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

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Abstract

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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; Schrieks 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,

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

66 To date, little research has focused on how alcohol intoxication can alter food-related AB. One
67 study found that AB towards food cues was increased by smelling alcohol odours, in the absence of
68 alcohol consumption (Karyadi & Cyders, 2019). However, another study showed that the magnitude
69 of food-related AB did not differ between consumption of a placebo-alcohol, and alcoholic doses of
70 0.3 g/kg or 0.65 g/kg (Monem & Fillmore, 2019). However, this study was powered to detect only a
71 medium-to-large effect size, which may explain why no difference was found, as evidence suggests
72 that the relationship between food craving and food-related AB is small (Hardman et al., 2020). Given
73 these discrepant findings, the present research aimed to further investigate whether acute alcohol
74 consumption can increase AB towards food cues.

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

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

89 temporary change to dietary intentions (Caton, Nolan, & Hetherington, 2015). This effect was first
90 studied by Polivy and Herman (1976a; 1976b) who found that when restrained eaters were aware of
91 the presence of alcohol, their eating behaviour became disinhibited. Whereas, when restrained eaters
92 were unaware of the presence of alcohol, food intake was suppressed (relative to unrestrained
93 individuals), suggesting that alcohol-related expectancy effects may contribute towards disinhibited
94 eating in restrained individuals. However, subsequent research has been unable to demonstrate that
95 restrained eaters are more susceptible to alcohol-induced increases in food intake (Christiansen et al.,
96 2016a; Poppitt et al., 1996; Yeomans, 2010b; Yeomans, Hails, & Nescic, 1999), even when they are
97 made aware of the presence of alcohol (Ouwens et al., 2003). Taken together, restraint is an important
98 variable to take into consideration when conducting research on alcohol and food intake.

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2. Study 1

101 Overview

102 Study 1 investigated whether food reward (measured using self-report appetite, snack urge
103 ratings, salivary response to food, and AB towards food-cues) and *ad libitum* food intake would differ
104 between administration of a placebo-alcohol and an alcoholic drink (dose = 0.3 g/kg). This dose was
105 chosen because although Monem and Fillmore (2019) were unable to show an enhanced food AB at
106 0.3 g/kg, this same dose has been shown to enhance AB towards other appetitive stimuli (i.e., alcohol)
107 relative to a placebo-alcohol (Duka & Townshend, 2004; Schoenmakers et al., 2008).

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

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

116 **2.1 Method**

117 **2.1.1 Participants**

118 At the time of data collection, no previous published studies had investigated the difference in
119 food-related AB between consumption of an alcoholic drink and placebo, therefore the study was
120 powered to detect a small-to-medium effect size ($d_z = 0.39$) for differences in food-related AB
121 between drink conditions. Using G*Power 3.1 (Faul, Erdfelder, Buchner & Lang, 2009) and based on
122 80% power and an alpha level of 5%, 43 participants were required. Forty-four participants (men =
123 22) aged between 18 and 54 y (Mean = 25.55, standard deviation = 8.22), were recruited in order to
124 achieve full counterbalancing of drink order. Participants were recruited through online and email
125 advertisement, and word-of-mouth and were eligible to take part if they were aged 18 – 65 y, had no
126 history of food allergies or intolerances, were regular consumers of alcohol (consuming alcohol at
127 least once a week and drinking at least 10 UK alcohol units per week), and enjoyed consuming
128 cookies and tortilla chips, as these were used as test foods. Participants were excluded if: they wore
129 glasses to correct their vision (due to interference with the eye-tracking camera); had a current or past
130 alcohol use or eating disorder; had a current or recent illness that may increase sensitivity to alcohol
131 (e.g., cold and flu); were taking medication that may be affected by alcohol; were currently
132 breastfeeding or pregnant. Participants were also required to consume a light meal, low in fat, one
133 hour prior to the test session. All participants provided written informed consent to participate in the
134 experiment, which was approved by the University of Liverpool Health and Life Sciences Research
135 Ethics Committee. Participants were reimbursed through either course credits or a £10 shopping
136 voucher.

137 **2.1.2 Design**

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

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

142 **2.1.3 Measures**

143 *Beverage Preparation and Administration.*

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

150 *Pictorial Stimuli*

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

161 *Visual Probe Task (VPT)*

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

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

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

179 *Salivation:*

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

184 *Bogus taste-test.*

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

191 15 minutes to complete this task and were told to consume as much of the foods as they liked if they
192 finished before the 15-minute period. Taste-test consumption was calculated by subtracting the post
193 taste-test weight from the pre-taste-test weight. Grams consumed was converted to kilocalories. The
194 bogus taste-test has been shown to be a valid measure of food intake (Robinson et al., 2017).

195 *Dutch Eating Behavior Questionnaire.*

196 The Dutch Eating Behaviour Questionnaire (DEBQ; van Strien, Frijters, Bergers, & Defares,
197 1986) is a 33-item questionnaire measuring eating styles associated with being overweight. The three
198 subscales are restraint ($\omega = .92$), emotional eating ($\omega = .95$), and external eating ($\omega = .86$).

199 *Timeline Follow Back.*

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

202 *Alcohol Use Disorders Identification Test.*

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

206 *Snack Urge Scale.*

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

212 *Appetite Ratings.*

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

216 **2.1.4 Procedure**

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

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

242

243 2.1.5 Data reduction and analysis

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

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

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

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

276 2.2 Results

277 2.2.1 Participant characteristics

278 Participant characteristics are shown in Table 1.

279 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 TLF _B (in units)	19.68 \pm 13.37

281 2.2.2 Mean attentional bias scores (Figure 1)

282 As shown in Figure 1, there were no significant main effects of drink $F(1, 39) = 0.36, p =$
 283 $.551, \eta_p^2 = .01$, or pair type $F(1.24, 48.41) = 1.42, p = .246, \eta_p^2 = .04$, and no significant drink x pair
 284 interaction $F(1.24, 48.41) = 0.80, p = .400, \eta_p^2 = .02$. One-sample t-tests revealed that mean bias
 285 scores were significantly greater than zero on the palatable vs. control trials in both the alcohol $t(39) =$

286 3.14, $p = .003$, $d = 0.50$ and placebo condition $t(39) = 3.14$, $p = .003$, $d = 0.50$, and for
 287 unpalatable/control trials in the placebo condition $t(39) = 2.41$, $p = .021$, $d = 0.38$, but not for any
 288 other trial type on for either drink condition. There was no significant correlation between average
 289 post-drink snack urge ratings and average mean bias scores for palatable vs control trials $r < -.01$, $p =$
 290 $.993$.

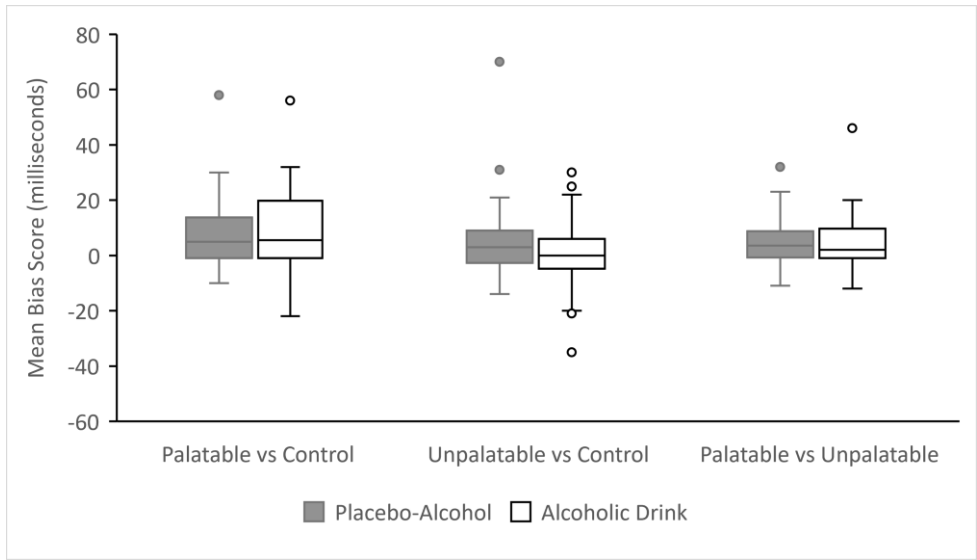


Figure 1. Boxplot displaying mean attentional bias scores split by pair type and drink condition. Positive scores indicate greater fixation duration towards palatable images for palatable vs control trials and palatable vs unpalatable trials. Positive scores indicate greater fixation duration towards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

301 2.2.3 Appetite Ratings (Figure 2a)

302 There was a significant main effect of time on appetite ratings $F(2.15, 90.36) = 47.25$, $p <$
 303 $.001$, $\eta_p^2 = .53$ (see Figure 2a for comparisons across time points). There was no main effect of
 304 condition $F(1, 42) = 1.20$, $p = .279$, $\eta_p^2 = .03$ or interaction between time and condition $F(2.45,$
 305 $103.09) = 1.22$, $p = .305$, $\eta_p^2 = .03$.

306

307 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.

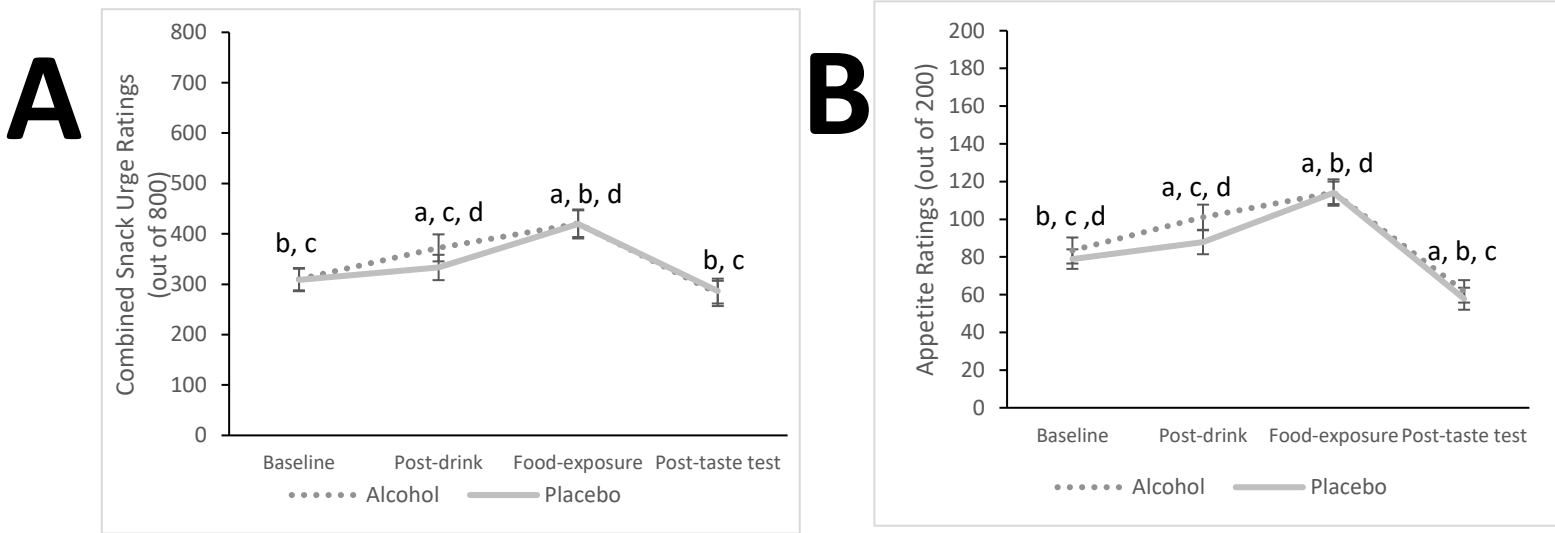


Figure 2. Snack Urge (2a) and Appetite ratings (2b) over time, by condition (Mean ± SEM). Letters refer to Bonferroni corrected pairwise comparisons breaking down significant differences ($p < .05$) between time points (collapsed across drink conditions): a = difference from baseline; b = difference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

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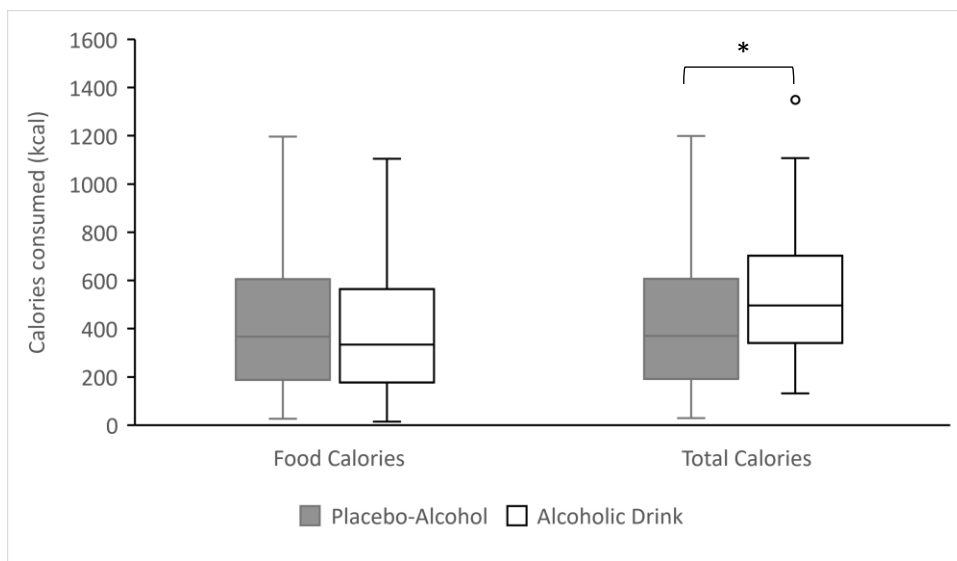
313 2.2.5 Salivation Measure

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.

321

322 2.2.6 Calorie Measures (Figure 3)

323 Paired sample t-tests revealed no significant difference between conditions for the amount of
 324 food calories consumed during the taste test, $t(43) = -0.92, p = .361, d = 0.14$. However there was a
 325 significant difference in total calories consumed (drink calories combined with food calories) $t(43) =$
 326 $3.37, p = .002, d = 0.51$, with participants in the alcohol condition consuming significantly more
 327 calories overall relative to the placebo condition. The moderation analysis revealed that DEBQ
 328 restraint scores did not moderate the effect of drink type on food intake $b = 87.23 [-15.45, 189.91], SE$
 329 $= 50.88, t(42) = 1.71, p = .094$.



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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

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2.3. Interim Discussion

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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

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

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

375 An additional aim was to examine the role of top-down and bottom-up processes in driving
376 alcohol-induced increases in food intake. Dual-process models argue that eating behaviour is
377 determined by an interaction of bottom-up drives relating to motivational orientation and food reward,
378 and top-down cognitive control (Appelhans, 2009). Several studies have demonstrated the combined
379 effect of food reward and impulsivity (indicating weaker top-down control) in predicting eating
380 behaviour and weight change (Appelhans et al., 2011; Kakoschke et al., 2015; Nederkoorn et al.,
381 2009; Nederkoorn et al., 2010; Price, Higgs, & Lee, 2015; Rollins, Dearing, & Epstein, 2010). For
382 example, Nederkoorn et al. (2009) showed that poor response inhibition (a type of impulsivity) was
383 related to overeating only when desire to eat was also high. It is therefore possible that top-down
384 control and bottom-up reward processes interact to facilitate alcohol-induced overeating. To test this,
385 Study 2 investigated whether trait impulsivity (specifically motor impulsivity) and alcohol-induced
386 changes in food-related AB (using the pictorial modified Stroop task) could interactively predict
387 changes in food intake across drink conditions. We chose motor impulsivity as our measure of
388 (weaker) top-down control because previous research has shown this to be positively associated with
389 disinhibited eating and BMI (Price et al., 2015; Van Koningsbruggen et al., 2013) and, most
390 relevantly, to interact with food-related AB to predict weight gain (Meule & Platte, 2016).

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

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

399 **3.1 Method**

400 3.1.1 Participants

401 The study was powered based on an earlier version of Hardman et al.'s (2020) meta-analysis
402 (Hardman et al., 2018) which found a correlation of $r = 0.14$ between food-related AB and food
403 craving. Based on 80% power and an alpha level of 5%, 81 participants would be needed in order to
404 detect the same effect size between drink conditions. 84 participants (men = 13) aged between 18 and
405 26 y ($M = 18.75$; $SD = 1.13$) completed both sessions in order to counterbalance the order of drink
406 condition and the order of target and neutral blocks in the Stroop task (see measures section for
407 further details). Six additional participants completed session one, but did not return for session 2, and
408 were therefore excluded from all analyses. Participants were recruited through the university
409 undergraduate credit scheme. Inclusion criteria was the same as in Study 1 with the following
410 changes: participants were able to take part if they wore glasses to correct their vision, but participants
411 were excluded if they were colour-blind. All participants provided written informed consent to
412 participate in the experiment, which was approved by the University of Liverpool Health and Life
413 Sciences Research Ethics Committee. Participants were reimbursed through course credits. The
414 method and analysis strategy for this study were pre-registered on the Open Science Framework
415 (<https://osf.io/cnaxr/>).

416 3.1.2 Design

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

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

421 3.1.3 Measures

422 *Modified Stroop Task*

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

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

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

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

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

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

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

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

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

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

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

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

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

482 *Beverage Preparation and Administration.*

483 This was the same as in Study 1 with the following changes: the alcohol dose was at 0.6 g/kg
484 (5.35 UK units of alcohol for a participant weighing 70 kg); participants consumed the test drink in
485 three separate portions, each served in set 5-minute intervals, meaning that participants consumed the
486 test drink in 15 minutes.

487 The following measures were used as in Study 1: DEBQ (restraint $\omega = .96$; emotional eating
488 $\omega = .96$; external eating $\omega = .92$); TLFB; AUDIT ($\omega = .74$), Snack Urge Scale, Appetite Ratings,
489 bogus taste-test.

490

491 3.1.4 Procedure

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

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

504

505 3.1.5 Data Analysis

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

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

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

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

535 3.2 Results

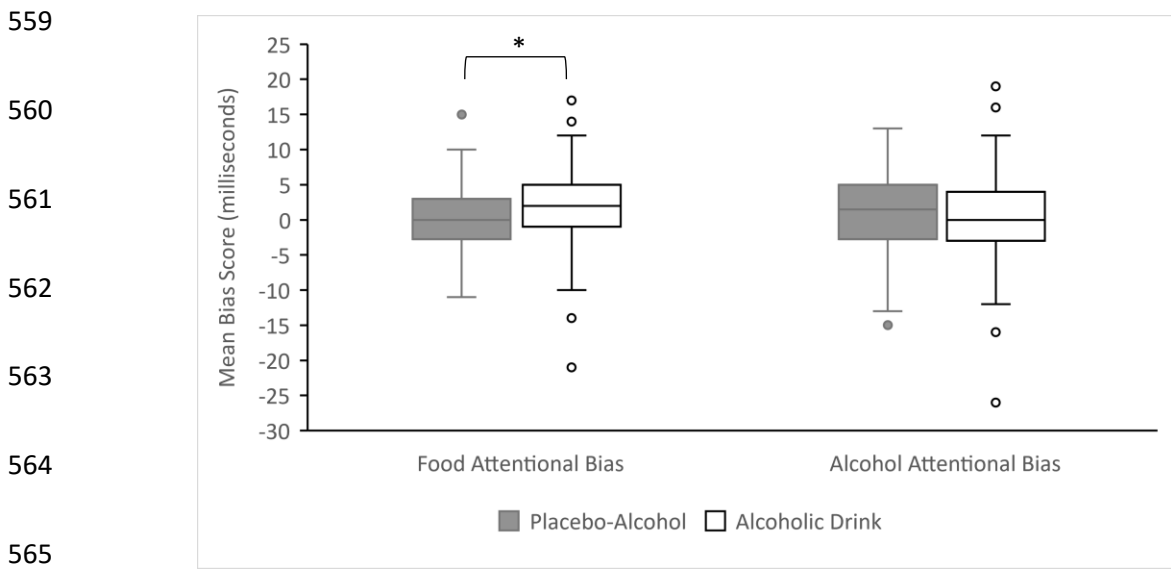
536 3.2.1 Participant characteristics are shown in Table 2.

537 Table 2. Sample characteristics (mean ± SD).

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

544 3.2.2 Modified Stroop (Figure 4)

545 There was a nonsignificant main effect of task on mean bias scores $F(1, 83) = .46, p = .501,$
 546 $\eta_p^2 = .01,$ a nonsignificant main effect of drink on mean bias scores $F(1, 83) = .44, p = .437, \eta_p^2 = .01,$
 547 but a significant task by drink interaction $F(1, 83) = 4.62, p = .034, \eta_p^2 = .05.$ Follow-up paired
 548 samples t-tests revealed that mean bias scores for the alcohol AB measure did not differ between
 549 drink conditions $t(83) = 1.05, p = .297, d = 0.08.$ However, mean bias scores on the food AB task
 550 were significantly greater in the alcohol condition relative to the placebo condition $t(83) = 2.28, p =$
 551 $.025, d = 0.18.$ One-sample t-tests revealed that mean bias scores in the food AB task in the alcohol
 552 condition were significantly greater than zero $t(83) = 3.33, p < .001, d = 0.36,$ but scores in the
 553 placebo-alcohol condition did not differ from zero $t(83) = 0.54, p = .593, d = 0.06.$ For the alcohol AB
 554 task, mean bias scores did not differ from zero in the alcohol condition $t(83) = 0.42, p = .679, d =$
 555 $0.05,$ but were significantly greater than zero in the placebo condition $t(83) = 2.01, p = .047, d = 0.22.$
 556 There were no significant correlations between average post-drink alcohol urge scores and average
 557 alcohol-related AB ($r = .13, p = .229$) or between average post-drink snack urge scores and average
 558 food-related AB scores ($r = -.07, p = .558$).



566 Figure 4. Boxplot displaying mean bias scores split by AB task and drink
 567 condition. Note: * $p = .025.$ For food attentional bias, positive scores indicate
 568 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.

569 3.2.3 Caloric Intake (Figure 5)

570 There was a greater number of calories consumed from the taste test in the alcohol condition
571 compared with the placebo condition $t(83) = 4.67, p < .001, d = 0.36$. Similarly, there was greater
572 total caloric intake in the alcohol condition compared with the placebo condition $t(83) = 15.11, p <$
573 $.001, d = 1.17$. The moderation analysis revealed that DEBQ restraint scores did not moderate the
574 effect of drink type on food intake $b = -15.03 [-57.85, 27.80], SE = 21.53, t(82) = 0.70, p = .487$.

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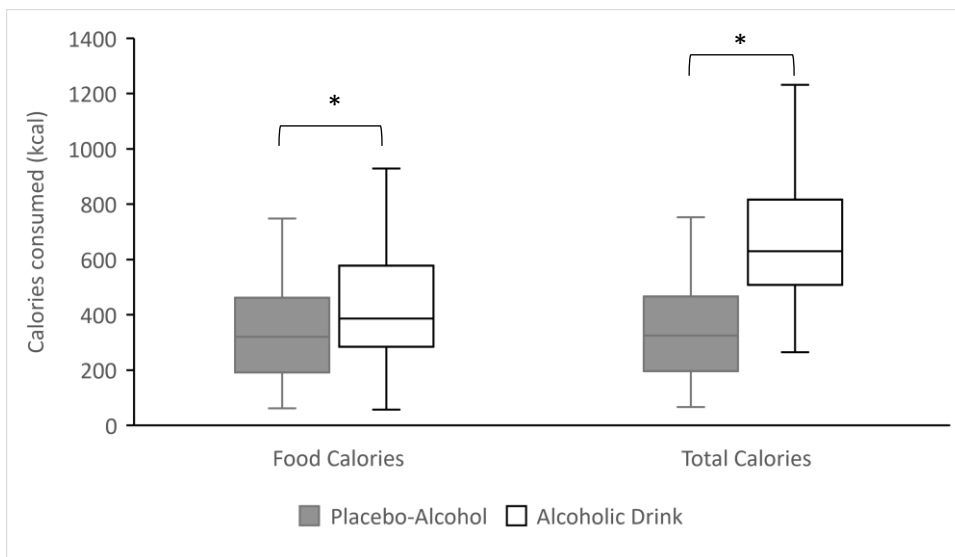
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Figure 5. Boxplot displaying number of calories consumed during the *ad libitum* taste test (food calories) and combined with calories consumed from the test drink (total calories), split by condition Note: * $p < .001$. Dots indicate outliers

592 3.2.4 Appetite Ratings (Figure 6a)

593 There was a significant main effect of drink on appetite ratings $F(1, 83) = 5.67, p = .019, \eta_p^2$
594 $= .06$, with consumption of the alcoholic drink producing greater appetite ratings. There was also a
595 significant main effect of time $F(1.79, 148.80) = 45.50, p < .001, \eta_p^2 = .35$. Pairwise comparisons
596 revealed that baseline appetite ratings were significantly lower than post-drink ratings ($p < .001$; mean
597 difference = 23.67; 95% CI [-32.04, -15.31]) but were significantly greater than post-taste test ratings

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

601 3.2.5 Snack Urge Ratings (Figure 6b)

602 There was a main effect of drink $F(1,83) = 10.54, p = .002, \eta_p^2 = .11$, with those in the alcohol
603 condition reporting greater snack urge. There was also a significant main effect of time $F(1.46,$
604 $121.54) = 13.13, p < .001, \eta_p^2 = .14$, and a significant drink by time interaction $F(1.85, 153.49) = 7.08,$
605 $p = .002, \eta_p^2 = .08$. See Figure 6b for comparisons of drink condition differences between time points.

606 3.2.6 Alcohol Urge Ratings (Figure 6c)

607 There was a significant main effect of drink $F(1,83) = 31.63, p < .001, \eta_p^2 = .28$, with
608 significantly greater alcohol urge ratings in the alcohol condition. There was also a significant main
609 effect of time $F(1.82, 32.79) = 30.85, p < .001, \eta_p^2 = .27$ and a significant drink by time interaction
610 $F(2,166) = 21.00, p < .001, \eta_p^2 = .20$. See Figure 6c for comparisons of drink condition differences
611 between time points.

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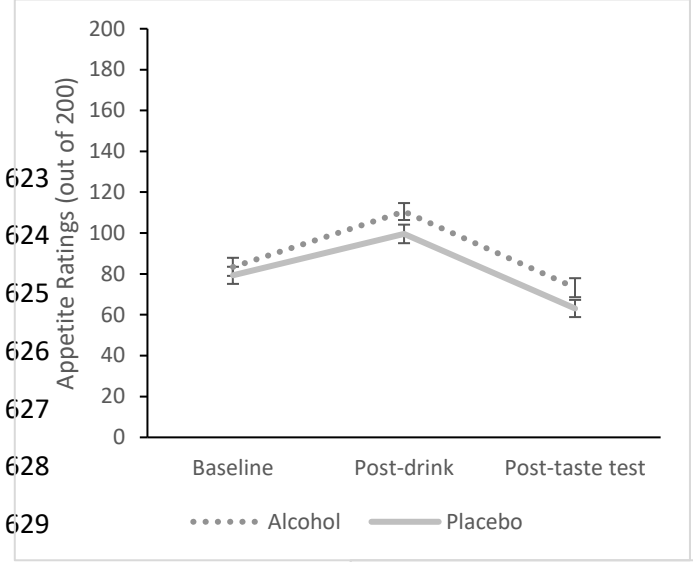
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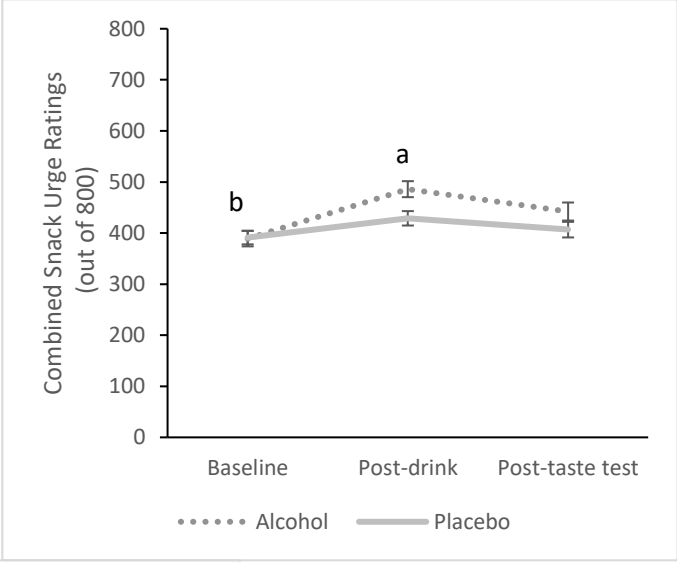
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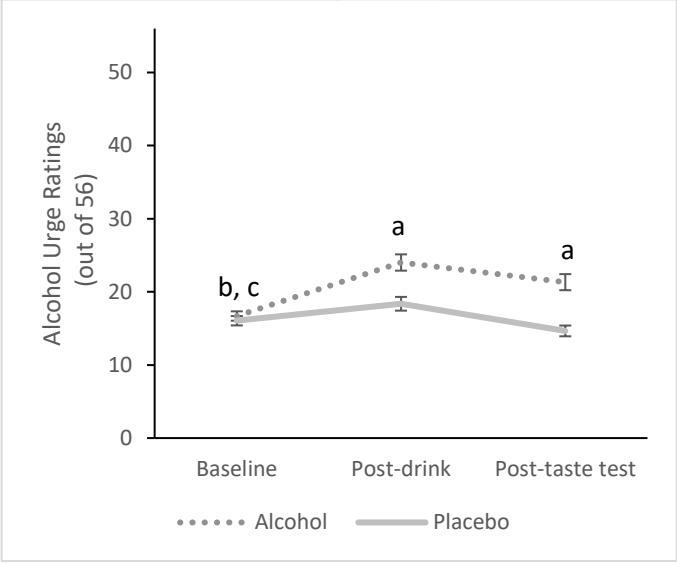


Figure 6 Appetite (6a), Snack urge (6b) and Alcohol urge ratings (6c) split by condition and across each time point. (Mean ± SEM) Note: Letters refer to Bonferroni corrected pairwise comparisons which compare difference scores between drink conditions across each time point ($p < .05$): a = different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

654 The regression analysis was performed to test whether motor impulsivity, differences in food-
655 related AB between conditions, and the interaction between them, predicted change in food intake
656 across the two conditions. The regression model predicted 13.9% of variance in change in food intake,
657 adjusted $R^2 = .14$, $F(6, 77) = 2.08$, $p = .065$. Dietary restraint ($\beta = -.06$, $p = .635$), BMI ($\beta = .31$, $p =$
658 $.014$) and food-related AB in the placebo condition ($\beta = -.20$, $p = .168$) predicted 9.6% of variance.
659 After controlling for these variables, step 2 predicted 4.3% of variance, with no significant predictor
660 variables: change in food-related AB ($\beta = -.14$, $p = .314$); motor impulsivity ($\beta = -.18$, $p = .101$);
661 change in food-related AB x motor impulsivity ($\beta < .01$, $p = .992$).

662 **4. General Discussion**

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

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

691 Study 2 failed to show an alcohol-induced increase in alcohol-related AB. Although
692 unexpected, this finding is in line with previous studies which have shown that alcohol consumption
693 fails to enhance AB towards alcohol cues at doses of 0.6 g/kg (Duka & Townshend, 2004) or 0.65
694 g/kg (Monem & Fillmore, 2019), but does increase self-reported urge to drink (e.g. Rose & Duka,
695 2006 – 0.6 g/kg). Overall, this suggests that alcohol consumption increases appetitive motivation for
696 alcohol, but that different assessment procedures may be focusing on different aspects of motivation
697 and/or value towards certain stimuli.

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

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

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

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

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

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

765

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