**Abstract**

A considerable evidence base has demonstrated that priming doses of alcohol impair inhibitory control and activate motivation to consume alcohol. There is, however, a lack of studies investigating the effect of placebo-alcohol on these processes and their association with alcohol outcome expectancies (AOE). We investigated the effect of placebo-alcohol on craving and inhibitory control, and the extent to which placebo effects correlated with AOE in 32 non-dependent drinkers. Participants completed questionnaires assessing typical alcohol use (fortnightly alcohol consumption, AUDIT) and AOE (measured using the Alcohol Outcome Expectancy Scale). On a within subjects basis participants consumed a placebo-alcohol drink and control drink. Measures of craving were taken pre and post-drink and participants completed a Go/No-Go task following the drink. Craving was increased by the placebo-alcohol and, importantly, placebo-alcohol impaired inhibitory control. Furthermore expectancies of cognitive and behavioural impairment were correlated with Go/No-Go task performance following a placebo. Increases in craving were associated with a range of elevated outcome expectancies. This suggests that the anticipated effects of alcohol can impair inhibitory control and increase craving; therefore studies using placebo vs. alcohol comparisons relativeto studies using a pure no-alcohol control are underestimating the real-world effect of alcohol on these processes which is a combination of pharmacological and anticipated effects of alcohol. Furthermore, individual differences in AOE may influence reactivity to the anticipated effects of alcohol.

Key words: Alcohol, Expectancies, Inhibition, Craving, Placebo,

**Introduction**

Recent research examining the determinants of loss of control over drinking has focused on the acute effect of alcohol on the substrates of behavioural regulation, particularly inhibitory control. There is a substantial evidence base demonstrating that moderate priming doses of alcohol (≥0.4g/kg) impair inhibitory control (e.g. [de Wit, Crean, & Richards, 2000](#_ENREF_7);  [Fillmore, Blackburn, & Harrison, 2008](#_ENREF_12); [Marczinski & Fillmore, 2005](#_ENREF_23)), as well as other measures of executive cognitive functioning ([Balodis, Johnsrude, & Olmstead, 2007](#_ENREF_2); [Christiansen, Rose, Cole, & Field, 2013](#_ENREF_4); [Guillot, Fanning, Bullock, McCloskey, & Berman, 2010](#_ENREF_19)). Similarly, alcohol primes have also been demonstrated to stimulate objective (e.g. *ad lib* consumption, alcohol choice) and subjective (e.g. craving) measures of alcohol seeking (e.g. [Christiansen, Cole, Goudie, & Field, 2012](#_ENREF_3); [De Wit & Chutuape, 1993](#_ENREF_6); [Fernie, Christiansen, Cole, Rose, & Field, 2012](#_ENREF_10); [Rose & Grunsell, 2008](#_ENREF_27)).

The anticipated effects of alcohol have, however, received considerably less attention. Stimulus substitution models argue that placebos produce drug-like effects when the unconditioned stimuli (the vehicle a drug is delivered in; e.g. a liquid that tastes and smells like alcohol) evokes conditioned responses after repeated pairings (Wickramasekera, 1990), although this is dependent upon conscious expectations of an effect (Montgomery & Kirsch, 1997). Significantly, both increased *ad lib* consumption and craving has been found following the administration of placebo-alcohol (e.g. [Christiansen, et al., 2013](#_ENREF_4); [Marlatt, Demming, & Reid, 1973](#_ENREF_24); [Rose, Hobbs, & Drummond, 2013](#_ENREF_28)). Moreover, [Leeman, Corbin, & Fromme, (2009](#_ENREF_20)) demonstrated that levels of craving following a placebo (but not alcohol) predicted *ad-lib* consumption. This suggests that increased desire to consume alcohol following an alcohol prime is, in part, the product of the anticipated effects of alcohol. Therefore, if studies wish to be analogues for real world priming they should also investigate the anticipated effects of alcohol, (i.e. an alcohol condition compared to a no expectancy, no alcohol condition) rather than just the pharmacological effects of alcohol (i.e., commonly used comparison between alcohol and placebo conditions).

Although the anticipated effects of alcohol have been demonstrated to increase alcohol seeking (as well as self report aggression and sexual arousal, for a meta analysis Hull & Bond 1986) findings regarding the anticipated effects of alcohol on cognitive processes are equivocal. There is evidence that placebo-alcohol primes automatic alcohol-approach tendencies ([Christiansen, et al., 2013](#_ENREF_4)), even though alcohol consumption does not ([Schoenmakers, Wiers, & Field, 2008](#_ENREF_31)). There is a lack of studies investigating the effects of placebo-alcohol on inhibitory control (or indeed working memory and switching, the hypothesised components of executive functioning) although attentional processing (measured with a Posner task) is slowed by placebo- alcohol ([Gilbertson, Prather, & Jo Nixon, 2010](#_ENREF_18)). A more significant body of research has focused on cognitive-motor performance. Reaction times are impaired by placebo-alcohol ([Finnigan, Hammersley, & Millar, 1995](#_ENREF_16)) as is performance on a pursuit rotor task after consuming placebo-alcohol ([Fillmore & Vogel-Sprott, 1994](#_ENREF_14); [Fillmore & Vogel-Sprott, 1995](#_ENREF_15)), with the former study finding impairment in those who believe alcohol impairs motor performance. Some studies investigating the effects of alcohol on cognitive-motor performance have found compensation for expected impairment. For example, experienced male drinkers exhibited improved performance on a pursuit rotor task following placebo-alcohol compared to less experienced drinkers ([Fillmore & Vogel-Sprott, 1996](#_ENREF_11)). Furthermore, explicit manipulation of task-specific expectancies (telling participants that alcohol will impair performance in the task conducted) has also been shown to improve performance following placebo-alcohol as participants compensate for task-specific impairment ([Fillmore, Mulvihill, & Vogel-Sprott, 1994](#_ENREF_13)).

Taken together, these studies suggest placebo-alcohol produces impairments in motor performance and attentional control; although compensatory effects on motor performance may evident in specific groups (i.e. experienced male drinkers) or when there has been task-specific explicit manipulation of expected impairment. The findings of [Fillmore & Vogel-Sprott, (1994](#_ENREF_14)) indicate the extent to which placebo-alcohol affects cognitive performance may be, in part, attributable to individual differences in alcohol outcome expectancies (AOE; for a related argument see Montgomery & Kirsch, 1997). AOE are beliefs that people have developed, through experience and observation, concerning the subjective effects of alcohol once consumed ([Donovan, Molina, & Kelly, 2009](#_ENREF_8)). Questionnaire assessment of AOE generally describe outcomes in terms of positive expectancies, (e.g. social facilitation and tension reduction), and negative expectancies, (e.g. negative affect, aggression; [Fromme & D'Amico, 2000](#_ENREF_17); [Leigh & Stacy, 1993](#_ENREF_21)). Of particular importance to investigations into cognitive performance is expectation of cognitive impairment. If participants have developed a broad expectancy that alcohol impairs their cognitive performance then a placebo-alcohol prime should produce impairment.

The current study aimed to investigate the effects of placebo-alcohol on craving and inhibitory control and determine whether AOE were associated with these effects. This will address the lack of research on the effects of placebo-alcohol on inhibitory control and the extent to which beliefs about the subjective effects of alcohol are associated with placebo effects. On a within subjects basis 32 participants consumed a control drink and a placebo-alcohol drink. Craving and subjective intoxication was measured pre and post-drink and participants completed a Go/No-Go task following drink consumption. We hypothesised we would replicate our previous finding that placebo-alcohol increased alcohol craving ([Christiansen, et al., 2013](#_ENREF_4)). We also hypothesised that the placebo alcohol would impair inhibitory control which would be associated with expectancies of cognitive impairment.

**Method**

**Participants**

An opportunity sample of thirty two participants (twenty-one female) aged between 18 and 23 years (mean 19.40 ±1.60) were recruited via word of mouth and intranet advertising from the University of Liverpool. Participants were invited to take part if they self-reported regular consumption of alcohol (at least one alcoholic drink per week). The study was approved by the University of Liverpool Ethics Committee.

**Design**

A within subjects design was employed: All participants completed a placebo session and a control session (completed one week apart, with order of conditions counterbalanced) and measures of craving and intoxication were taken twice (pre-drink and post-drink). The Go/No-Go was completed once in each session.

**Materials**

***Drink preparation (based on*** [***Christiansen, et al., 2013***](#_ENREF_4)***)***

The placebo-alcohol drink consisted of 500ml chilled lemonade with Vodka applied to the rim of the glass; an atomiser was used to spray vodka mist on the surface of the drink. The control drink consisted of 500ml chilled water only. Participants were informed the drink was alcoholic in the placebo condition, and that it was non-alcoholic in the control condition.

***Questionnaires***

*Time Line Follow Back (TLFB;* [*Sobell & Sobell, 1990*](#_ENREF_32)*)*. The TLFB self-report questionnaire was used to assess alcohol consumption. Participants had to estimate the number of alcohol units consumed over the preceding 14 days (one UK unit =8g of alcohol).

*Alcohol Use Disorders Identification Test (AUDIT;* [*Saunders, Aasland, Babor, De la Fuente, & Grant, 1993*](#_ENREF_30)*)*. The AUDIT was used to assess hazardous drinking. The AUDIT consists of ten fixed-response questions regarding alcohol consumption and consequences of drinking. Scores on the AUDIT range between 0 -40 with scores >8 indicating hazardous or harmful alcohol use.

*Desires for Alcohol Questionnaire – brief version (DAQ; (*[*Love, James, & Willner, 1998*](#_ENREF_22)*).* The DAQ is a 14-item multidimensional state alcohol craving scale. Given the inconsistencies in the DAQ factor structure we analysed the mean scale score (α = .83).

*Subjective intoxication scale (SIS;* [*Duka, Tasker, & Stephens, 1998*](#_ENREF_9)*).* The SIS consisted of six 1-100mm VAS (strongly disagree to strongly agree) assessing subjective feelings of light-headed, irritable, stimulated, alert, relaxed and contented. All items were analysed separately.

*Alcohol Outcome Expectancies Scale (AOES;* [*Leigh & Stacy, 1993*](#_ENREF_21)*)* – The AOES was used to measure expectations following alcohol consumption. It consists of 34 items and has four positive outcome expectancy scales; social facilitation (α = .83), fun (α = .89), sex (α = .91), and tension reduction (α = .77) and four negative outcome expectancy scales; social (α = .84), emotional (α = .81), physical (α = .70), and cognitive performance (α = .79). Each item is scored on a 1-6 scale with 1 being no chance of happening and 6 being certain to happen.

***Go/No-Go***

*Passive avoidance Go/No-Go task* [*Newman & Kosson, 1986*](#_ENREF_26)*).* This Go/No-Go task was programmed in Inquisit version 1.33 (Millisecond software, 2002). The task requires participants to learn through trial and error which numerical stimuli are ‘correct’ (go cues) and which are ‘incorrect’ (no-go cues). Participants were instructed to withhold responses to the incorrect stimuli, but respond quickly to correct stimuli by pressing the spacebar on the keyboard (incorrect responses to No-Go cues resulted in “Wrong” appearing in red, correct responses with “Correct” in green). On each trial of the task, one of eight two-digit numbers was presented. Four numbers were go cues and four were no-go cues (different numbers were used in each session). Participants initially completed 8 practice trials, in which each number was presented once, followed by 72 experimental trials. Each of the eight numbers was presented nine times each in the main task. Number or No-Go errors, responding to incorrect numbers was taken as the dependent variable, (mean Go reaction times, time taken to respond to a correct cue, were also recorded).

**Procedure**

Testing sessions took place between 12pm and 5pm in the Department of Psychological Sciences. Participants were informed that the study was an investigation into the effects of alcohol on reaction time, to abstain from drinking alcohol before each session, and to avoid heavy drinking the night before. All participants provided a zero breath alcohol reading before each session (Lion Alcometer 500, Lion Laboratories, UK). Firstly participants completed a battery of questionnaires (demographics, TLFB, and AUDIT; first session only). They then completed the DAQ and SIS. Dependent on condition participants were then served either the placebo or control drink and were instructed to consume the drink within five minutes followed by a five minute “absorption period”. After this participants completed a further DAQ and SIS and provided a breath alcohol sample that the experimenter pretended to make a note of (all samples were .00 BAC). Participants then completed the Go/No-Go task before then estimating how many standard UK units of vodka (25ml) they believed were in the drink (‘Unit estimate’). Each testing session lasted approximately 30 minutes, and at the end of the second session informal debriefing indicated that no participants had guessed the aims of the experiment. Three participants believed that the placebo drink contained less than one unit of alcohol, removal of these participants left the pattern of results unaffected.

**Results**

*Participant characteristics (Table 1)*

Participants were heavy drinkers scoring, on average, above eight on the AUDIT. No difference between males and females were found for any drinking indices or AOE. Self report data were analysed using 2 x 2 within subjects ANOVA with factors of time (pre-drink, post-drink) and drink (placebo, control). Go/No-Go performance was analysed using a paired samples t-test. There was no effect of session order on any of the results reported (all main effects of, and interactions with, session order were non-significant ps>.1).

*Craving (Table 2)*

For DAQ scores there was no main effect of drink (F(1,31) =1.70, p=.20, ηp2 =.05). There was a main effect of time (F(1,31) = 11.80, p=.002, ηp2 =.28), although this was subsumed by the interaction (F(1,31) = 6.49, p=.016, ηp2 =.17). The interaction was the result of increased craving in the placebo *t*(31)=4.07, p<.001, *d* = 0.88, but not the control condition (p=.61).

*SIS- Light-headed (Table 2)*

There was no main effect of drink (F(1,31) =3.41, p=.07, ηp2 =.10). There was a main effect of time (F(1,31) = 6.16, p=.019, ηp2 =.17), again subsumed by the drink by time interaction (F(1,31) = 5.93, p=.021, ηp2 =.16). The interaction was the result of an increase in light-headed ratings in the placebo *t*(31)=2.65, p=.013, *d* = 0.49, but not the control condition *(*p=.95). As with previous studies from our lab (e.g. [Christiansen, et al., 2013](#_ENREF_4)) there were no effects of the placebo on the other SIS items, (data not reported but are available on request)

*Inhibitory control (Table 2)*

Due to a software malfunction data for one participant was missing from a placebo session. Participants made more No-Go errors in the placebo compared to the control condition *t*(29)=2.48, p=.019, *d* =0.49). There was no effect of condition on Go reaction times (p=.52).

*Correlations between outcome expectancies and craving, light-headedness and inhibitory control (Table 3).*

As predicted, following placebo-alcohol No-Go errors were positively correlated with expectations of impaired Cognitive Performance only (these variables were uncorrelated in the control condition). Craving change and light-headed change scores (post-drink – pre-drink scores) were calculated. In the placebo condition craving change was positively correlated with both positive and negative AOE (Social Facilitation, Tension reduction, Social Negative, Emotional Negative and Cognitive Performance). Light-headed change scores were only correlated with Sex. No correlations between change scores and AOE were found in the control condition.

**Discussion**

The current study investigated the effects of a placebo-alcohol prime on alcohol craving and inhibitory control, and the extent to which these effects were correlated with AOE. Results were supportive of our hypotheses; placebo-alcohol increased craving and caused significant impairments in inhibitory control. Impairments in inhibitory control were positively correlated with alcohol outcome expectancies of impaired cognitive performance. In addition, increases in craving were positively associated with both positive (Tension Reduction, Social Facilitation) and negative (Social, Emotional, Cognitive Performance) outcome expectancies.

Previous research has revealed that the anticipated effects of alcohol can affect performance on a range of cognitive and motor tasks ([Christiansen, et al., 2013](#_ENREF_4); [Fillmore & Vogel-Sprott, 1994](#_ENREF_14); [Finnigan, et al., 1995](#_ENREF_16); [Gilbertson, et al., 2010](#_ENREF_18)) although the current study is, to our knowledge, the first example of inhibitory control being impaired by placebo-alcohol. These findings are in contrast to studies that have reported compensation effects ([Fillmore & Vogel-Sprott, 1996](#_ENREF_11); [Fillmore, et al., 1994](#_ENREF_13)). This inconsistency is likely due to differences in methodologies and samples. Specifically, in the current study we assessed the general construct of expectancies of cognitive impairment rather than manipulating task-specific outcome expectancies ([Fillmore, et al., 1994](#_ENREF_13)). Although compensation was found without explicit manipulations of expectancies by [Fillmore & Vogel-Sprott, (1996](#_ENREF_11)) they used a sample of all male experienced drinkers whereas the current study was predominantly female. This indicates gender and drinking experience may moderate the anticipated effects of alcohol. Furthermore, the anticipated effects of alcohol also increased craving in the current study (see also [Rose, Hobbs, & Drummond, 2013](#_ENREF_20); [Christiansen, et al., 2012](#_ENREF_3)), and can prime *ad lib* consumption ([Marlatt, et al., 1973](#_ENREF_24)). Taken together, this is indicative of participants exhibiting conditioned responses to placebo-alcohol. We argue that this suggests much recent research into the acute effects of alcohol is therefore silent on what may be important anticipated effects. When designing alcohol priming studies researchers should consider including a no-expectancy, no-alcohol condition, to allow investigation of these effects.

Inhibitory control following placebo-alcohol was correlated with expectation of impaired cognitive performance but no other outcome expectancies (see also [Fillmore & Vogel-Sprott, 1994](#_ENREF_14)). Placebo-alcohol effects on cognition are therefore consistent with specific expectations of the effects of alcohol on cognition (see also Montgomery & Kirsch, 1997). Interestingly, increases in craving were associated with a broad range of AOE (positive and negative) which is suggestive of a general sensitivity to the effects of alcohol being critical to the effects of placebo-alcohol on craving.

The current study has limitations. The sample was predominantly undergraduate female students and therefore future research needs to determine whether these findings are also applicable to other populations. However, it is important to note that binge drinking (a likely outcome of alcohol priming) is common in the general population of young females and males (18 & 22% of 16-24 y/o; IAS 2013) and that the mean AUDIT score of our sample was well above the cut off for hazardous drinking. This suggests that our findings may apply to a more general group of young hazardous drinkers, although gender differences and drinking experience may be critical ([Fillmore & Vogel-Sprott, 1996](#_ENREF_11)). An additional limitation is that the passive avoidance Go/No-Go task is not a “pure” measure of inhibitory control as it also requires working memory and learning; therefore it is possible that placebo-alcohol is influencing these processes more than the inhibition component. Although inhibitory control and working memory are likely be determined by the same underlying processes (and share considerable common variance, [Miyake & Friedman, 2012](#_ENREF_25)), it is important that future research establishes whether this finding can be replicated in other related measures such as Stop-Signal and the cued Go/No-Go tasks.

In summary the current study has demonstrated that the anticipated effects of alcohol can significantly impair the behavioural substrates of self-control and influence craving. This has significant implications for the study of alcohol priming effects suggesting that the much-used alcohol vs. placebo comparisons will be underestimating alcohol priming effects in the real world (which are the product of anticipated plus pharmacological effects). The correlations between AOE and placebo-induced impairment and craving suggest that individual differences in AOE should be monitored in priming studies.

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Table 1: Participant characteristics (values are mean ± SD)

Sample Male (n = 11) Female (n = 21) *t*

Age 19.40 (±1.60) 19.27 (±0.09) 19.33 (±1.06) 0.16

TLFB 58.81 (±41.85) 57.82 (±34.97) 53.24 (±45.78) 0.29

AUDIT 13.69 (±4.95) 14.36 (±4.65) 13.33 (±5.22) 0.55

AOES Social Facilitation 5.44 (±0.70) 4.56 (±0.71) 4.38 (±6.70) 0.69

AOES Fun 3.86 (±0.58) 3.73 (±0.47) 3.94 (±0.62) 0.97

AOES Sex 3.49 (±0.96) 3.48 (±0.94) 3.50 (±0.99) 0.06 AOES Tension reduction 4.13 (±0.89) 4.24 (±0.94) 4.06 (±0.88) 0.53 AOES Social Negative 2.11 (±0.87) 2.06 (±0.80) 2.14 (±0.92) 0.25 AOES Emotional Negative 2.43 (±0.91) 2.24 (±0.78) 2.52 (±0.98) 0.83 AOES Physical Negative 3.48 (±1.07) 3.02 (±1.05) 3.73 (±1.02) 1.82 AOES Cognitive Performance 3.09 (±0.62) 2.85 (±0.58) 3.22 (±0.62) 1.62

TLFB = 14 day alcohol consumption in UK units, 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). No significant differences between groups found. AOE S= Alcohol outcome expectancy scale, scores range from 1 (minimum) to 6 (maximum).

Table 2: Descriptive statistics for craving, light-headed ratings, No-Go errors and unit estimate (values are mean ± SD)

Placebo Control

Pre-drink Post-drink Pre-drink Post-drink

DAQ 2.25 (±0.79) 3.02 (±1.21) 2.54 (±0.77) 2.60 (±0.64)

Light-headed 11.56 (±17.37) 18.56 (±22.33) 9.00 (±13.15) 8.94 (±10.80)

No-Go Errors - 10.33 (±5.92) - 7.47 (±3.86)

Unit Estimate - 1.20 (±0.72) - 0.00 (±0.00)

DAQ = Mean scores on desires for alcohol questionnaire, score range from 1(minimum) to 7(maximum). Subjective intoxication light-headed item scores, scores range from 0 (minimum) to 100 (maximum). No-Go Errors = Number of incorrect responses to No-Go cues scores range from 0 (minimum ) to 36 (maximum). Unit est = number of 25ml vodka measures (1 measure = 8g of alcohol, one UK unit) participants believed to be in the priming drink.

Table 3: Pearson’s correlations between post-placebo inhibitory control, changes in craving and light-headedness, and alcohol outcome expectancies

No Go Errors DAQ craving change Light-headed change

AOES Social Facilitation -.17 .34\* -.02

AOES Fun .25 .24 .09

AOES Sex .13 -.07 .46\*\*

AOES Tension reduction -.19 .37\* -.06

AOES Social Negative .02 .43\*\* .12

AOES Emotional Negative .13 .43\*\* .20

AOES Physical Negative .26 .08 .14

AOES Cognitive Performance .31\* .48\*\* .16

AUDIT scores .23 .35\* -.11

14 day alcohol consumption -.17 -.04 -.24

\*p<.05, \*\*p<.01