Abstract

Background:

The pharmacological effects of alcohol on executive function, craving and subsequent alcohol-seeking have been well-documented. Yet, insufficient methodological controls within existing alcohol administration paradigms have meant that the relative importance of alcohol's pharmacological and anticipatory effects remain in need of further elucidation.

Aims:

To disentangle alcohol's pharmacological from its anticipatory effects on alcohol-related cognitions and subsequent consumption.

Methods:

Inhibitory control, attentional bias and craving were assessed pre- and post-consumption in 100 participants who were randomly allocated to one of four beverage conditions in a two by two design: (i) alcohol aware (alcohol with participant knowledge [pharmacological/anticipation effects]) (ii) alcohol blind (alcohol without participant knowledge; in a novel grain alcohol masking condition [pharmacological/no anticipation effects]) (iii) placebo (no alcohol but participants were deceived [anticipation/non-pharmacological effects]) and (iv) pure control (no alcohol with participant knowledge [no anticipation/non-pharmacological effects]).

Results:

Findings suggest that the pharmacological effects of alcohol result in greater inhibitory control impairments compared with anticipated effects. Anticipatory but not the pharmacological effects of alcohol were found to increase attentional bias. Both pharmacology and anticipation resulted in increased craving, though higher levels of craving were observed due to alcohol's pharmacology. Furthermore, alcohol pharmacology resulted in heightened *ad libitum* consumption, however, anticipation did not. Changes in craving

partially mediated the relationship between initial intoxication and subsequent drinking, while inhibitory control impairments did not.

Conclusions:

Successive alcohol consumption appears driven primarily by the pharmacological effects of alcohol which are exerted via changes in craving.

Introduction

Research has established that both the pharmacological effects of intoxication and the psychologically anticipated effects of alcohol consumption impact alcohol behaviours in important ways. For example, low doses of alcohol have been found to increase subsequent consumption (Fernie et al., 2012), possibly by impairing inhibitory control mechanisms (Weafer & Fillmore, 2016) or by eliciting craving (McNeill et al., 2021). It is also established that psychosocially negotiated expectations about one's own and others' alcohol behaviours (McAndrew & Edgerton, 1969) and related cognitions (Marlatt et al., 1973) can exert significant influences on alcohol behaviours (Christiansen et al., 2017). To date, however, it has not been possible to fully disentangle alcohol's pharmacological and anticipated effects. This is because existing paradigms necessitate that participants either experience anticipation (in placebo conditions where non-alcoholic drinks are made to appear alcoholic) or pharmacological effects of alcohol combined with anticipation (in conditions where participants are given alcohol to drink). In other words, to date it has not been possible to assess the pharmacological effects of alcohol without also, to a greater or lesser degree, eliciting anticipatory effects. In order to achieve intoxication without anticipation, the current study addresses this limitation by introducing a methodology where beverages are also administered in a novel experimental condition in which participants are deceived about their alcoholic contents.

Existing research on how alcohol pharmacology impacts cognitive processing and consumption behaviour has primarily used a placebo alcohol condition as the basis for comparing the relative effects of intoxication. Here effects are assessed between participants who knowingly receive alcohol and those in the placebo group, who falsely believe their drinks contain alcohol. This approach has illuminated how alcohol affects various cognitive

processes including impairing inhibitory control (e.g., Caswell et al., 2013; Weafer & Fillmore, 2008), dose dependent effects on attentional bias (e.g., Duka & Townshend, 2004; Weafer & Fillmore, 2013), heightened craving (e.g., Rose & Grunsell, 2008) and increased risk taking (e.g., Rose et al., 2014).

The effects of alcohol pharmacology on cognitive processes have therefore been well documented, and the resulting transient changes have been found to be associated with subsequent alcohol consumption (Field et al., 2010). Specifically, consuming alcohol (sometimes referred to in the literature as alcohol pre-load, or 'priming' conditions), appears to be associated with increased alcohol seeking behaviours (including consumption; de Wit, 1996) and it has been suggested that these changes in behaviour are mediated by transient impairments in inhibitory control (Field et al., 2010). This theoretical view is informed by Weafer and Fillmore's (2008) work indicating that *ad libitum* consumption in a follow-up session correlated with alcohol induced inhibitory control impairments. While this research points to important relationship facets between inhibitory control changes and consumption, owing to its correlational nature, it was not designed to examine this relationship causally. Attempts to establish a causal link between inhibitory control and consumption by administering Transcranial Magnetic Stimulation to the right Dorsolateral Prefrontal Cortex (rDLPFC) have yielded mixed findings and highlight the possibility that other mechanisms may underpin this relationship (McNeill et al., 2018).

Alcohol-related attentional bias, identified as a further potential driver of consumption (Field & Cox, 2008), has also generated inconsistent findings. On the one hand, a bidirectional association between heightened attentional bias and alcohol-seeking behaviours has garnered support (ibid), and recent studies indicate that alcohol-related attentional bias is

most pronounced immediately prior to a drinking (Spanakis et al., 2019). However, the acute effects of alcohol on attentional bias also appear to be affected by peoples' previous alcohol involvement and acute intoxication. For instance, one study found that dose increases were associated with attentional bias decreases in heavy social drinkers but not among moderate social drinkers (Weafer & Fillmore, 2013). On the other hand, Fernie and colleagues (2012) found no effect of low doses of alcohol on attentional bias in heavy social drinkers, but an increase in light social drinkers. In both of these studies, changes in attentional bias were unrelated to *ad libitum* consumption. The existing literature therefore points to an intricate relationship between initial alcohol consumption and attentional bias, and further research to unpick the relative contributions of alcohol pharmacology and anticipation is required.

Theoretically it has been proposed that alcohol anticipation is driven by the associated reinforcing effects of its pharmacology (Marlatt et al., 1973) in that alcohol initiates a 'priming' effect, triggering alcohol seeking behaviours and eliciting cognitive changes which, to a greater or lesser extent, are comparable to those of alcohol itself (ibid). Complimenting cognitive approaches, McAndrews and Edgerton (1969) suggest theoretically that alcohol-associated environments and beliefs about normative alcohol behaviours can induce similar changes in alcohol seeking behaviour. Both cognitive and social research and theoretical contributions therefore converge to suggest that the mere suggestion of alcohol, whether that be smell of alcohol (e.g., Monk et al., 2016) or alcohol-related contexts (e.g., Field & Jones, 2017), can induce both cognitive and behavioural changes akin to actual alcohol consumption.

Overall, significant attempts have been made to understand the contributions of alcohol anticipation on inhibitory control and attentional bias, with the inclusion of pure

control conditions where participants are administered a beverage free of alcohol and informed of this (e.g., Christiansen et al., 2013), with resultant performance of these participants compared against those in placebo and alcohol aware conditions (i.e.. the pp knowingly consume alcohol prior to testing). These studies have found the effects of anticipation on inhibitory control and craving to be relatively consistent. Specifically, they indicate that placebo-alcohol produces inhibitory control impairments (which are indicative of expectancy effects), while these impairments are not comparable with those evident in intoxicated participants in the alcohol aware condition (e.g., Christiansen et al., 2016). Similarly, there is evidence to suggest that placebo alcohol also heightens craving (Christiansen et al., 2013; Christiansen et al., 2016), but that patterns of craving differ in alcohol aware and placebo conditions (Rose et al., 2013). However, the literature evidencing the relationship between anticipation and attentional bias is scant, with one study demonstrating a heightening of attentional bias following placebo in heavy drinkers (Weafer & Fillmore, 2013). Furthermore, findings supporting the assertion that anticipation provokes increases in seeking behaviour are inconsistent (e.g., Christensen et al., 2017; Christiansen et al., 2013). So, while the inclusion of pure control designs has illuminated the relative contribution of anticipation to alcohol-related cognitions, findings have remained mixed and study designs have not been able to fully extricate the impact of anticipation from that of pharmacology.

To this end, the current study sought to build on the growing body of literature in this domain, by administering an alcohol blind condition. Here, researchers covertly dispensed alcohol within provided beverages, while participants were informed they are in a control condition and that their drink is free from alcohol. By introducing this protocol alongside a traditional alcohol aware, placebo and pure control administration protocols, it aimed to tease

apart the relative influence of pharmacology and anticipation exert on alcohol-related cognitions and consumption. Specifically, a 2 x 2 between participants design was employed with independent variables of anticipation (anticipation vs no anticipation) and pharmacology (pharmacological vs non-pharmacological) to test the following hypotheses:

1. Both the anticipatory effects of alcohol (present in the placebo condition) and alcohol's pharmacology (present in the alcohol aware and alcohol blind conditions) will result in i) impaired inhibitory control and ii) heightened craving.
2. The pharmacological effects of alcohol (in the alcohol aware and alcohol blind conditions) will result in significantly greater i) impairments in inhibitory control and ii) increases in craving.
3. Alcohol-related attentional bias will be significantly more pronounced in alcohol anticipation (in the placebo condition) rather than pharmacology conditions (in the alcohol aware and alcohol blind conditions).
4. *Ad libitum* consumption will be increased by alcohol's pharmacological effects (in the alcohol aware and alcohol blind conditions).
5. These patterns of *ad libitum* consumption will be mediated by i) inhibitory control impairments and ii) heightened craving.

Method

Participants

One hundred (57 female) participants aged 18 to 49 (M = 21.18, SD = 4.73) who were fluent English speakers and whose weekly consumption regularly exceeded the recommended limit of 14 UK units were recruited. Participants were from the student and staff populations of a University in Northwest England. They completed a medical screen questionnaire and participants indicating that they had medical conditions or were taking medications known to

interact with alcohol were excluded from the study. Those with a personal or family history of Alcohol Use Disorder were also excluded. Participants were reimbursed for their time by way of course credit or £10. The study was given ethical scrutiny and approval by the Faculty of Arts and Sciences Research Ethics Committee, Edge Hill University.

Design

A randomised 2 x 2 between participants design was implemented, with independent variables of: anticipation (anticipation vs no anticipation) and pharmacology (pharmacological vs non-pharmacological). This was assessed using four beverage conditions: alcohol aware (pharmacology with anticipation), alcohol blind (pharmacology without anticipation), placebo (anticipation without pharmacology) and pure control (neither anticipation or pharmacology). Measures of inhibitory control, attentional bias and craving were taken both pre- and post-beverage administration, followed by a measure of *ad libitum* alcohol consumption. Dependent variables were computed by subtracting pre-beverage scores from post-beverage scores on each of the measures to produce a change score.

Materials

TimeLine 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. This was shown to be reliable in the current sample ($\alpha = .77$).

Barratt 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 four 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 ($\alpha = .81$).

Desire for Alcohol Questionnaire – brief form (DAQ; Love et al., 1998). The DAQ is a 14-item four-dimensional alcohol craving questionnaire. The factors include; positive and negative reinforcement, strong desires and intentions, and mild desires and intentions. The scale is scored on 1-7 Likert scale, with higher scores indicative of higher craving. Reliability analysis revealed the DAQ to be reliable in study 2 both pre ($\alpha = .78$) and post ($\alpha = .80$) beverage administration, both showing acceptable internal reliability.

Mood and Subjective Intoxication Scales. The scales consisted of 10 statements to which participants responded on a 100mm Visual Analogue Scale ranging from 'Not at all' to 'Extremely'. A total of six mood statements (e.g., 'I feel happy, I feel sad'), 3 each for positive and negative moods and 4 intoxication statements (e.g., 'I feel drunk, I feel dizzy').

Computerised cognitive tasks

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 times 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 uses 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). The task provides 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 both pre- and post-manipulation (pre $\alpha = .77$, post $\alpha = .71$) both indicating acceptable reliability.

Visual Probe task (Schoenmakers, Wiers & Field, 2008). The visual probe task was programmed in Experiment Builder and deployed in concurrence with the Eye-link 1000 eye-tracker (SR Research, Mississauga, ON, Canada) to assess attentional-bias. The task begins with the presentation of a fixation cross, signalling the beginning of each trial. Following this, manual submission of any key triggers the exhibition of images which are presented side-by-side 60mm apart, in alcohol/neutral pairs. Each trail had a duration of 2000ms and the task consisted of 40 trials in total. During each trail participants were instructed to allow their attention to drift over the images naturally, but they do not need to manually respond to the pictures. The pictorial stimuli consisted of ten alcohol-related pictures (as used by Christiansen et al., 2013) were matched with ten control pictures, based on brightness, complexity and valence. The alcohol-related pictures portrayed alcohol (e.g., bottle of beer, wine or spirits) or alcohol being consumed. The neutral pictures portrayed stationary (e.g., pens) or close-up action shots of stationary in use (e.g., a person licking an envelope). Pictures were each presented four times in pseudorandom order, twice on the right and twice on the left. Alcohol-related images (e.g., beer bottle) were always paired with stationary only images (e.g., pens), while images of alcohol being consumed were paired with action shots of stationary in use. Pictures were presented in landscape (125mm wide x 100mm high). 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 for alcohol and neutral stimuli calculated. The reliability of the Visual Probe task was shown to be fairly poor, both pre ($\alpha = .53$) and post ($\alpha = .36$), however, this is consistent with previous findings (e.g., Field & Christiansen, 2012).

Alcohol Administration

All drinks were divided into 3 equal glasses and the participants were given 10 minutes to consume the total volume of the presented mixtures, followed by a 20 minutes rest period prior to completing post-consumption experimental tasks.

Alcohol aware and placebo conditions

The administration of an alcohol here is to assess its acute pharmacological effects. As per Rose and Grunsell (2008), participants were administered a volume of alcohol based on their weight (.4g/kg). In the alcohol aware condition, alcohol in the form of vodka was mixed with fresh orange juice and tonic water in equal parts. It was made clear to participants the beverage contained alcohol. In the placebo condition, participants were administered equal parts fresh orange juice and tonic water, which has a natural bitterness to heighten the sense of alcohol's presence. To enhance the illusion of alcohol consumption, vodka (>2ml) was sprayed lightly on the rim of the glass and participants were told their drink contained alcohol. Overall, the placebo condition is designed to present a non-alcoholic drink as a drink containing alcohol using deception.

Alcohol blind condition

For the alcohol blind condition, grain ethanol (.4g/kg) was mixed with orange juice, calculated to match the dilution of the alcohol aware condition mixture. Grain ethanol was opted for over vodka as a significantly lower volume of liquid is required to meet the .4g/kg. This increased the possibility of masking the presence of alcohol. Ten millilitres of liquid sweetener (Canderel®) were added to the mixture to further mask the bitterness of the ethanol (informed by pilot testing) and participants were asked to consume a strong mint immediately prior to drinking as a further concealment. Unlike the other beverage conditions (alcohol aware, placebo), the alcohol blind beverage did not contain tonic water, due the

natural bitterness associated, thus, mitigating the use of sweetener. Participants were informed that they were in a control condition and that their beverages had been prepared to match the calorific content of drinks in the alcohol condition.

Pure control condition

For the pure control condition, participants were given fresh orange juice (tonic was not used as its bitterness may inappropriately suggest the presence of alcohol) of volume equivalent to other conditions and based on their body mass, and the participants were informed the drink contained no alcohol. They were also presented the drink in 3 equal measures and had 10 minutes to consume, followed by the same 20-minute rest period.

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 (Christiansen et al., 2015). 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

Each experimental session took place between 12-6pm in a laboratory. Participants were informed that they were required to refrain from drinking alcohol a minimum 12 hours prior and upon arriving at the laboratory a breathalyser reading was taken in order to ensure a reading of .00mg/l (Lion Alcolmeter 400, Lion Laboratories, Vale of Glamorgan, United

Kingdom). Participants were also asked to avoid eating 3 hours before the session. After providing informed consent, participants completed an initial questionnaire battery (TLFB, AUDIT, BIS, DAQ, mood and intoxication scales), followed by baseline measures of inhibitory control and attentional bias, in a counterbalanced order. Participants were then provided with a beverage, dependent on their randomly allocated beverage condition (n = 25 per condition). They were informed they had 10 minutes to drink the whole drink and they were then given a 20-minute rest period. Participants were then breathalysed for a second time, before repeating the DAQ, mood and subjective intoxication scales, measures of inhibitory control and attentional bias, all in a counterbalanced order. Finally, participants completed the bogus taste task to measure their *ad libitum* consumption. Experimental sessions took approximately 1 hour 40 minutes and all participants were fully debriefed following completion of the study.

Results

Sample Characteristics

A MANOVA was performed to assess any differences between conditions or genders in terms of sample characteristics (e.g., AUDIT, BIS). No significant differences were revealed between conditions *Pillai's Trace* = .20, $F(18, 279) = 1.30$, $p = .32$, $\eta_p^2 = .07$ or between genders *Pillai's Trace* = .09, $F(6, 93) = 1.61$, $p = .15$, $\eta_p^2 = .09$. A further ANOVA was performed to ensure that there were no difference between genders for breath alcohol level post beverage (only alcohol containing conditions assessed), $F(1, 48) = .20$, $p = .66$, $\eta_p^2 = .004$. Table 1 contains means and standard deviations for all baseline measures.

Table 1: *Means (and standard deviations) for sample characteristics, broken down by gender and beverage condition*

	Male (n = 43)	Female (n = 57)	Pure Control (n = 25)	Placebo (n = 25)	Alcohol Blind (n = 25)	Alcohol Aware (n = 25)	Total (n = 100)
AUDIT	12.44	10.35	10.36	9.44	12.40	12.80	11.25
	(4.66)	(4.56)	(4.88)	(3.63)	(4.12)	(5.17)	(4.64)
TLFB	38.21	25.81	25.08	26.42	32.74	40.34	31.15
	(26.72)	(22.64)	(21.93)	(21.22)	(19.67)	(33.66)	(25.12)
BIS Attentional	17.02	16.12	16.44	15.56	17.40	16.64	16.51
	(3.23)	(3.59)	(3.55)	(3.87)	(3.75)	(3.36)	(3.45)
BIS Motor	25.02	22.88	22.00	24.24	24.24	24.72	23.80
	(5.13)	(4.19)	(4.16)	(5.09)	(4.02)	(5.26)	(4.72)
BIS Non-planning	25.56	23.77	23.80	23.68	26.12	25.25	24.54
	(5.10)	(4.33)	(4.58)	(4.54)	(4.80)	(4.66)	(4.74)
BIS Total	66.92	62.30	61.08	62.88	67.24	65.96	64.29
	(10.59)	(8.58)	(9.48)	(10.89)	(8.46)	(9.22)	(9.72)

AUDIT = Alcohol Use Disorder Identification Test, TLFB = Timeline Followback a measure of UK units consumed over the past 14 days, BIS = Barrett Impulsivity Scale and Attentional, Motor and Non-planning are subscales of the BIS

A MANOVA was undertaken to assess if any differences existed between conditions for pre-beverage scores, no significant effect of beverage condition was observed *Pillai's Trace* = .09, $F(15, 282) = .56$, $p = .91$, $\eta_p^2 = .03$. Subsequently, pre-beverage scores were subtracted from the post-beverage score to produce change scores. Table 2 contains means and standard deviations for pre- and post-beverage, plus change scores for positive and negative mood, and each of the dependent variables (SSRT, AB and DAQ).

Table 2: *Mean and standard deviations for pre- and post-beverage dependent variable scores, and change scores*

	Pure Control (n = 25) Mean (SD)	Placebo (n = 25) Mean (SD)	Alcohol Blind (n = 25) Mean (SD)	Alcohol Aware (n = 25) Mean (SD)
Pre-positive	214.56 (45.89)	203.36 (37.08)	203.60 (42.37)	205.80 (42.58)
Post-positive	216.64 (49.79)	207.12 (32.47)	209.68 (41.53)	210.20 (42.88)
Positive change	2.08 (15.50)	3.76 (20.32)	6.08 (17.94)	4.40 (18.32)
Pre-negative	62.20 (35.49)	78.20 (28.07)	72.84 (30.50)	79.68 (38.00)
Post-negative	80.37 (40.72)	74.88 (23.83)	75.48 (32.24)	76.60 (36.06)
Negative change	18.16 (23.48)	-3.32 (13.28)	2.64 (18.75)	-3.08 (17.54)
Pre-SSRT	196.81 (40.36)	184.33 (41.13)	189.85 (64.63)	201.35 (65.64)
Post-SSRT	197.43 (35.83)	247.30 (39.52)	282.21 (55.57)	284.90 (60.07)
SSRT Change	.62 (38.79)	62.98 (52.83)	92.36 (59.69)	83.55 (60.06)
Pre-AB	120.06 (137.60)	125.11 (143.14)	124.98 (163.64)	166.28 (176.89)
Post-AB	145.87 (112.86)	294.05 (134.04)	142.97 (117.08)	145.54 (228.19)
AB Change	25.81 (115.38)	168.94 (155.80)	18.00 (232.14)	-20.72 (302.54)
Pre-DAQ	38.60 (10.07)	38.68 (7.08)	38.92 (7.10)	38.61 (6.70)
Post-DAQ	37.96 (11.44)	46.84 (10.13)	49.52 (11.34)	54.68 (8.84)
DAQ Change	.64 (5.79)	8.16 (9.04)	10.60 (9.20)	16.32 (6.85)

Positive/Negative = mood state, SSRT = Stop-Signal Reaction Time, AB = alcohol-related attentional bias, DAQ = Desires for Alcohol Questionnaire. Change scores were calculated by subtracting pre- from post-beverage scores.

Mood and subjective intoxication

Two 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVAs were employed to assess changes in mood controlling for AUDIT, one for positive and one for negative mood. For positive mood scores, there was no main effect of anticipation $F(1, 95) = .00$, $p > .999$, $\eta_p^2 = .00$ or pharmacology $F(1,95) = .41$, $p = .52$, $\eta_p^2 = .004$. Nor was there a significant interaction $F(1,95) = .22$, $p = .64$, $\eta_p^2 = .002$. There were, however, significant main effects observed for changes in negative mood. Specifically, scores were significantly lower in the anticipation conditions (i.e., placebo and alcohol aware) compared with the no anticipation conditions (i.e., pure control and alcohol blind) $F(1, 95) = 13.14$, $p < .001$, $\eta_p^2 = .12$, and lower in the pharmacological (i.e., alcohol aware and alcohol blind) compared with the non-pharmacological conditions (i.e., pure control and placebo) $F(1, 95) = 4.12$, $p = .045$, $\eta_p^2 = .04$. There was also a significant interaction between

anticipation and pharmacology $F(1, 95) = 4.33$, $p = .04$, $\eta_p^2 = .05$, with Bonferroni corrected pairwise comparisons indicating that the non-anticipatory and non-pharmacological condition (i.e., pure control) showed significantly higher negative mood scores compared with the non-anticipatory and non-pharmacological condition (alcohol blind) ($p = .004$, $d = .75$). See figure 1 for means and standard errors.

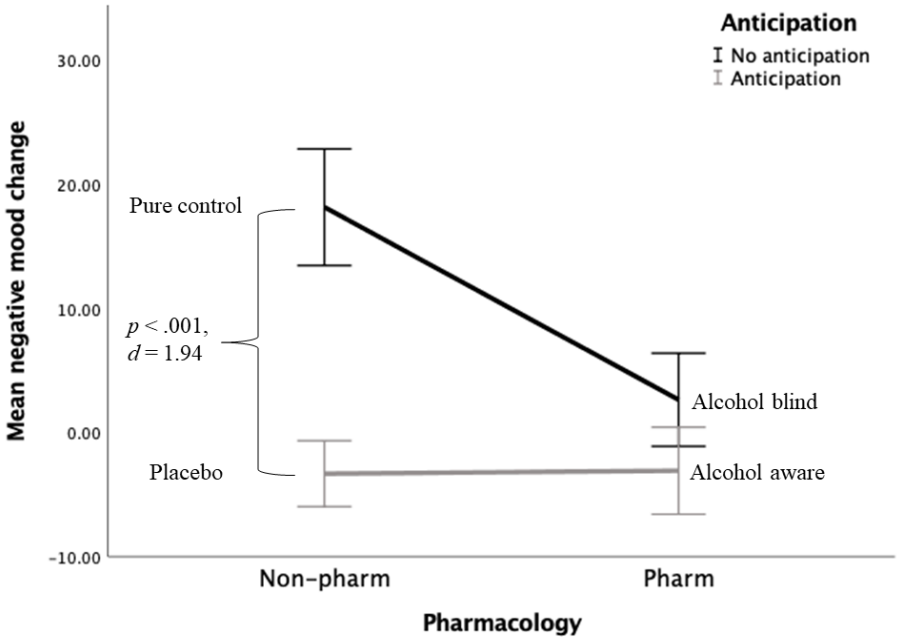

Figure 1: Mean and standard errors bars for negative mood state change for both anticipation and pharmacological conditions.

A 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVA was employed to assess changes in subjective intoxication. There was a significant effect of anticipation on subjective intoxication $F(1, 95) = 39.91$, $p < .001$, $\eta_p^2 = .30$, with subjective intoxication higher in the anticipation condition (i.e., placebo and alcohol aware). Also, a significant effect of pharmacology was observed on subjective intoxication $F(1, 95) = 51.34$, $p < .001$, $\eta_p^2 = .35$. Specifically, subjective intoxication was higher for pharmacological conditions (i.e., alcohol aware and alcohol blind). There was also a significant anticipation x pharmacology interaction $F(1, 95) = 4.18$, $p = .04$, $\eta_p^2 = .04$.

Bonferroni pairwise comparisons indicating that for both non-pharmacological (i.e., placebo; $p < .001$, $d = 1.94$) and pharmacological conditions (i.e., alcohol aware; $p = .003$, $d = .79$), anticipation resulted in higher ratings of intoxication than where there was no anticipation of alcohol. See figure 2 for means and standard errors.

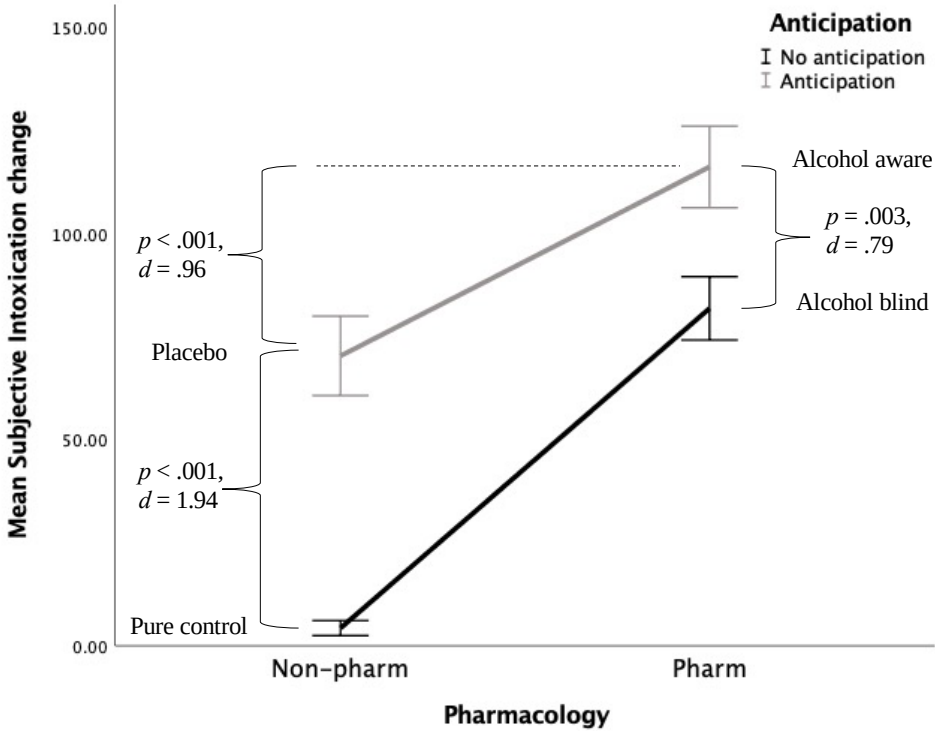

Figure 2: Mean and standard errors for subjective intoxication for both anticipation and pharmacological conditions.

Inhibitory control

A 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVA were utilised to examine changes in stop-signal reaction time (SSRT), controlling for AUDIT and trait impulsivity (BIS-11). There was a significant main effect of anticipation $F(1,94) = 6.16$, $p = .015$, $\eta_p^2 = .06$, demonstrating that where there was anticipation of alcohol (i.e., plaebo and alcohol aware) there were greater impairments in inhibitory control compared to conditions where there was no anticipation (i.e., pure control and alcohol blind). There was also a main effect of pharmacology $F(1, 94) = 21.43$, $p < .001$, $\eta_p^2 = .19$, with

pharmacological conditions resulting in greater inhibitory control impairments. These main effects were qualified by a significant anticipation x pharmacology interaction was observed $F(1, 94) = 9.86$, $p = .002$, $\eta_p^2 = .10$, with Bonferroni corrected pairwise comparisons revealing significantly more impaired inhibitory control in non-pharmacological conditions with anticipation (placebo) versus where there was no anticipation (pure control) ($p < .001$, $d = 1.37$), but no significant difference between anticipation (in the alcohol aware condition) and no anticipation (the alcohol blind condition) for pharmacological conditions ($p = .56$, $d = .15$). See figure 3 for means and standard errors.

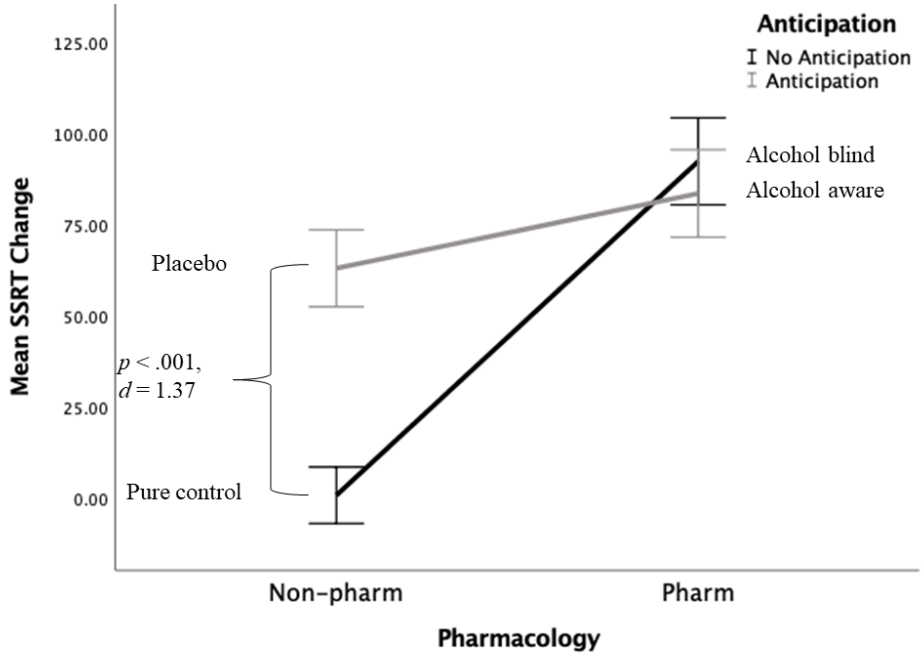

Figure 3: Means and standard errors for stop-signal reaction time (SSRT) for both anticipation and pharmacological conditions

Attentional Bias

A 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVA examined changes in alcohol-related attentional bias (AB), controlling for AUDIT and BIS-11. The effect of anticipation was non-significant $F(1, 94) = 1.56$, $p = .21$, $\eta_p^2 = .02$. There was however, a significant main effect of pharmacology $F(1, 94) = 5.33$, $p < .025$, $\eta_p^2 = .05$, with alcohol-related AB significantly higher in the non-pharmacological conditions.

There was also a significant anticipation x pharmacology interaction $F(1, 96) = 4.31$, $p = .04$, $\eta_p^2 = .04$. Bonferroni pairwise comparisons revealed that within non-pharmacological conditions, AB was significantly elevated where there was anticipation of alcohol (in the placebo condition) rather than where there was no anticipation (the pure control condition) ($p = .015$, $d = 1.07$). In pharmacological conditions, there was no difference in AB irrespective of anticipation ($p = .48$, $d = .15$). See figure 4 for means and standard errors[1].

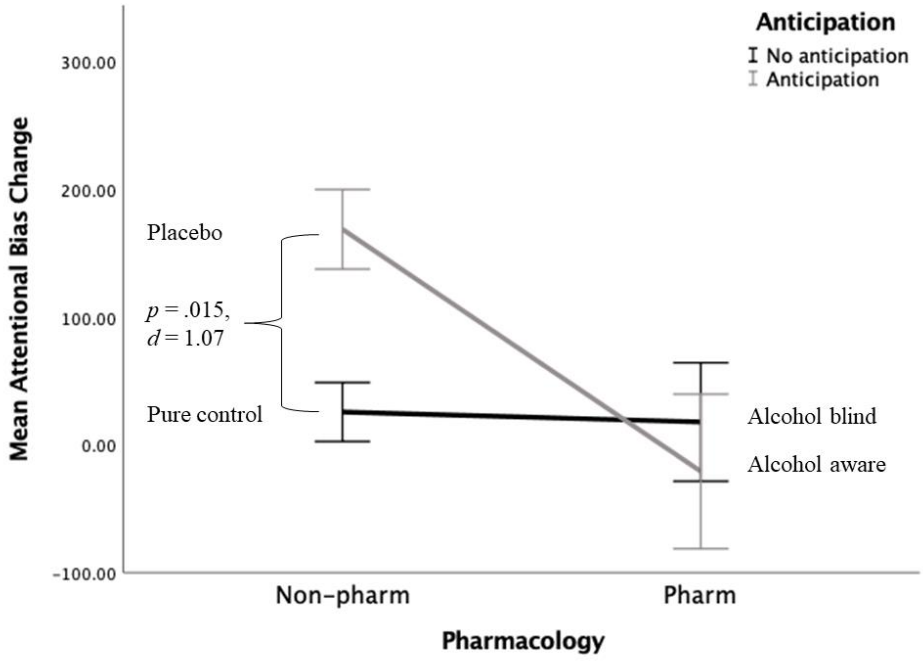

Figure 4: Means and standard errors for alcohol-related attentional bias (AB) for both anticipation and pharmacological conditions. (NB: Positive AB change scores indicate greater dwell time on alcohol-related images compared to neutral)

Craving

A 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVA were utilised to assess craving change, controlling for AUDIT. The was a significant main effect of anticipation $F(1, 95) = 21.05$, $p < .001$, $\eta_p^2 = .18$, demonstrating

[1] Positive AB values are indicative of greater dwell time on alcohol-related images compared with neutral images.

elevated craving associated with anticipation (i.e., placebo and alcohol aware) There was also a significant main effect of pharmacology $F(1, 95) = 35.26$, $p < .001$, $\eta_p^2 = .27$, with pharmacological conditions showing higher craving (alcohol aware and alcohol blind). There was however, no significant anticipation x pharmacology interaction $F(1, 95) = .91$, $p = .34$, η_p^2 .01. See figure 5 for means and standard errors.

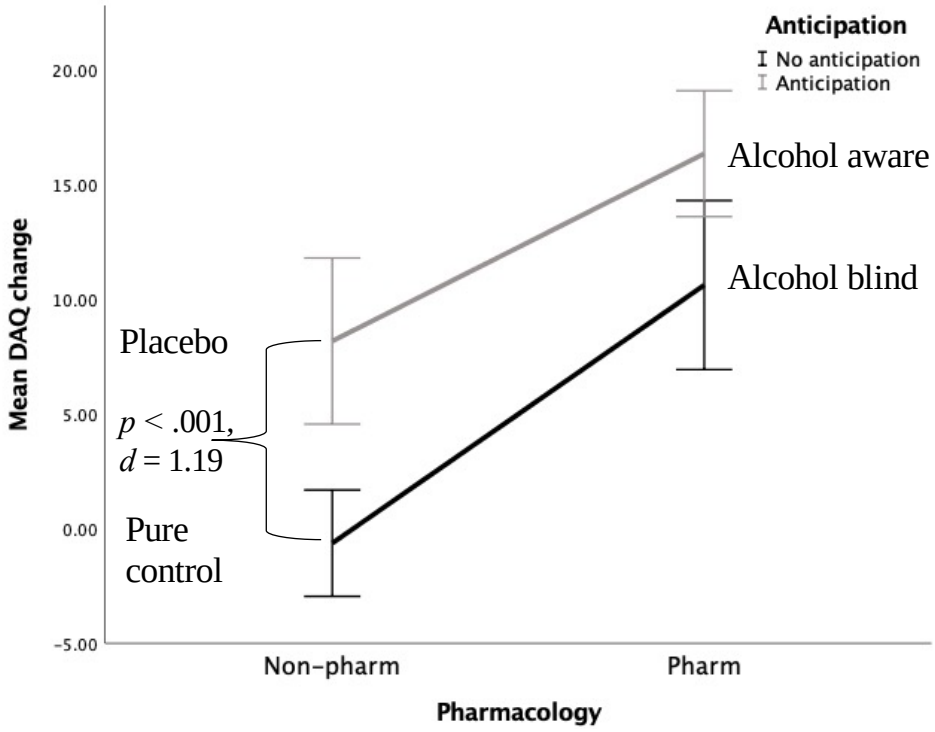

Figure 5: Means and standard errors for Desires for Alcohol (DAQ; craving) for both anticipated and pharmacology conditions.

Supplementary correlation analysis was undertaken to examine the relationship between attentional bias and craving. Overall, there was no correlation evident $r = .09$, $p = .35$. When analysed by condition however, a significant correlation was seen for anticipation/non-pharmacological (placebo) $r = .48$, $p < .025$, but not for any other conditions (p's > .29).

Ad libitum consumption

A 2 (anticipation vs no anticipation) x 2 (pharmacological vs non-pharmacological) ANCOVA were employed to test the effect of beverage administration on *ad libitum*

consumption, controlling for AUDIT and BIS-11. There was no main effect of anticipation $F(1, 94) = 1.24$, $p = .27$, $\eta_p^2 = .01$, but there was a significant effect of pharmacology $F(1, 94) = 21.00$, $p < .001$, $\eta_p^2 = .18$. Specifically, pharmacological conditions resulted in higher *ad libitum* consumption. There was no anticipation x pharmacology interaction found $F(1, 94) = .05$, $p = .82$, $\eta_p^2 = .00$. See figure 6 for means and standard errors.

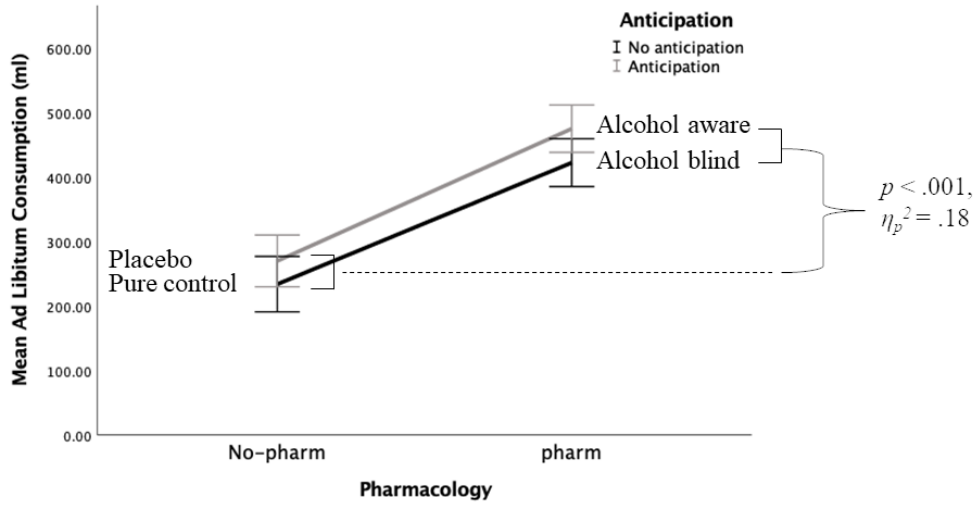

Figure 6: Means and standard errors for ad libitum consumption for both anticipated and pharmacology conditions.

Mediation

All Mediation analyses were undertaken using the PROCESS 3.4 macro for SPSS. First, to assess if SSRT change mediate the relationship between initial intoxication and subsequent consumption, the SSRT change score was calculated by subtracting the pre-beverage SSRT from post-beverage SSRT. This new variable was then used as the mediator in the analysis. Using the multicategorical function, dummy variables were formed, comparing each condition to control (X1 = placebo vs pure control, X2 = alcohol blind vs pure control, X3 = alcohol aware vs pure control). There was a significant direct effect (*c* path) of alcohol present conditions on *ad libitum* consumption, X2 $t(96) = 3.39$, $p < .01$, 95%CI [78.10, 299.02], X3 $t(96) = 4.34$, $p < .001$, 95%CI [130.82, 351.74], however, the placebo condition did not show elevated consumption X1 $t(96) = .65$, $p = .52$, 95%CI [-74.46, 146.46]. Both

alcohol and placebo conditions predicted changes in SSRT (*a* path), X1 $t(96) = 5.01$, $p < .001$, 95%CI [40.53, 93.67], X2 $t(96) = 6.98$, $p < .001$, 95%CI [66.92, 120.07], X3 $t(96) = 8.14$, $p < .001$, 95%CI [83.44, 135.58]. SSRT change however, did not predict *ad libitum* consumption (*b* path) $t(95) = .66$, $p = .51$, 95%CI [-.56, 1.12]. The *c'* pathway however, remained significant for alcohol conditions X2 $t(95) = 2.37$, $p < .025$, 95%CI [26.32, 298.45], X3 $t(95) = 2.90$, $p < .025$, 95%CI [66.67, 354.84] and placebo remaining non-significant X1 $t(95) = .66$, $p = .78$ 95%CI [-107.24, 141.68]. Indicating that SSRT change did not act as a mediator. See Figure 7 for mediation model.

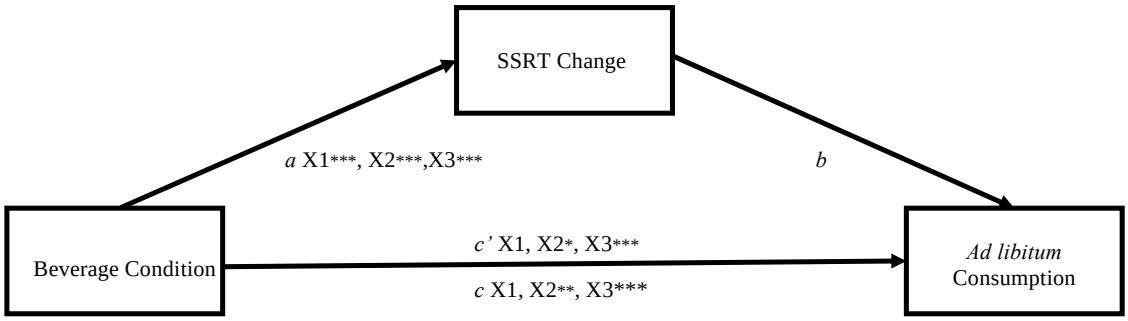

Figure 7: Mediation model examining change in stop-signal reaction time (SSRT) post-beverage as a mediator in the relationship between beverage condition and *ad libitum* consumption. Dummy variables computed comparing each experimental condition to pure control (X1 = placebo vs pure control, X2 = alcohol blind vs pure control, X3 = alcohol aware vs pure control). *$p < .05$, **$p < .01$, ***$p < .001$

Second, to assess if changes in craving as measured by the DAQ mediates the relationship between initial intoxication and subsequent consumption, a craving change score was calculated by subtracting the pre-beverage DAQ score from the post-beverage score. This new variable was used as the mediator in the analysis. Using the multicategorical function dummy variables were formed, comparing each condition to control (X1 = placebo vs pure control, X2 = alcohol blind vs pure control, X3 = alcohol aware vs pure control). For *c* path,

values remained the same (see Figure 7). Both alcohol and placebo condition predicted changes in craving (*a* path), X1 $t(96) = 3.96$, $p < .001$, 95%CI [4.39, 13.21], X2 $t(96) = 5.06$, $p < .001$, 95%CI [6.83, 15.65], X3 $t(96) = 7.63$, $p < .001$, 95%CI [12.54, 21.37]. Change in craving predicted *ad libitum* consumption (*b* path) $t(95) = 1.99$, $p < .05$, 95%CI [.019, 10.01]. The *c'* pathway however, remained significant for alcohol conditions X2 $t(95) = 2.14$, $p < .05$, 95%CI [9.75, 254.64], X3 $t(95) = 2.25$, $p < .05$, 95%CI [18.31, 294.14]. Therefore, indicating that pharmacologically driven changes in craving partially mediate subsequent consumption. See Figure 8 for mediation model.

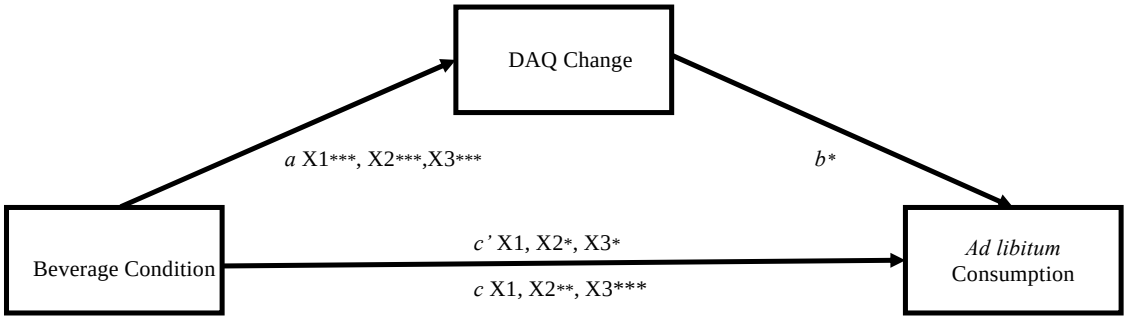

Figure 8: Mediation model examining change in craving post-beverage as a mediator in the relationship between beverage condition and *ad libitum* consumption. Dummy variables computed comparing each experimental condition to pure control (X1 = placebo vs pure control, X2 = alcohol blind vs pure control, X3 = alcohol aware vs pure control). *$p < .05$, **$p < .01$, ***$p < .001$

A final mediation model was conducted with the same parameters as above, this time to assess the role of AB as a potential mediator between beverage condition and *ad libitum* consumption. Pre-beverage AB was subtracted from post-beverage AB to provide a AB change score which was used as the mediator in the model. See previous mediation analysis for *c* path, as values remained the same. Only placebo predicted AB change (*a* path) X1 $t(96) = 2.37$, $p < .025$, 95%CI [23.05, 263.23]. AB change did not significantly predict *ad libitum* consumption (*b* path) $t(95) = .05$, $p = .96$, 95%CI [-.19, .18]. The *c'* path remained stable with alcohol blind X2 $t(95) = 3.37$, $p < .01$, 95%CI [77.46, 299.59] and alcohol aware X3 3

t(95) = 4.30, *p* < .001, 95%CI [129.69, 352.47], but not placebo *t*(95) = .63, *p* = .53, 95%CI [-77.63, 150.85] predicting *ad libitum* consumption. Indicating no mediation present. See figure 9 for mediation model.

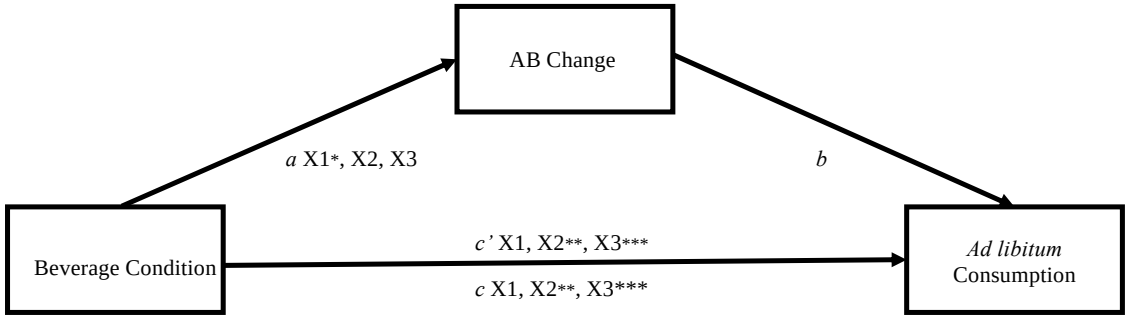

Figure 9: Mediation model examining change in AB post-beverage as a mediator in the relationship between beverage condition and *ad libitum* consumption. Dummy variables computed comparing each experimental condition to pure control (X1 = placebo vs pure control, X2 = alcohol blind vs pure control, X3 = alcohol aware vs pure control). *p < .05, **p < .01, ***p < .001

Discussion

The current study introduced a novel alcohol blind administration condition whereby participants were administered a dose of alcohol under deceptive conditions. Specifically, it combined this alcohol blind condition alongside widely-used administration techniques, which use the following two or three conditions: alcohol aware [alcohol consumed with participants' knowledge], pure control [no alcohol supplied and participants are aware of this] and placebo [no alcohol consumed but participants are deceived). In so doing, this allowed for better isolation of alcohol's pharmacological and anticipated effects, and for systematic exploration of their relative influence on inhibitory control, attentional bias, craving and alcohol seeking behaviours. Findings can be summarised as follows. In line with the first hypothesis, both the anticipatory and pharmacological effects of alcohol (placebo, alcohol aware, alcohol blind conditions) resulted in inhibitory control impairments and elevated craving. The pharmacological effects of alcohol (evident in alcohol aware and

alcohol blind conditions) also appeared to be associated with the greatest changes in inhibitory control and craving, in accordance with hypothesis two. The anticipatory but not the pharmacological effects of alcohol resulted in elevated alcohol-related attentional bias, in-keeping with the prediction of hypothesis three and, in line with hypothesis four, *Ad libitum* consumption was heightened as a consequence of pharmacology, but not anticipation. Finally, as indicated in hypothesis five, increased *ad libitum* consumption associated with alcohol pharmacology was partially mediated by changes in craving, but not transient changes in inhibitory control as had also been predicted.

In comparison with anticipated effects, the current findings identify alcohol's pharmacological effects as the primary drivers of subsequent consumption. Specifically, elevated *ad libitum* consumption was only evident in the pharmacological conditions (in the alcohol aware and blind conditions), suggesting anticipation does not exert an influence on continued consumption. This is consistent with research findings which show heightened consumption following alcohol, but not placebo (Christiansen et al, 2013; McNeill et al., 2021) and, to our knowledge, only one comparable study finding heightened consumption following placebo (Chirstiansen et al., 2017). Current findings from the alcohol blind beverage condition suggest that in the absence of anticipation, *ad libitum* consumption was akin to that observed in the traditional alcohol aware condition (where there is both the impact of pharmacology with anticipation). Moreover, pharmacological conditions displayed the greatest elevation in craving levels, with associations between the craving and activation in the dorsal striatum being well documented in the literature (see Volkow et al., 2016). Current findings, in this way, lend support to the notion that the maintenance of consumption is driven to a greater degree by alcohol's pharmacological, as imposed to its anticipatory, effects.

However, while our research did not directly implicate the anticipated effects of alcohol in successive consumption, their influence on the alcohol-related cognitions could be ascertained. Specifically, anticipation was associated with significant increases in alcohol-related attentional bias. This is consistent with the 'satiation hypothesis' suggesting that the absence of 'reward' satiation in striatum activates hypervigilance for alcohol-related stimuli, in an attempt to attain satiety (Monem & Fillmore, 2019). Specifically, findings by Monem and colleagues indicate that attentional bias for alcohol-related stimuli show dose-dependent decreases; however, the same pattern of decreases was not observed for other appetitive stimuli (ibid). These findings are also consistent with those suggestive of elevated attentional bias following placebo (McNeill et al., 2021). However, they are inconsistent with the notion that that elevated levels of craving should be associated with heightened attentional bias (e.g., Franken, 2003). In this way, the current findings showed elevated attentional bias due to anticipation and, while this was correlated with changes in craving, the relationship was not observed in the other conditions. Furthermore, craving associated with anticipation was lower in anticipatory than in pharmacological conditions, neither of which appeared to exhibit pronounced attentional bias. Therefore, future research is required to disentangle the evidently nuanced relationship between craving and attentional bias, and the role anticipation may play.

In relation to inhibitory control and craving, the present findings do not appear to offer support for notion that anticipation exerts an additive effect on the impact of alcohol intoxication (e.g., Fillmore & Blackburn, 2002). Specifically, it appears that impaired inhibition and elevations in craving may be associated with alcohol's anticipated effects; these changes were significantly more pronounced in relation to alcohol pharmacology.

These findings were not unexpected and are observed in the literature (e.g., McNeill et al., 2021). However, the administration of alcohol by deception (in the absence of anticipation – the alcohol blind condition) appears to shed new light on the relative contributions of anticipation and pharmacology where inhibitory control and craving are concerned. Importantly, the absence of significant differences in inhibition and craving between alcohol aware and blind conditions suggests that observed changes in inhibitory control and craving may be driven solely by the pharmacological effects of alcohol. In short, while anticipation alone may impact these important processes, according to our initial findings using this novel paradigm, alcohol intoxication appears to supersede any effects on these processes. Future research incorporating such alcohol blind designs is clearly required to explore this further.

Finally, although inhibitory control impairments were evident across all experimental manipulations, these did not appear to mediate *ad libitum* consumption, as previously suggested (Field et al., 2010; Jones et al., 2013). Rather, craving appeared to emerge as a more central influence on the initiation and maintenance of drinking, indicated by the partial mediatory association between initial intoxication and *ad libitum* consumption. This extends previous work indicating that transient impairments in inhibitory control are not directly associated with heightened consumption (McNeill et al., 2018; McNeill et al., 2021) and that implementation intentions and craving appear to supersede daily fluctuations in inhibitory control in predicting drinking episodes (Jones et al., 2018). Overall, there therefore appears to be a growing convergence of evidence to support the assertion that successive consumption may be driven primarily by the pharmacological effects of alcohol which are exerted via changes in craving.

Several limitations must be borne in mind when considering current findings. First, increases in subjective intoxication were observed in the alcohol blind condition. While subjective intoxication did significantly increase from baseline, it did not vary from placebo and more importantly, it was significantly lower than in the alcohol aware condition. So, while one can be reasonably assured that participants in the alcohol blind condition were deceived regarding the presence of alcohol, it should be noted that anticipated effects cannot be ruled out entirely. In future, qualitative assessment of the degree to which participants were deceived concerning the presence of alcohol in the alcohol blind condition should be undertaken during debriefing, as this could afford an additional layer of certainty. Indeed, procedural signalling (Davies & Best, 1996) could mean that probing participants about alcohol-related craving may have intimated the presence of alcohol and it may therefore be possible to refine how participants are briefed about the research. In short, while the methodology developed may constitute a useful step forward in untangling the pharmacological from the anticipated effects of alcohol, there would appear to be room for further refinement.

It should also be noted that, as with much of the laboratory-based alcohol research, the sample was student-centric. As such, future research is recommended in order to examine the extent to which the current findings generalise to wider populations where heavier, atypical drinking behaviour is less common (Borsari & Carey, 2001; Karam et al., 2007; Knight et al., 2002). Finally, it should be noted that the visual prob task produced low reliability levels. While this has also been found in other research in this area (e.g., Field & Christiansen, 2012), future research may benefit from the development of more rigorous cognitive tasks and more appropriate stimuli (Pennington et al., 2021) to verify that the current findings can be replicated using other methodological approaches.

In conclusion, the current study incorporated a novel alcohol blind administration method (alongside alcohol aware, placebo and pure control conditions) in a first attempt to disentangle the pharmacological from the anticipated effects of alcohol on inhibitory control, attentional bias, and craving. Findings suggest that when compared with anticipation, pharmacology emerges as the primary driver of subsequent alcohol consumption. Specifically, the current research indicates that pharmacology induces the greatest cognitive changes and that the associated increases in consumption are mediated by changes in craving and not inhibitory control. Overall, it therefore appears prudent for future research to recognise the apparent dominant impact of alcohol's pharmacological influences on craving, and we hope to have helped map out a new way of examining systematically the nuanced ways in which these processes interact to shape alcohol drinking behaviours.

References

Band, G. P. H., van der Molen, M. W., & Logan, G. D. (2003). Horse-race model simulations of the stop-signal procedure. *Acta Psychologica*, *112*(2), 105–142.

Borsari, B., & Carey, K. B. (2001). Peer influences on college drinking: A review of the research. *Journal of Substance Abuse*, *13*(4), 391–424.

Caswell, A. J., Morgan, M. J., & Duka, T. (2013). Acute alcohol effects on subtypes of impulsivity and the role of alcohol-outcome expectancies. *Psychopharmacology*, *229*(1), 21–30.

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

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

388.

Christiansen, P., Mansfield, R., Duckworth, J., Field, M., & Jones, A. (2015). Internal reliability of the alcohol-related visual probe task is increased by utilising personalised stimuli and eye-tracking. *Drug and Alcohol Dependence*, *155*, 170–174.

Christiansen, P., Townsend, G., Knibb, G., & Field, M. (2017). Bibi ergo sum: the effects of a placebo and contextual alcohol cues on motivation to drink alcohol. *Psychopharmacology*, *234*(5), 827–835.

Davies, J. B., & Best, D. W. (1996). Demand characteristics and research into drug use. *Psychology & Health*, *11*(2), 291–299.

de Wit, H. (1996). Priming effects with drugs and other reinforcers. *Experimental and Clinical Psychopharmacology*, *4*(1), 5–10.

Duka, T., & Townshend, J. M. (2004). The priming effect of alcohol pre-load on attentional bias to alcohol-related stimuli. *Psychopharmacology*, *176*(3–4), 353–362.

Fernie, G., Christiansen, P., Cole, J. C., Rose, A. K., & Field, M. (2012). Effects of 0.4g/kg alcohol on attentional bias and alcohol-seeking behaviour in heavy and moderate social drinkers. *Journal of Psychopharmacology*, *26*(7), 1017–1025.

Field, M., & Christiansen, P. (2012). Commentary on Ataya et al. (2012), Internal reliability of measures of substance-related cognitive bias. *Drug and Alcohol Dependence*, *124*(3), 189–190.

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence*, *97*(1–2), 1–20.

Field, M., & Jones, A. (2017). Elevated alcohol consumption following alcohol cue exposure is partially mediated by reduced inhibitory control and increased craving. *Psychopharmacology*, *234*(19), 2979–2988.

Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute

alcohol effects on inhibitory control and implicit cognition: Implications for loss of control over drinking. *Alcoholism: Clinical and Experimental Research*, *34*(8), 1346–1352.

Fillmore, M. T., & Blackburn, J. (2002). Compensating for alcohol-induced impairment: Alcohol expectancies and behavioral disinhibition. *Journal of Studies on Alcohol*, *63*(2), 237–246.

Fillmore, M. T., & Weafer, J. (2013). Behavioral Inhibition and Addiction. In *The Wiley-Blackwell Handbook of Addiction Psychopharmacology* (pp. 135–164). Wiley-Blackwell.

Franken, I. H. A. (2003). Drug craving and addiction: Integrating psychological and neuropsychopharmacological approaches. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *27*(4), 563–579.

Jones, A., Christiansen, P., Nederkoorn, C., Houben, K., & Field, M. (2013). Fluctuating disinhibition: Implications for the understanding and treatment of alcohol and other substance use disorders. *Frontiers in Psychiatry*, *4*, 1–36.

Jones, A., Guerrieri, R., Fernie, G., Cole, J., Goudie, A., & Field, M. (2011). The effects of priming restrained versus disinhibited behaviour on alcohol-seeking in social drinkers. *Drug and Alcohol Dependence*, *113*(1), 55–61.

Jones, A., Tiplady, B., Houben, K., Nederkoorn, C., & Field, M. (2018). Do daily fluctuations in inhibitory control predict alcohol consumption? An ecological momentary assessment study. *Psychopharmacology*, *235*(5), 1487–1496.

Karam, E., Kypri, K., & Salamoun, M. (2007). Alcohol use among college students: An international perspective. *Current Opinion in Psychiatry*, *20*(3), 213–221.

Knight, J. R., Wechsler, H., Kuo, M., Seibring, M., Weitzman, E. R., & Schuckit, M. A. (2002). Alcohol abuse and dependence among U.S. college students. *Journal of Studies*

on Alcohol, *63*(3), 263–270.

Logan, G. D., Cowan, W. B., & Davis, K. A. (1984). On the ability to inhibit simple and choice reaction time responses: A model and a method. *Journal of Experimental Psychology: Human Perception and Performance*, *10*(2), 276–291.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science*, *8*(1), 60–64.

Love, A., James, D., & Willner, P. (1998). A comparison of two alcohol craving questionnaires. *Addiction*, *93*(7), 1091–1102.

MacAndrew, C., & Edgerton, R. B. (1969). *Drunken comportment : a social explanation*. Percheron Press.

Marlatt, G. A., Demming, B., & Reid, J. B. (1973). Loss of control drinking in alcoholics: an experimental analogue. *Journal of Abnormal Psychology*, *81*(3), 233–241.

McNeill, A., Monk, R. L., Qureshi, A. W., Makris, S., & Heim, D. (2018). Continuous Theta Burst Transcranial Magnetic Stimulation of the Right Dorsolateral Prefrontal Cortex Impairs Inhibitory Control and Increases Alcohol Consumption. *Cognitive, Affective and Behavioral Neuroscience*, *18*(6), 1198–1206.

McNeill, A. M., Monk, R. L., Qureshi, A. W., Litchfield, D., & Heim, D. (2021). The effects of placebo and moderate dose alcohol on attentional bias, inhibitory control and subjective craving. *Alcohol and Alcoholism* https://doi.org/10.1093/alcalc/agab011

Monem, R., & Fillmore, M. T. (2019). Alcohol Administration Reduces Attentional Bias to Alcohol-related But Not Food-related Cues: Evidence for a Satiety Hypothesis HHS Public Access. *Psychol Addict Behav*, *33*(8), 677–684.

Monk, R. L., Sunley, J., Qureshi, A. W., & Heim, D. (2016). Smells like inhibition: The effects of olfactory and visual alcohol cues on inhibitory control. *Psychopharmacology*, *233*(8), 1331–1337.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology*, *51*(6), 768–774.

Pennington, C. R., Jones, A., Bartlett, J. E., Copeland, A., & Shaw, D. J. (2021). Raising the bar: improving methodological rigour in cognitive alcohol research. *Addiction*, 10.1111/add.15563. Advance online publication. https://doi.org/10.1111/add.15563

Rose, A K, Hobbs, M., & Drummond, C. (2013). Differentiating the Contribution of Pharmacological from Alcohol Expectancy Effects to Changes in Subjective Response and Priming Over Successive Drinks. *Alcoholism: Clinical and Experimental Research*, *37*(4), 687–695.

Rose, A K, Jones, A., Clarke, N., & Christiansen, P. (2014). Alcohol-induced risk taking on the BART mediates alcohol priming. *Psychopharmacology*, *231*(11), 2273–2280.

Rose, Abigail Katherine, & Grunsell, L. (2008). The subjective, rather than the disinhibiting, effects of alcohol are related to binge drinking. *Alcoholism: Clinical and Experimental Research*, *32*(6), 1096–1104.

Saunders, J. B., Aasland, O. G., Babor, T. F., & la Fuente de. (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption: II. *Addiction*, *88*(6), 791–804.

Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology*, *197*(1), 169–178.

Sobell, L. C., & Sobell, M. B. (1990). Self-report issues in alcohol abuse: State of the art and future directions. *Behavioral Assessment*, *12*(1), 77–90.

Spanakis, P., Jones, A., Field, M., & Christiansen, P. (2019). A Stroop in the Hand is Worth Two on the Laptop: Superior Reliability of a Smartphone Based Alcohol Stroop in the Real World. *Substance Use and Misuse*, *54*(4), 692–698.

Verbruggen, F., Logan, G. D., & Stevens, M. A. (2008). STOP-IT: Windows executable software for the stop-signal paradigm. *Behavior Research Methods*, *40*(2), 479–483.

Volkow, N. D., Koob, G. F., & McLellan, A. T. (2016). Neurobiologic Advances from the Brain Disease Model of Addiction. *New England Journal of Medicine*, *374*(4), 363–371.

Weafer, J., & Fillmore, M. T. (2008). Individual differences in acute alcohol impairment of inhibitory control predict ad libitum alcohol consumption. *Psychopharmacology*, *201*(3), 315–324.

Weafer, J., & Fillmore, M. T. (2016). Low-Dose Alcohol Effects on Measures of Inhibitory Control, Delay Discounting, and Risk-Taking. *Current Addiction Reports*, *3*(1), 75–84.