The effects of placebo and moderate dose alcohol on attentional bias, inhibitory control and subjective craving

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Abstract

Aims Previous research indicates that acute alcohol intoxication and placebo can inhibit people's control over consumption behaviour and heighten attentional bias (AB) towards alcohol-related stimuli, and craving. We designed a study to disentangle anticipated from pharmacological effects of alcohol in order to gain a clearer view of their relative contributions to alcohol consumption. *Methods:* In a within-participants design (moderate alcohol dose, placebo and control), and over a minimum 2-week period, participants completed a battery of questionnaires and cognitive tasks, followed by a bogus taste task to measure *ad libitum* consumption. *Results:* Both alcohol pre-load and placebo resulted in cognitive and psychological changes, including impaired inhibitory control, heightened AB and craving. However, *ad libitum* consumption only increased following alcohol and not placebo. Furthermore, inhibitory control impairments did not mediate the relationship between initial intoxication and *ad libitum* consumption, and findings indicate that increases in craving may mediate this association. *Conclusions:* Psychological processes such as craving may be more important in driving consummatory behaviour, relative to transient changes in cognitive processes such as inhibitory control.

Short summary

Alcohol pre-load and placebo resulted in impaired inhibitory control, elevated attentional bias and craving compared to placebo. Greater changes in inhibitory control and craving were observed following alcohol pre-load, but only placebo heighten attentional bias. Alcohol-related changes in craving appear to be the strongest driving factor in subsequent alcohol consumption.

Introduction

By now it has become apparent that drinking alcohol is both a driver and a consequence of a range of cognitive processes that are implicated in the consumption of alcohol. These include people's ability to exert control over their behaviours (see de Ridder et al., 2012), to attend to stimuli (see Field et al., 2016), as well as their desire to consume the substance in question (e.g.,Ostafin & Palfai, 2006). Sometimes, however, the mere anticipated effects of alcohol are sufficient to impact people's cognitions in a manner akin to that observed when alcohol is consumed (e.g., Christiansen et al., 2016). This presents a challenge to researchers seeking to disentangle the relative contribution of pharmacological and anticipated effects of alcohol since the belief that alcohol has been consumed alone can be sufficient to induce expectation effects. The implementation of placebo-controlled research has helped address this, however, a limitation of this body of work has been that most studies do not include a pure control condition, which diminishes the extent to which anticipatory effects can be ascertained.

The relationship between alcohol consumption, executive control and cognitive mediators is likely to be far from straightforward because alcohol affects a wide array of cognitive and psychological processes in distinct ways at different stages of (anticipated) consumption. This complexity is reflected in the literature. With regards to attentional bias (AB), for example, some studies find heightened AB following initial intoxication and that these effects are dose dependent (e.g., Duka & Townshend, 2004), while other research indicates that these changes are dependent primarily upon individuals' drinking experiences (e.g., Fernie et al., 2012). The picture for inhibitory control is similar. Some research suggests that impairments in inhibitory control are predictive of continued alcohol consumption

following initial intoxication (e.g., Weafer & Fillmore, 2008), while others have failed to demonstrate this (e.g., Christiansen et al., 2013), or suggest that subjective craving is a stronger predictor of alcohol involvement (Rose & Grunsell, 2008).

These discrepancies in this literature may, to a greater or lesser degree, be related to methodological differences between studies. For example, Leeman and colleagues (2009) found that craving following placebo, but not alcohol, correlated with *ad libitum* consumption. However, the absence of a pure control, in this instance, makes it difficult to ascertain whether placebo has induced any change in craving or whether those changes, in turn, predict consumption. Accordingly, research has begun to include a non-alcohol control condition to explore the relative contributions of the anticipated and pharmacological effects of alcohol, and the resultant changes to alcohol consumption behaviours.

However, to date relatively few studies have utilised this more stringently controlled design. Doing so, Christiansen and colleagues (2016) found both impaired inhibitory control and increases in subjective craving following placebo, relative to non-alcohol control. This study utilised a moderate dose consumption condition (.65g/kg), alongside placebo and control, revealing that alcohol impaired executive functioning compared to both placebo and control. Christiansen and colleagues (2013) also found increases in craving and automatic approach tendencies following placebo, however, these did not translate into increases in *ad libitum* consumption, which was only evident following an alcohol dose. Their work is important in advancing understanding regarding the influence of anticipation on alcohol-related cognitions and consumption behaviours in the absence of pharmacological driven effects.

Indeed, a pure control condition avoids any suggestion that alcohol has been ingested and, in contrast to traditional placebo conditions, alcohol-related cues (both visual and olfactory) are consequently removed - which is potentially important in light of early suggestions that alcohol-related smells (Monk et al., 2016) or visual stimuli (Qureshi et al., 2017) may reduce inhibitory control. Early research in this field therefore appears to suggest that the anticipated effects of alcohol may result in changes in cognition but not necessarily alcohol consumption (Christiansen et al., 2013), although more research is needed to establish the validity of these early findings. Specifically, although AB is implicated as an important contributory factor shaping drinking motivations and consumption behaviour (see Fadardi & Cox, 2008; Field & Cox, 2008; Field et al., 2016), it is necessary for research to tease apart pharmacological from anticipatory effects of alcohol on such cognitive biases.

Theoretically, alcohol-related attentional processing can be explained using two theoretical approaches: Alcohol myopia theory (AMM) posits that there is a narrowing of attention following intoxication, with attention being directed towards alcohol-related cues (Steele & Josephs, 1990). On the other hand, observed dose dependent reductions in AB (e.g., Duka & Townshend, 2004; Weafer & Fillmore, 2013) point to the possibility of the *Satiety Hypothesis.* Specifically, acute intoxication satiates ones appetitive for alcohol, reduces the salience of alcohol-related stimuli, thus reduced AB. The introduction of placebo and control conditions alongside alcohol pre-loads may therefore represent a means of testing the utility of these contrasting theoretical predictions regarding alcohol's effects on cognitive processes and subsequent consumption behaviours. As such, myopia theory would predict increased

attentional allocation for alcohol related cues as a consequence of intoxication, while the *Satiety Hypothesis* suggests a reduction in AB for alcohol-related stimuli.

The present study aims to examine the effects of alcohol on inhibitory control, attentional bias, craving, and how these processes impact further consumption. Specifically, by introducing a control beverage condition alongside more commonly used alcohol and placebo pre-loads, the present study uses a within-participant design to disentangle anticipated from the pharmacological effects of alcohol in order to gain a clearer view of their relative contributions. It was hypothesised that both alcohol pre-load and placebo consumption would impair inhibitory control relative to the control beverages, and that alcohol pre-load would result in greater impairments compared to placebo. Alcohol pre-load was expected to lead to increased craving and *ad libitum* alcohol consumption to a greater extent than placebo, with craving increases and inhibitory control impairments hypothesised to mediate the relationship between initial intoxication and *ad libitum* consumption. Finally, both alcohol pre-load and placebo were expected to result in heightened AB and, in turn, *ad libitum* consumption.

Method

Participants

Participants (n = 30**,** 17 female) aged between 18 and 27 years (M = 20.23, SD = 1.96) were recruited from a UK university. Participants were invited to take part if they self-reported regular consumption of alcohol and consumed over the recommended guidelines of 14 units per week, spoke fluent English and were aged between 18 and 49 years. Exclusion was restricted to those who had suffered from an alcohol dependence disorder or had sought help

regarding their consumption previously. Participants were either awarded course credit or £20 as reimbursement for their time. This study was sanctioned ethically by the Faculty Research Ethics Committee.

Design

A counterbalanced within-participants design was implemented, with all participants completing measures of inhibitory control (Stop-Signal task), attentional bias (Alcohol Dot Probe task) and *ad libitum* alcohol consumption (bogus taste test) during each study session in three different alcohol administration condition (alcohol pre-load, a placebo and a control session). Sessions were a minimum of 48 hours apart.

Materials

Questionnaires

Time Line Followback (TLFB: Sobell & Sobell, 1990) . Participants retrospectively self-report their alcohol consumption (in units) for the previous 14 days. Requiring participants to provide the number of units of alcohol consumed each day.

The Alcohol Use Disorders Identification Test (AUDIT: Saunders et al., 1993). The AUDIT consists of 10 items regarding alcohol consumption and its consequences. Scores range from 0-40, with scores ≥ 8 representative alcohol consumption of a hazardous level. The AUDIT has previously been validated in university student populations (e.g., Kokotailo et al., 2004) and is shown to be reliable in the current sample ($\alpha = .71$).

Barrett Impulsivity Scale (BIS-11: Patton et al., 1995). The BIS is a multidimensional scale, consisting of three subscales; attentional, motor and non-planning impulsiveness. BIS-11 includes thirty fixed response items (e.g., I plan tasks carefully), each on a 4 point scale

(rarely/never – almost always/always); higher scores are indicative of increased impulsivity. The scale was found to be reliable in the current sample (α = .87).

Alcohol Urge Questionnaire (AUQ: Bohn et al., 1995). The AUQ is a three-dimensional measure of subjective alcohol craving; desire for alcohol, positive expectations of alcohol consumption and ones' inability to avoid consuming alcohol that is available. Eight items (e.g., It would be difficult to turn down a drink at this minute) on scored on a seven-point scale ranging from 'strongly disagree' to 'strongly agree'. The AUQ produces a single factor for alcohol urges and shows high internal consistency within the present sample (α = .83).

Alcohol Outcome Expectancies Scale (AOES: Leigh & Stacy, 1993). The AOES assess both positive and negative outcome expectancies on a 6-point Likert scale (1= no chance of happening and 6 = certain to happen), consisting of 34 items (e.g., WHEN I DRINK ALCOHOL 'I feel guilty'). The questionnaire consists of four positive subscales (social facilitation, fun, sex and tension reduction) and four negative subscales (social, emotional, physical and cognitive/performance); both the positive and negative subscales are shown to be reliable measures with Cronbach's of .90 and .86 respectively, comparable previous analyses indicating alpha values of .94 and .88 in turn (see Leigh & Stacy, 1993).

Behavioural measures

Stop-signal task (SST: Verbruggen et al., 2008). The Stop Signal task consists of two concurrent tasks: A Go task (75% of trials), which is a choice reaction task where participants categorise arrows on the screen based on their orientation (left or right) and a stop task (25% of trials) where an auditory tone (the stop signal) indicates that participants should inhibit their response to the go signal. Participants are required to respond as quickly and accurately

as possible to the stimuli with a predetermined corresponding key. Upon hearing the auditory tone (the stop signal) participants are required to inhibit their response. After 2000ms the trial timed out.

On the stop trials, tones are delivered at fixed delays (known as Stop-signal delays or SSD) of between 50ms and 500ms following the presentation of the go stimulus. The stop signal task used these SSDs dynamically, based on participant performance. The *one-up one-down tracking procedure* (Logan et al., 1997) was implemented, which adjusts the SSDs after each trial. After successful inhibition trials, the SSD increases by 50ms, handicapping the stop signal process on the next stop signal trial. Unsuccessful inhibition trials result in the SSD decreasing by 50ms. In accordance with the 'horse race' model, the degree of difficulty in inhibiting responding increases as the delay between the go stimulus and the stop signal increases (Logan et al., 1984). Providing an outcome variable of stop-signal reaction time (SSRT). SSRT is calculated by extracting the percentage errors (failure to inhibit response on stop trials) at each of the SSDs (50 – 500ms, at 50ms intervals), then calculating a SSRT value for each SSD based on the reaction time (RT) distribution. Overall SSRT score was calculated by averaging the SSRT values for each of the SSD's. Impaired response inhibition is demonstrated through longer SSRT values; SSRT represents an estimate of the time required to stop initiated Go response (see Band et al., 2003). Participants received 3 experimental blocks of 64 trials, allowing for a short break between each block. Internal reliability was assessed between each of the 3 experimental blocks for each condition, alcohol pre-load (α = .79), placebo (α = .83) and control (α = .83), all were acceptable.

Alcohol visual dot probe task (Schoenmakers et al., 2008). The alcohol visual dot probe task was used concurrently with the Eye-link 1000 eye-tracker to measure attentional-bias. The pictorial stimuli consisted of ten alcohol-related pictures (as used by Christiansen et al., 2013) were matched with ten neutral pictures (e.g., pens), based on brightness, complexity and valence. Pictures were presented in landscape (125mm wide x 100mm high). Each trial was initiated by the presentation of a black fixation cross in the centre of the computer screen for 1000ms. Two pictures were then presented side-by-side 60mm apart, in alcohol-neutral pairs for 2000ms. The probe (a black 'X') is then presented on either the right or left side of the screen until the participant responds with the corresponding key; 'V' if the probe appears on the left or 'N' if the probe appears on the right, or until the trial timed out at 6000ms. The alcohol and neutral pictures are presented an equal number of times on the right and left side of the screen in a random order. Probes replace alcohol pictures on 50% of the trails and neutral pictures on 50%. The task consisted of 40 trials, each image appeared on the both sides of the screen twice, once replaced with the probe and once not.

Dwell time was calculated by summing the time (ms) participants spent focusing on both the alcohol and neutral stimuli for each trail, with a subsequent mean calculated. Prior to probe RTs being calculated, trail with RTs <100ms and 3 standard deviations above the participants mean were removed. A mean RT for alcohol and neutral probes was then calculated. Reliability analysis of the current sample revealed that both the probe reaction time (α = .59) and dwell time (α = .61) indices to be unreliable, comparable with other such findings (e.g., Field & Christiansen, 2012).

Alcohol Administration

Alcohol pre-load/placebo

The administration of an alcohol pre-load is used to assess the acute pharmacological effects of alcohol. As per Rose and Grunsell (2008), participants were administered a volume of alcohol based on their weight and gender; 0.5g/kg for females and 0.6g/kg for males. Alcohol in the form of vodka was mixed with fresh orange juice and tonic water in equal parts and divided into three glasses. The placebo consists of equal parts fresh orange juice and tonic water, the mixture and the rim of the glass is lightly sprayed with vodka (<2ml). The control drink consisted of orange juice, and the participants are informed the drink contains no alcohol. The participants were required to consume a strong mint to mask the taste of the alcohol (see Hopthrow et al., 2007) and then given 10 minutes to consume the total volume of the presented mixtures, followed by 20 minutes rest period prior to completing any other experimental tasks.

Ad libitum consumption

Ad libitum consumption is a method of measuring immediate alcohol consumption, indicative of motivation to drink, following an experimental manipulation (e.g., the administration of an alcohol pre-load). The bogus taste test is a means of assessing *ad libitum* alcohol consumption while reducing participant demand characteristics, using deception (Jones et al., 2016a). Participants were presented with three different beers (330ml of each) and asked to rate the taste of each beer on ten dimensions (e.g., pleasant and light; see Jones et al., 2011). For rating purposes, participants were informed that they may drink as little or as much as they need. The remaining volume was then measured and subtracted from the initial volume to indicate how much the participant consumed.

Procedure

All study sessions took place in the laboratory between 12 and 6pm. Participant were told

they should refrain from consuming alcohol for a minimum of 12 hours and eating for 3 hours prior to participation; all participants provided a breath alcohol level of 0.0mg/l (Lion Alcolmeter 400, Lion Laboratories, Vale of Glamorgan, United Kingdom) before commencing each study session. During the first session, participants completed a battery of questionnaires including demographic information, the TLFB and AUDIT. The within-participant order of conditions was allocated at random and counterbalanced; dependent on condition participants were either presented with an alcohol pre-load, placebo or control drink. Participants had 10 minutes to consume the drink, followed by a 20 minute absorption period. Two further questionnaires were completed, the AUQ and AOES. Participants then completed a battery of cognitive tasks (alcohol dot probe task and SST), the order the alcohol dot probe task and saccade tasks were presented was counterbalanced. Finally, participants completed the bogus taste task to measure their *ad libitum* consumption. Each study session lasted approximately 1 hour and participants were fully debriefed following completion of the final study session.

Results

Sample characteristics

Table 1 shows the means and standard deviations for the sample characteristics. A one-way ANOVA was used to assess gender differences in the sample characteristics, including previous drinking involvement. No significant gender differences were observed (*p's* > .05).

Table 1: *Descriptive statistics of participant characteristics*

	M	*SD*
Age	20.23	1.96
TLFB	24.07	15.82

AUDIT	11.10	3.58
BIS	63.53	11.86

TLFB = Timeline Follow back; 14-day alcohol consumption in UK units. AUDIT = Alcohol Use Disorder Identification Test, scores >8 indicative of hazardous drinking. BIS = Barratt Impulsivity Scale.

Cognitive tasks

Inhibitory control

The effects of beverage condition on inhibitory control, indicated by SSRT, were analysed using a repeated measures ANOVA, $F(2, 58) = 44.04$, $p < .001$, $\eta_p^2 = .60$. Bonferroni corrected pairwise comparisons demonstrated that greater impairments in inhibitory control following acute alcohol, compared with placebo ($p < .001$), which, in turn, showed greater levels of impairment in comparison with control ($p < .001$). See Table 2 for means and standard deviations.

Table 2: *Means and standard deviations for stop-signal reaction time, subjective craving and ad libitum beer consumption for each condition; alcohol, placebo and control*

	Alcohol		Placebo		Control	
	Mean	SD	Mean	SD	Mean	SD
SSRT	275.95	47.34	241.88	37.11	220.42	28.34
Craving (AUQ)	29.14	9.42	19.82	7.71	15.00	4.93
Ad libitum (ml)	427.70	316.41	241.03	239.89	205.23	232.82

SSRT = Stop-Signal Reaction Time (ms) AUQ = Alcohol Urge Questionnaire

Attentional bias

A 3x2 repeated measures ANOVA was performed with beverage condition (acute alcohol pre-load, placebo and control) and cue type (alcohol versus neutral) as within participant variable to assess dwell time. There was a significant beverage by cue type interaction $F(2, 58) = 13.71$, $p < .001$, $\eta_p^2 = .32$. Bonferroni adjusted simple main effects analysis revealed

that for alcohol cues dwell time was significantly greater following placebo compared with alcohol preload ($p < .01$) and control ($p < .001$). However, there was no difference between alcohol pre-load and control ($p > .999$). As for neutral cues there was no significant differences in dwell time between any of the beverage conditions (all p's > .45). Furthermore, there was significant differences between alcohol and neutral cues following acute alcohol pre-load ($p < .001$), placebo ($p < .001$) and control ($p < .01$). This suggests there was AB for alcohol cues following each of the conditions, however, it would appear AB was heightened following placebo compared with alcohol pre-load and control. See table 3 for means and standard deviations.

A second 3x2 repeated measures ANOVA was performed with beverage condition (acute alcohol pre-load, placebo and control) and cue type (alcohol versus neutral) as within participant variable to assess probe response RT. The was no significant beverage by cue type interaction $F(2, 58) = 2.09$, $p = .13$, $\eta_p^2 = .07$. There was also no significant effect of cue type on probe response RT $F(1, 29) = .01$, $p = .92$, $\eta_p^2 = .00$. On the other hand, there was a significant main effect of beverage condition $F(2, 58) = 9.33$, $p < .001$, $\eta_p^2 = .24$, indicating that probe response RTs were significantly slower following the acute alcohol pre-load compared both placebo ($p < .02$) and control ($p = .001$). There was no significant difference between placebo and control ($p = .76$). These finding fail to validate the dwell time AB from the previous analysis. See table 3 for means and standard deviations.

An additional AB variable was computed by subtracting dwell times for neutral images from dwell times for alcohol images (Weafer & Fillmore, 2013). Bivariate correlations were used to assess the relationship between AB, subjective craving and *ad libitum* consumption, all were insignificant (p's > .05).

Table 3: *Means and standard deviations for Alcohol Dot Probe task dwell time and probe reaction time for each condition; alcohol, placebo and control*

	Alcohol		Placebo		Control	
	Mean	SD	Mean	SD	Mean	SD
Alcohol Dwell (ms)	420.93	88.52	508.97	96.06	409.48	73.48
Neutral Dwell (ms)	333.62	63.62	333.71	71.85	347.34	62.75
Alcohol RT (ms)	472.03	74.12	445.24	74.13	435.12	71.38
Neutral RT (ms)	480.32	71.30	441.46	73.48	432.20	67.19

Alcohol and neutral dwell refer to the dwell time in milliseconds for alcohol-related and neutral images. Alcohol and neutral RT refers to the response reaction time (RT) in milliseconds when the probe replaced an alcohol-related or neutral image respectively

Self-report measures

Subjective craving

A repeated measures ANOVA was used to analyse subjective craving, as measured by the AUQ, in acute alcohol, placebo and control conditions. Overall, there was significant of beverage condition on AUQ $F(2, 58) = 21.98$, $p < .001$, $\eta_p^2 = .43$, with Bonferroni pairwise comparisons revealing higher ratings of subjective craving following acute alcohol compared with placebo ($p < .01$) and control ($p < .001$). Subjective craving was also higher following placebo compared with control ($p = .029$). See Table 2 for means and standard deviations.

Outcome-expectancies

Two further repeated measures ANOVAs were used to investigate effects of acute alcohol and placebo on positive and negative outcome expectancies. Neither positive $F(2, 58) = .34$, $p = .71$, $\eta_p^2 = .01$ or negative $F(2, 58) = .45$, $p = .96$, $\eta_p^2 = .002$ outcome expectancies were

found to be affected by alcohol or placebo. Post hoc analyses were performed on each of the AOES subscales, revealing no significant outcomes ($p > .05$).

Ad libitum alcohol consumption

A final repeated measures ANOVA was undertaken to analyse the volume of beer (ml) consumed *ad libitum* following acute alcohol, placebo and control $F(2, 58) = 22.18$, $p < .001$, $\eta_p^2 = .43$. Bonferroni corrected pairwise comparisons revealed that significantly more beer was consumed following acute alcohol compared with placebo ($p < .001$) and control ($p < .001$). However, there was no significant difference in the volume of beer consumed following placebo in comparison with control ($p = .86$). See Table 2 for means and standard deviations.

Mediation analysis

Repeated measures mediation analyses were undertaken using the MEMORE macro with SPSS (Montoya & Hayes, 2017) to assess the mechanisms that may mediate the relationship between acute alcohol intoxication and subsequent *ad libitum* consumption. The first path-analytic model assessed the relationship between acute alcohol intoxication, inhibitory control (SSRT) and *ad libitum* consumption. There was an effect of alcohol intoxication on *ad libitum* consumption (c_1) $t(26) = 4.42$, $p < .001$ 95% CI [101.14, 276.93]. There was also a significant relationship between alcohol intoxication and inhibitory control (a_1) $t(26) = 7.70$, $p < .001$, 95% CI [39.72, 71.36], but the relationship between inhibitory control and *ad libitum* consumption (b_1) was not significant $t(26) = .74$, $p = .47$, 95% CI [-4.12, 1.94]. The relationship between acute alcohol intoxication and *ad libitum* consumption remained significant (c'_1) $t(26) = 2.69$, $p = .01$, 95% CI [58.36, 440.62], suggesting that impairments in inhibitory control do not mediate subsequent alcohol consumption. See pathway 1 in Figure

1.

A further repeated measures mediation analysis was undertaken using subjective craving a mediator. Results showed a significant relationship between acute alcohol intoxication and *ad libitum* consumption (c_2) $t(28) = 4.97$, $p < .001$, 95% CI [133.52, 320.62]. A significant relationship between alcohol intoxication and subjective craving was also found, (a_2) $t(28) = 6.83$, $p < .001$, 95% CI [9.46, 17.57]. The path between subjective craving and consumption (b_2) however, only approached significance $t(26) = 1.89$, $p = .07$, 95% CI [-11.84, 7.98], while the relationship between alcohol intoxication and *ad libitum* consumption (c'_2) remained significant $t(26) = 3.26$, $p < .01$, 95% CI [93.36, 412.99]. See pathway 2 in Figure 1.

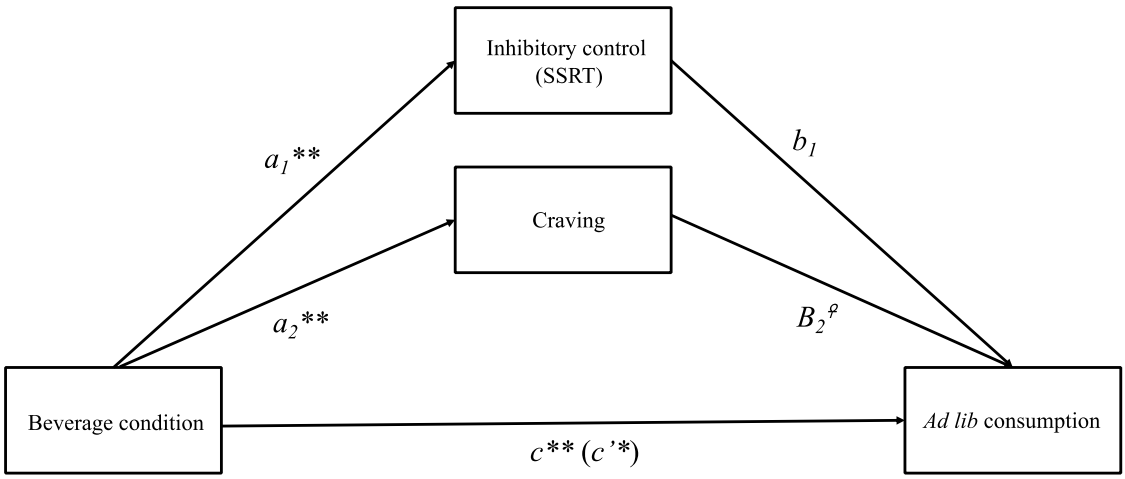

Figure 1: Path-analytic mediation model. Pathway 1 assesses inhibitory control impairments as a mediator between alcohol beverage condition and *ad libitum* consumption. Pathway 2 examines craving as the mediator between beverage condition and subsequent consumption. **$p < .001$, *$p < .01$ † $p < .07$

The aim of the present study was to examine the effects of alcohol on inhibitory control, attentional bias and subjective craving to elucidate how these processes impact further consumption. By introducing a control beverage condition alongside more commonly used alcohol and placebo pre-loads, the present study used a within-participant design to disentangle anticipated from the pharmacological effects of alcohol to ascertain the way in

which intoxication contributes to both alcohol-related cognitions and further alcohol consumption. As predicted, and in line with previous research, results suggest that, in comparison to control, both acute alcohol (e.g., Caswell et al., 2013; Weafer & Fillmore, 2008) and placebo (Christiansen et al., 2016) impaired inhibitory control, with the greatest impairments being observed in the alcohol pre-load condition. With regard to subjective craving, the current study also found increases in craving following both alcohol and placebo, and these were once again higher following alcohol compared to placebo, consistent with previous research (e.g., Christiansen et al., 2013; Rose & Grunnsell, 2008). However, impairments in inhibitory control did not mediate subsequent consumption, while findings may point to subjective craving partially mediating this relationship.

While no heightened AB was found following control, AB was found to increase in both alcohol and placebo admin conditions and the strength of this effect was observed to be higher after placebo relative to alcohol consumption. Whilst not in line with predictions, this is interesting as it may suggest that anticipated effects play a greater role in driving AB than pharmacological effects. There are two potential explanations for the current findings, alcohol-related oculomotor impairments or the "alcohol satiety hypothesis". First, a number of studies have demonstrated that alcohol intoxication can reduce saccade velocity and impair ocular location and fixation of specified targets (e.g., Abroms et al., 2006; Roche & King, 2010). These oculomotor impairments are however more commonly observed at doses higher than those of the current study. The second and more plausible explanation is that of the 'alcohol satiety hypothesis'. This posits that the transient reductions in AB following alcohol intoxication detected in the pre-load condition, are the result of alcohol's rewarding effects satiating one's motivations to consume and by extension attentional allocation to alcohol-related stimuli (i.e., Duka & Townshend, 2004; Weafer & Fillmore, 2013). Further support comes from recent findings that indicate that AB for alcohol-related stimuli but not food stimuli is reduced following acute alcohol intoxication (Monem & Fillmore, 2019). On the

other hand, the placebo condition would be expected to elicit anticipatory effects (see Marlatt, Demming, & Reid, 1973) without the associated dampening effects of satiation on AB, as might be expected as a result of intoxication. This may potentially explain why AB appeared to be stronger for those in the placebo relative to the alcohol condition as, from this perspective, the pharmacological effects of alcohol may have impacted AB adversely. While speculative and requiring further exploration, such suggestions appear consistent with findings from Weafer and Fillmore (2013), who identified dose dependent decreases in AB in heavy drinkers, and also in line with research from the field of subjective craving (Rose et al., 2013).

It has previously been proposed that transient inhibitory control impairments may mediate the association between initial intoxication and continued consumption (see Field et al., 2010; Jones et al., 2013). However, while current findings evidence increased *ad libitum* consumption following alcohol pre-load, there were no apparent differences between placebo and control conditions. This may suggest that pharmacological, but not anticipatory effects, may drive *ad libitum* consumption (Christiansen et al., 2013). Furthermore, impairments in inhibitory control did not appear to mediate the association between initial intoxication and *ad libitum* consumption, although increased craving appeared to be implicated in heightened *ad libitum* consumption. It may therefore be postulated that people's desires rather than transient changes in cognitive processes (e.g., inhibitory control) may be more important in driving consummatory behaviour. Such an assertion would seem to be in line with research from Rose and Grunsell (2008), which indicates that alcohol-induced increases in craving may be a better predictor of binge drinking, compared with inhibitory control. As such, while inhibitory control is potentially implicated in the maintenance of consumption, our findings suggest that these impairments may not be the central mechanism driving alcohol

consumption.

By separating the pharmacological from anticipated effects using pure control, placebo and acute alcohol, the current study contributes to the emerging literature which seeks to study the effects of cognitive and psychological changes in the maintenance of alcohol consumption. Nevertheless, a number of limitations need to be taken into consideration when interpreting the findings of the current study. First, while the within-participants design allowed us to control for conceivable individual differences effects, the current sample size could be limiting the ability to detect mediatory effects. Post hoc sensitivity analysis was conducted, data revealed the current sample size (n = 30) yielded a minimum observed power of .49 on the indirect effects path (c'), below the .80 deemed adequate (Onwuegbuzie & Leech, 2004). The analysis revealed that a sample size greater than 60 would be required to meet the minimum .80 for all paths of the mediation model. An element of caution is therefore advised when interpreting the results of the mediation models. Although, it is also worth considering that an increased sample and associated power may allow for the detection of the partial mediation speculated upon. Second, while a review of taste tasks indicated no overall effect of time of day or day of the week on volume consumed (Jones et al., 2016), it is worth noting that data collection took place outside time associated with consumption. Finally, the current research used a student sample. University students are immersed in a heavy drinking culture (Borsari & Carey, 2001; Karam et al., 2007; Knight et al., 2002) and it is therefore possible that findings may not generalize to populations with different drinking experiences (see Albery et al., 2015).

In conclusion, the current study sought to examine the effects of alcohol-related cognitive and psychological changes in the maintenance of alcohol consumption, separating the pharmacological from anticipated effect using pure control, placebo and acute alcohol.

Findings suggest that AB may be more susceptible to anticipated relative to pharmacological effects, as higher levels of AB were found following placebo relative to alcohol and control. This could potentially be explained by the devaluation of alcohol-related stimuli as a consequence of satiation following alcohol pre-load. Furthermore, both alcohol and placebo conditions resulted in impaired inhibitory control and heightened craving, with the greatest changes being observed following alcohol intoxication. However, only the pharmacological effects (evidenced in the alcohol condition) appeared to be associated with increased consumption. Here, impaired inhibitory control was not found to mediate the association between initial alcohol intoxication and continued consumption, while, on the other hand, results may suggest that craving could partially mediate this relationship. In sum, results suggest that psychological processes such as craving may be more important in driving consummatory behaviour, relative to potentially more transient changes in cognitive processes.

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