychopharmacology*, 188(3), 324-334.

786 Appelhans, B. M. (2009). Neurobehavioral inhibition of reward-driven feeding: implications for
787 dieting and obesity. *Obesity*, 17(4), 640-647.

788 Appelhans, B. M., Woolf, K., Pagoto, S. L., Schneider, K. L., Whited, M. C., & Liebman, R. (2011).
789 Inhibiting food reward: delay discounting, food reward sensitivity, and palatable food intake
790 in overweight and obese women. *Obesity*, 19(11), 2175-2182.

791 Ataya, A. F., Adams, S., Mullings, E., Cooper, R. M., Attwood, A. S., & Munafò, M. R. (2012).
792 Internal reliability of measures of substance-related cognitive bias. *Drug and alcohol*
793 *dependence*, 121(1-2), 148-151.

794 Baines, L., Field, M., Christiansen, P., & Jones, A. (2019). The effect of alcohol cue exposure and
795 acute intoxication on inhibitory control processes and ad libitum alcohol
796 consumption. *Psychopharmacology*, 236(7), 2187-2199.

797 Blundell, J., De Graaf, C., Hulshof, T., Jebb, S., Livingstone, B., Lluich, et al. (2010). Appetite
798 control: methodological aspects of the evaluation of foods. *Obesity reviews*, 11(3), 251-270.

799 Bohn, M. J., Krahn, D. D., & Staehler, B. A. (1995). Development and initial validation of a measure
800 of drinking urges in abstinent alcoholics. *Alcoholism: Clinical and Experimental*
801 *Research*, 19(3), 600-606.

802 Brunstrom, J. M., Yates, H. M., & Witcomb, G. L. (2004). Dietary restraint and heightened reactivity
803 to food. *Physiology & behavior*, 81(1), 85-90.

804 Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R.
805 L. (2009). Obese adults have visual attention bias for food cue images: evidence for altered
806 reward system function. *International Journal of Obesity*, 33(9), 1063-1073.

807 Caton, S. J., Ball, M., Ahern, A., & Hetherington, M. M. (2004). Dose-dependent effects of alcohol
808 on appetite and food intake. *Physiology & behavior*, 81(1), 51-58.

809 Caton, S. J., Bate, L., & Hetherington, M. M. (2007). Acute effects of an alcoholic drink on food
810 intake: aperitif versus co-ingestion. *Physiology & behavior*, 90(2), 368-375.

811 Caton, S. J., Nolan, L. J., & Hetherington, M. M. (2015). Alcohol, appetite and loss of
812 restraint. *Current obesity reports*, 4(1), 99-105.

813 Chapman, C. D., Benedict, C., Brooks, S. J., & Schiöth, H. B. (2012). Lifestyle determinants of the
814 drive to eat: a meta-analysis. *The American journal of clinical nutrition*, 96(3), 492-497.

815 Christiansen, P., Jennings, E., & Rose, A. K. (2016b). Anticipated effects of alcohol stimulate craving
816 and impair inhibitory control. *Psychology of Addictive Behaviors*, 30(3), 383.

817 Christiansen, P., Rose, A. K., Cole, J. C., & Field, M. (2013). A comparison of the anticipated and
818 pharmacological effects of alcohol on cognitive bias, executive function, craving and ad-lib
819 drinking. *Journal of psychopharmacology*, 27(1), 84-92.

820 Christiansen, P., Rose, A., Randall-Smith, L., & Hardman, C. A. (2016a). Alcohol's acute effect on
821 food intake is mediated by inhibitory control impairments. *Health Psychology*, 35(5), 518.

822 Duka, T., Jackson, A., Smith, D. C., & Stephens, D. N. (1999). Relationship of components of an
823 alcohol interoceptive stimulus to induction of desire for alcohol in social drinkers.
824 *Pharmacology Biochemistry and Behavior*, 64(2), 301-309.

825 Duka, T., Tasker, R., & Stephens, D. N. (1998). Alcohol choice and outcome expectancies in social
826 drinkers. *Behavioural pharmacology*.

827 Duka, T., & Townshend, J. M. (2004). The priming effect of alcohol pre-load on attentional bias to
828 alcohol-related stimuli. *Psychopharmacology*, 176(3-4), 353-361.

829 Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G* Power
830 3.1: Tests for correlation and regression analyses. *Behavior research methods*, 41(4), 1149-
831 1160.

832 Field, M., Hogarth, L., Bleasdale, D., Wright, P., Fernie, G., & Christiansen, P. (2011). Alcohol
833 expectancy moderates attentional bias for alcohol cues in light drinkers. *Addiction*, 106(6),
834 1097-1103.

835 Field, M., Werthmann, J., Franken, I., Hofmann, W., Hogarth, L., & Roefs, A. (2016). The role of
836 attentional bias in obesity and addiction. *Health Psychology*, 35(8), 767.

837 Fillmore, M. T., & Vogel-Sprott, M. (1999). An alcohol model of impaired inhibitory control and its
838 treatment in humans. *Experimental and clinical psychopharmacology*, 7(1), 49.

839 Gearhardt, A. N., Treat, T. A., Hollingworth, A., & Corbin, W. R. (2012). The relationship between
840 eating-related individual differences and visual attention to foods high in added fat and
841 sugar. *Eating behaviors*, 13(4), 371-374.

842 Graham, R., Hoover, A., Ceballos, N. A., & Komogortsev, O. (2011). Body mass index moderates
843 gaze orienting biases and pupil diameter to high and low calorie food images. *Appetite*, 56(3),
844 577-586.

845 Hardman, C. A., Jones, A., Burton, S., Duckworth, J. J., McGale, L. S., Mead, B. R., ... &
846 Werthmann, J. (2020). Food-related attentional bias and its associations with appetitive
847 motivation and body weight: A systematic review and meta-analysis. *Appetite*, 104986.

848 Hardman, C. A., Jones, A., Field, M., & Werthmann, J. (2018). A meta-analytic investigation of the
849 associations between food-related attentional bias, body weight and appetite. *Appetite*, 123,
850 451.

851 Hardman, C. A., Rogers, P. J., Dallas, R., Scott, J., Ruddock, H. K., & Robinson, E. (2015). "Food
852 addiction is real". The effects of exposure to this message on self-diagnosed food addiction
853 and eating behaviour. *Appetite*, 91, 179-184.

854 Hardman, C. A., Scott, J., Field, M., & Jones, A. (2014). To eat or not to eat. The effects of
855 expectancy on reactivity to food cues. *Appetite*, 76, 153-160.

856 Higgs, S., & Woodward, M. (2009). Television watching during lunch increases afternoon snack
857 intake of young women. *Appetite*, 52(1), 39-43.

858 Karyadi, K. A., & Cyders, M. A. (2019). Preliminary support for the role of alcohol cues in food
859 cravings and attentional biases. *Journal of health psychology*, 24(6), 812-822.

860 Jones, A., Hogarth, L., Christiansen, P., Rose, A. K., Martinovic, J., & Field, M. (2012). Reward
861 expectancy promotes generalized increases in attentional bias for rewarding stimuli. *The*
862 *Quarterly Journal of Experimental Psychology*, 65(12), 2333-2342.

863 Kakoschke, N., Kemps, E., & Tiggemann, M. (2015). Combined effects of cognitive bias for food
864 cues and poor inhibitory control on unhealthy food intake. *Appetite*, 87, 358-364.

865 Keesman, M., Aarts, H., Vermeent, S., Häfner, M., & Papies, E. K. (2016). Consumption simulations
866 induce salivation to food cues. *PloS one*, 11(11), e0165449.

867 Kwok, A., Dordevic, A. L., Paton, G., Page, M. J., & Truby, H. (2019). Effect of alcohol consumption
868 on food energy intake: A systematic review and meta-analysis. *British Journal of Nutrition*,
869 121(5), 481-495.

870 Mattes, R. D. (1996). Dietary compensation by humans for supplemental energy provided as ethanol
871 or carbohydrate in fluids. *Physiology & behavior*, 59(1), 179-187.

872 Meule, A., & Platte, P. (2016). Attentional bias toward high-calorie food-cues and trait motor
873 impulsivity interactively predict weight gain. *Health psychology open*, 3(1),
874 2055102916649585.

875 Mogg, K., Bradley, B. P., Hyare, H., & Lee, S. (1998). Selective attention to food-related stimuli in
876 hunger: are attentional biases specific to emotional and psychopathological states, or are they
877 also found in normal drive states?. *Behaviour research and therapy*, 36(2), 227-237.

878 Monem, R., & Fillmore, M. T. (2019). Alcohol administration reduces attentional bias to alcohol-
879 related but not food-related cues: Evidence for a satiety hypothesis. *Psychology of Addictive*
880 *Behaviors*, 33(8), 677-684.

881 Montoya, A. K., & Hayes, A. F. (2017). Two condition within-participant statistical mediation
882 analysis: A path-analytic framework. *Psychological Methods*, 22(1), 6-27.

883 Moser, A., Heide, W., & Kömpf, D. (1998). The effect of oral ethanol consumption on eye
884 movements in healthy volunteers. *Journal of Neurology*, 245(8), 542-550.

885 Mulvihill, L. E., Skilling, T. A., & Vogel-Sprott, M. (1997). Alcohol and the ability to inhibit
886 behavior in men and women. *Journal of studies on alcohol*, 58(6), 600-605.

887 Nederkoorn, C., Guerrieri, R., Havermans, R. C., Roefs, A., & Jansen, A. (2009). The interactive
888 effect of hunger and impulsivity on food intake and purchase in a virtual
889 supermarket. *International journal of obesity*, 33(8), 905-912.

890 Nederkoorn, C., Houben, K., Hofmann, W., Roefs, A., & Jansen, A. (2010). Control yourself or just
891 eat what you like? Weight gain over a year is predicted by an interactive effect of response
892 inhibition and implicit preference for snack foods. *Health Psychology*, 29(4), 389.

893 Nijs, I. M., Franken, I. H., & Muris, P. (2010). Food-related Stroop interference in obese and normal-
894 weight individuals: Behavioral and electrophysiological indices. *Eating behaviors*, 11(4),
895 258-265.

896 Nijs, I. M., Muris, P., Euser, A. S., & Franken, I. H. (2010). Differences in attention to food and food
897 intake between overweight/obese and normal-weight females under conditions of hunger and
898 satiety. *Appetite*, 54(2), 243-254.

899 Ouwens, M. A., van Strien, T., & van der Staak, C. P. (2003). Absence of a disinhibition effect of
900 alcohol on food consumption. *Eating Behaviors*, 4(4), 323-332.

901 Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness
902 scale. *Journal of clinical psychology*, 51(6), 768-774.

903 Peirce, J., Gray, J. R., Simpson, S., MacAskill, M., Höchenberger, R., Sogo, H., ... & Lindeløv, J. K.
904 (2019). PsychoPy2: Experiments in behavior made easy. *Behavior research methods*, 51(1),
905 195-203.

906 Polivy, J., & Herman, C. P. (1976a). Effects of alcohol on eating behavior: Influence of mood and
907 perceived intoxication. *Journal of Abnormal Psychology*, 85(6), 601.

- 908 Polivy, J., & Herman, C. P. (1976b). The effects of alcohol on eating behavior: disinhibition or
909 sedation?. *Addictive Behaviors*, 1(2), 121-125.
- 910 Poppitt, S. D., Eckhardt, J. W., McGonagle, J., Murgatroyd, P. R., & Prentice, A. M. (1996). Short-
911 term effects of alcohol consumption on appetite and energy intake. *Physiology &*
912 *behavior*, 60(4), 1063-1070.
- 913 Price, M., Higgs, S., & Lee, M. (2015). Self-reported eating traits: underlying components of food
914 responsivity and dietary restriction are positively related to BMI. *Appetite*, 95, 203-210.
- 915 Robinson, E., Haynes, A., Hardman, C. A., Kemps, E., Higgs, S., & Jones, A. (2017). The bogus taste
916 test: Validity as a measure of laboratory food intake. *Appetite*, 116, 223-231.
- 917 Rogers, P. J., & Brunstrom, J. M. (2016). Appetite and energy balancing. *Physiology &*
918 *behavior*, 164, 465-471.
- 919 Rogers, P. J., & Hardman, C. A. (2015). Food reward. What it is and how to measure it. *Appetite*, 90,
920 1-15.
- 921 Rohrbaugh, J. W., Stapleton, J. M., Parasuraman, R., Frowein, H. W., Adinoff, B., Varner, J. L., ... &
922 Linnoila, M. (1988). Alcohol intoxication reduces visual sustained attention.
923 *Psychopharmacology*, 96(4), 442-446.
- 924 Rollins, B. Y., Dearing, K. K., & Epstein, L. H. (2010). Delay discounting moderates the effect of
925 food reinforcement on energy intake among non-obese women. *Appetite*, 55(3), 420-425.
- 926 Rose, A. K., & Duka, T. (2006). Effects of dose and time on the ability of alcohol to prime social
927 drinkers. *Behavioural Pharmacology*, 17(1), 61-70.
- 928 Rose, A. K., Hardman, C. A., & Christiansen, P. (2015). The effects of a priming dose of alcohol and
929 drinking environment on snack food intake. *Appetite*, 95, 341-348.
- 930 Ruddock, H. K., Field, M., & Hardman, C. A. (2017). Exploring food reward and calorie intake in
931 self-perceived food addicts. *Appetite*, 115, 36-44.
- 932 Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of
933 the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early
934 detection of persons with harmful alcohol consumption II. *Addiction*, 88, 791– 804. [http://dx](http://dx.doi.org/10.1111/j.1360-0443.1993.tb02093.x)
935 [.doi.org/10.1111/j.1360-0443.1993.tb02093.x](http://dx.doi.org/10.1111/j.1360-0443.1993.tb02093.x)
- 936 Schrieks, I. C., Stafleu, A., Griffioen-Roose, S., de Graaf, C., Witkamp, R. F., Boerriqter-Rijneveld,
937 R., & Hendriks, H. F. (2015). Moderate alcohol consumption stimulates food intake and food
938 reward of savoury foods. *Appetite*, 89, 77-83.
- 939 Schmitz, F., Naumann, E., Trentowska, M., & Svaldi, J. (2014). Attentional bias for food cues in
940 binge eating disorder. *Appetite*, 80, 70-80.
- 941 Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive
942 biases and craving in heavy drinkers. *Psychopharmacology*, 197(1), 169-178.
- 943 Sayon-Orea, C., Martinez-Gonzalez, M. A., & Bes-Rastrollo, M. (2011). Alcohol consumption and
944 body weight: a systematic review. *Nutrition Reviews*, 69(8), 419-431.
- 945 Sobell, L. C., & Sobell, M. B. (1990). Self-report issues in alcohol abuse: State of the art and future
946 directions. *Behavioral Assessment*, 12, 77–90.
- 947 Tapper, K., Pothos, E. M., & Lawrence, A. D. (2010). Feast your eyes: Hunger and trait reward drive
948 predict attentional bias for food cues. *Emotion*, 10(6), 949.

- van Ens, W., Schmidt, U., Campbell, I. C., Roefs, A., & Werthmann, J. (2019). Test-retest reliability of attention bias for food: Robust eye-tracking and reaction time indices. *Appetite*, 136, 86-92.
- Van Koningsbruggen, G. M., Stroebe, W., & Aarts, H. (2013). Successful restrained eating and trait impulsiveness. *Appetite*, 60, 81-84.
- van Strien, T., Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, 5, 295–315.
[http://dx.doi.org/10.1002/1098-108X\(198602\)5:2 295::AID-EAT22600502093.0.CO;2-T](http://dx.doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT22600502093.0.CO;2-T)
- Waters, A. J., Sayette, M. A., & Wertz, J. M. (2003). Carry-over effects can modulate emotional Stroop effects. *Cognition and Emotion*, 17(3), 501–509
- Werthmann, J., Roefs, A., Nederkoorn, C., & Jansen, A. (2013). Desire lies in the eyes: attention bias for chocolate is related to craving and self-endorsed eating permission. *Appetite*, 70, 81-89.
- Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can (not) take my eyes off it: Attention bias for food in overweight participants. *Health Psychology*, 30(5), 561.
- Wooley, O. W., & Wooley, S. C. (1981). Relationship of salivation in humans to deprivation, inhibition and the encephalization of hunger. *Appetite*, 2(4), 331-350.
- Yeomans, M. R. (2010a). Alcohol, appetite and energy balance: is alcohol intake a risk factor for obesity?. *Physiology & behavior*, 100(1), 82-89.
- Yeomans, M. R. (2010b). Short term effects of alcohol on appetite in humans. Effects of context and restrained eating. *Appetite*, 55(3), 565-573.
- Yeomans, M. R., Hails, N. J., & Nesic, J. S. (1999). Alcohol and the appetizer effect. *Behavioural pharmacology*, 10(2), 151-161.
- Yeomans, M. R., & Phillips, M. F. (2002). Failure to reduce short-term appetite following alcohol is independent of beliefs about the presence of alcohol. *Nutritional neuroscience*, 5(2), 131-139.

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