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What is This?
A comparison of the anticipated and pharmacological effects of alcohol on
cognitive bias, executive function, craving and ad-lib drinking

Paul Christiansen, Abigail K Rose, Jon C Cole and Matt Field

Abstract

Acute alcohol administration alters automatic processing of alcohol-related cues, impairs executive functions and increases alcohol seeking. Few studies have investigated the effects of expecting to receive alcohol on these measures. Thirty-one social drinkers completed three experimental sessions receiving either 0.65 g/kg alcohol, a placebo and a control beverage (which they knew was not alcoholic) before reporting craving and completing a test battery including a measure of automatic alcohol-approach tendencies (stimulus response compatibility task), a measure of executive function (Controlled Oral Word Association Task (COWAT)) and a taste test assessing ad-lib drinking. Results indicated that alcohol administration impaired performance on the COWAT and increased ad-lib drinking; however, there were no significant differences on these measures after administration of placebo versus control beverages. Craving was increased after alcohol and (to a lesser extent) after placebo. Automatic alcohol-approach tendencies were pronounced after both alcohol and placebo compared to the control beverage, with no difference between alcohol and placebo. Results suggest craving is sensitive to the anticipated and pharmacological effects of alcohol, alcohol-approach tendencies are particularly sensitive to the anticipated effects of alcohol, and measures of executive function and ad-lib drinking are affected by the pharmacological, but not the anticipated, effects of alcohol.

Keywords

Alcohol, automatic approach tendencies, executive cognitive function, priming, expectancies, placebo

Introduction

Administration of alcohol affects a broad range of cognitive functions, for example, it alters the automatic processing of alcohol-related cues and impairs executive function. In a recent review, Field et al. (2010) argued that these cognitive responses to alcohol may be associated with the alcohol ‘priming’ effect, whereby alcohol craving and drinking behaviour are increased after consumption of an initial dose of alcohol (de Wit, 1996). However, previous research into the acute effects of alcohol on cognitive functions has focussed almost exclusively on the pharmacological effects of alcohol, with minimal research exploring the anticipated effects of alcohol. In the present study, our aim was to contrast the pharmacological and anticipated effects of alcohol on the automatic processing of alcohol cues, executive function, craving and alcohol-seeking behaviour.

Executive cognitive function refers to a set of inter-connected abilities which subserve the ability to inhibit pre-potent responses, hold information in working memory and switch between different mental sets (Miyake et al., 2000). Each of these aspects of executive cognitive functioning is impaired by alcohol consumption (Marczinski et al., 2005; de Wit et al., 2000; Grattan-Miscio and Vogel-Sprott, 2005; Guillot et al., 2010), as is performance on tasks that assess a cluster of executive cognitive functions, such as phonemic fluency tasks (e.g. Peterson et al., 1990; see Fillmore, 2007 and Field et al., 2010, for reviews). A separate body of evidence reveals that chronic alcohol use is associated with increased allocation of attention towards alcohol-related stimuli (‘attentional bias’) and automatic approach responses elicited by alcohol-related cues (Field and Cox 2008; Field et al., 2009; Roefs et al., 2011; Rooke et al., 2008; Stacy and Wiers, 2010). Specific doses of alcohol (0.3–0.4 g/kg) increase attentional bias for alcohol-related cues (Adams et al., 2012; Duka and Townshend, 2004; Fernie et al., 2012; Schoenmakers et al., 2008), but automatic alcohol approach responses are unaffected (Fernie et al., 2012; Schoenmakers et al., 2008). Finally, ‘priming’ doses of alcohol increase subjective craving and voluntary drinking behaviour in social drinkers (Chutuape et al., 1994; de Wit and Chutuape, 1993), and alcohol-dependent individuals (Hodgson et al., 1979; Ludwig et al., 1974).

Despite these findings, there is a degree of ambiguity in the literature. For example, there have been numerous failures to replicate the acute effects of alcohol on some components of executive cognitive function (e.g. Rose and Duka, 2007; Weissenborn and Duka, 2003). Likewise, some studies failed to replicate the effects of acute alcohol on attentional bias (Miller and Fillmore, 2011), particularly at higher doses (Duka and...
Hull and Bond (1986) reported that the anticipated effects of meta-analysis of studies utilising the balanced placebo design, characterise responses to the anticipated effects of alcohol. In their effects in naturalistic settings. Alcohol and placebo are unlikely to be representative of alcohol related behaviours such as binge drinking, comparisons between anticipated effects of the drug. In this sense, when seeking to as responses to alcohol in naturalistic settings (e.g. Friday night in alcohol with a control beverage rather than a placebo would offer a not just a practical issue. Arguably, comparing the effects of slower after the control beverage and slowest after alcohol. This is of alcohol. If the placebo response to alcohol does in fact mimic the pharmacological effects of alcohol (Stewart et al., 1984), and therefore reaction time would be slow after administration of both alcohol and placebo; if these expectancy effects are large but the actual pharmacological effects of alcohol are relatively small, there would be no difference between alcohol and placebo and researchers would conclude that this specific dose of alcohol did not influence choice RT. Alternatively, the response to a placebo may be a form of compensatory response, which runs counter to (opposes) the pharmacological effects of alcohol (Siegel 1999, 2005). Again, if this compensatory response is large in comparison to a (relatively small) pharmacological effect of alcohol, there would be no difference between alcohol and placebo, and researchers would inappropriately conclude that there is no difference between alcohol and placebo.

If a study were to administer alcohol and a placebo and contrast those with a third beverage which participants knew to be pharmacologically inactive, this would help to clarify and distinguish between the pharmacological versus anticipated effects of alcohol. If the placebo response to alcohol does in fact mimic the pharmacological effect, choice RT would be slower after both alcohol and placebo, compared to after the control beverage. If the placebo response to alcohol counteracts the pharmacological effect (RT slowing), choice RT would be fastest after placebo, slightly slower after the control beverage and slowest after alcohol. This is not just a practical issue. Arguably, comparing the effects of alcohol with a control beverage rather than a placebo would offer a more ecologically valid assessment of the acute effects of alcohol, as responses to alcohol in naturalistic settings (e.g. Friday night in a bar) will inevitably reflect the combined pharmacological and anticipated effects of the drug. In this sense, when seeking to understand the consequences of alcohol intoxication for health-related behaviours such as binge drinking, comparisons between alcohol and placebo are unlikely to be representative of alcohol effects in naturalistic settings.

A relatively small number of studies have attempted to characterise responses to the anticipated effects of alcohol. In their meta-analysis of studies utilising the balanced placebo design, Hull and Bond (1986) reported that the anticipated effects of alcohol did not consistently influence cognitive, non-social processes such as memory, but they exerted a clear influence on social behaviours such as sexual arousal and aggression. More recently, a series of studies by Fillmore and colleagues found evidence for anticipated effects of alcohol on complex motor skill tasks. Fillmore and Vogel-Sprott (1995) found that performance on a pursuit rotor task was impaired among participants who expected to experience behavioural impairment, compared to those who did not expect impairment, regardless of whether alcohol or placebo were actually administered. Fillmore et al. (1994) administered a placebo to participants and informed them that it would either improve or impair performance on a pursuit rotor task; participants who were informed that psychomotor skill would be impaired performed significantly better than those expecting improvement. The latter study suggests that compensatory responses can be facilitated by a placebo, but this is dependent on participants’ expectations. There is also evidence that responses to a placebo may be dependent upon drinking history. Fillmore and Vogel-Sprott (1996) reported that experienced drinkers had improved performance on a pursuit rotor task following a placebo, whereas novice drinkers did not show this compensatory response. In addition, acute alcohol led to impaired performance on the task, but experienced drinkers showed tolerance to this effect.

Although there is evidence for both drug-like and compensatory responses to placebo on tasks that assess psychomotor function, no previous studies have investigated the anticipated effects of alcohol on automatic cognitive processing of alcohol cues or the aspects of executive cognitive function that are linked to loss of control over drinking (see Field et al., 2010). In addition, recent studies into the alcohol priming effect have also neglected to investigate the anticipated effects of alcohol on craving and ad-lib alcohol consumption. There is, however, evidence that the anticipated effects of alcohol have an important role in the alcohol priming effect. For example, the meta-analysis reported by Hull and Bond (1986) found that increased desire for alcohol was the result of the anticipated rather than the pharmacological effects of alcohol. To give an example of a specific study, Marlatt et al. (1973) demonstrated that if participants were informed that drinks contained alcohol, both a priming dose of alcohol and a placebo increased voluntary alcohol consumption in a subsequent taste test, compared to if participants were informed that drinks did not contain alcohol. These effects were seen in current alcoholics and social drinkers. Although they did not use a balanced placebo design, Leeman et al. (2009) found that alcohol craving following the administration of a placebo (but not alcohol) was associated with increased ad-lib alcohol consumption. This suggests that the anticipated effects of alcohol on ad-lib drinking may be mediated by increases in craving.

In the present study, we investigated the pharmacological and anticipated effects of alcohol, alone and in combination, by contrasting the effects of alcohol (0.65 g/kg), placebo and a control beverage which participants knew contained no alcohol. After drink administration, participants completed a battery of tasks including measures of executive cognitive function (phonemic fluency), automatic alcohol approach tendencies, alcohol craving and overt alcohol-seeking behaviour. Our experimental design permitted us to contrast responses to alcohol and placebo beverages to give a relatively pure measure of alcohol’s pharmacological effects and to contrast the placebo and the control beverage in order to examine the anticipated effects of...
alcohol, uncontaminated by its pharmacological effects. We hypothesised that alcohol would strengthen automatic alcohol approach tendencies, impair executive function, increase self-reported craving and increase ad-lib beer consumption compared to placebo. We also hypothesised that the anticipated effects of alcohol would have drug-like effects on automatic approach tendencies (as exposure to alcohol-related cues strengthens automatic cognitive processes (Lindgren et al., 2009; Schulze and Jones, 1999), self-reported craving and beer consumption, but have compensatory (drug-opposite) effects on phonemic fluency, as participants would compensate for anticipated impairment.

Method

Participants

Thirty-one participants (19 female) aged between 18 and 40 years (mean 21.03 ± 4.11) were recruited from the University of Liverpool via Intranet advertisements. Inclusion criteria were fluency in English and normal or corrected to normal vision. Participants were invited to take part if they self-reported consuming at least 15 units of alcohol (females) or 22 units (males) each week, i.e. in excess of UK government guidelines for safe drinking (Edwards, 1996). Exclusion criteria included current or past self-reported alcohol use disorder, current or recent illness which may increase sensitivity to alcohol (e.g. colds and flu), taking medication that is contraindicated for alcohol (e.g. antidepressants, anxiolytics) and aversion or allergy to any of the drink constituents (vodka, tonic water, or Tabasco sauce). Additional exclusion criteria for female participants included current breastfeeding or pregnancy; the latter was confirmed with a pregnancy test at the beginning of the first session. All participants provided informed consent before taking part in the study, which was approved by the University of Liverpool Research Ethics Committee. Participants received either course credits or £30 as compensation for their travel expenses and time.

Design

The study utilised a within-subjects partially balanced placebo design. After a familiarisation session, participants attended the laboratory for three sessions, with an interval of at least two days between sessions. During the three sessions, participants consumed an alcoholic drink (they were informed that it contained alcohol), a placebo drink (again, they were informed that it contained alcohol) and a control drink (they were informed that the drink was non-alcoholic). A fully balanced placebo design (which would include a fourth session in which alcohol is administered but participants expect to receive a non-alcoholic drink) was not used because the pharmacological effects of 0.65 g/kg alcohol render the deception ineffective (Sayette et al., 1994). Our design enabled us to contrast the combined anticipated and pharmacological effects of alcohol (by contrasting alcohol vs. control), the pharmacological effects of alcohol in addition to its anticipated effects (by contrasting alcohol vs. placebo) and the purely anticipated effects of alcohol (by contrasting placebo vs. control). The alcohol and placebo drink were administered double blind but the control drink was not as it was integral to the study design that the participants knew that the control drink did not contain any alcohol. Session order was counterbalanced across participants.

Materials

Drink preparation

The alcoholic drink contained vodka (Smirnoff Red, 37.5% alcohol by volume (ABV)); the dose was calculated as 0.65 g of pure alcohol per kg of body weight, up to a maximum of 200 mL of vodka. The drink was mixed with chilled tonic water in the ratio one part vodka to three parts tonic. The placebo drink consisted of chilled tonic water only (identical total volume to the alcoholic drink). For both alcoholic and placebo drinks, a few drops of Tabasco sauce were added, vodka was smeared on the rim of the glass, and an atomiser was used to spray vodka mist on the surface of the drink. The control drink consisted of chilled water only, in the same total volume as alcoholic and placebo drinks.

Questionnaires

Time Line Follow Back (TLFB; Sobell and Sobell, 1990). The TLFB self-report questionnaire was used to assess weekly alcohol consumption. Participants had to estimate the number of alcohol units consumed over the preceding seven days.

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993). The AUDIT was used to assess hazardous drinking. The AUDIT consists of 10 fixed response questions regarding alcohol consumption and consequences of drinking. Scores on the AUDIT range between 0 and 40 with scores of 8 or above indicating hazardous or harmful alcohol use.

Desire for Alcohol Questionnaire – brief version (DAQ; Love et al., 1998). The DAQ is a 14-item multidimensional alcohol craving scale that yields scores on four different factors of craving: Positive and Negative Reinforcement, Strong Desires and Intentions, Mild Desires and Intentions, and Perceived Control Over Drinking. Scores on each factor range from 1 to 7, with higher scores indicative of higher craving. We report the average score across all four subscales.

Subjective intoxication scales (SIS; Duka et al., 1998). The SIS consisted of six 100 mm visual analogue scales which assessed subjective feelings of ‘light headed’, ‘irritable’, ‘stimulated’, ‘alert’, ‘relaxed’ and ‘contented’.

Pictorial stimuli

The stimulus response compatibility task (SRC) used a picture set containing 14 alcohol-related pictures and 14 (matched) alcohol-unrelated pictures. Alcohol pictures consisted of alcohol-related scenes (such as a bottle and a glass of wine presented on a table); the alcohol-unrelated pictures were matched to the alcohol pictures on perceptual characteristics but did not contain any alcohol-related cues (e.g. a bottle and a glass of water presented on a table). All the pictures were 100 mm high × 125 mm wide. The picture set was identical to that used by Field and Eastwood (2005) and Field et al. (2008).
Cognitive tasks

SRC task (Field et al., 2008). The SRC task is a measure of automatic approach tendencies elicited by alcohol-related cues. The task was programmed in Inquisit version 1.33 (Millisecond software, 2002). Each trial of the task commenced with the presentation of either an alcohol-related picture or an alcohol-unrelated (control) picture in the centre of the screen along with a small manikin above or below the picture. Participants were instructed to move the manikin either toward or away from the picture by pressing up or down on a two-button response box according to the task instructions (see below). If participants made the appropriate response, the manikin moved towards or away from the picture. If they made an inappropriate response (e.g. pushing the ‘up’ button when a ‘down’ response was required), a large red cross was presented in the centre of the screen for 1000 ms.

There were 128 trials of the task in total, split into two blocks of 64 trials. In the ‘approach alcohol’ block, participants were instructed to move the manikin towards alcohol-related pictures, and away from alcohol-unrelated pictures. These instructions were reversed in the ‘avoid alcohol’ block. Each block began with eight practice trials in which four alcohol-related and four alcohol-unrelated pictures were presented. After the practice trials, the instructions were then reiterated before participants completed 56 experimental trials. During these trials, the 14 alcohol-related and 14 alcohol-unrelated pictures were each presented twice, once with the manikin above the picture and once with the manikin below the picture. Trials were presented in random order. The order of completion of ‘approach alcohol’ and ‘avoid alcohol’ blocks was counter-balanced across participants. RTs (the time taken to initiate movement of the manikin) was measured on each trial and the dependent variables were mean RT during experimental trials of the ‘approach alcohol’ and ‘avoid alcohol’ blocks. Before analysis of RTs, outliers were removed in order to eliminate RTs that were too fast (indicative of pre-emptive responding) or too slow (indicative of poor concentration) according to criteria used in previous reports (e.g. Field et al., 2008). RTs less than 200 ms, greater than 2000 ms, and then those RTs that were more than three standard deviations above the individual mean were discarded. RTs from error trials were also discarded. Fewer than 5% of trials were discarded as errors or outliers.

Controlled Oral Word Association Test (COWAT; Benton 1968). The COWAT was used to assess phonemic fluency, which taps into multiple components of executive functioning. Participants were given a letter and instructed that they had one minute to verbally state as many words beginning with that letter as possible (excluding proper nouns and identical words with a different suffix). A voice recorder (Sony IC-B600) was used to record responses for future analysis. To reduce practice effects, participants were given different letters/letter combinations in each session (F, A and S; P, L and W; C, F and L), the order of which was counterbalanced across conditions. These letter combinations were found to produce a similar number of words in previous studies (Ross et al., 2007). The dependent measure from the COWAT was the total number of switches between word clusters (with a greater number of switches indicative of good executive cognitive functioning). Word clusters were defined as consecutive words which began with the same two letters, differed by a vowel, homonyms or rhyming words (Troyer et al., 1997). This method for assessing switches was found to best reflect frontal functioning in phonemic fluency as well as having high test-retest reliability (Ross et al., 2007; Troyer et al., 1998).

Taste test

The taste test was based on that used by Fernie et al. (2012). Participants were given a 275 ml bottle of Becks non-alcoholic beer and a 275 ml bottle of Orange and Passion Fruit J20 (a non-alcoholic beverage). The labels from both bottles were removed and participants were not informed that the beer was non-alcoholic. We elected to use non-alcoholic beer because of ethical concerns about administering too much alcohol to participants; furthermore, previous studies from our laboratory have shown that participants do not detect that this brand of beer is non-alcoholic, and it has been used in previous taste test procedures which were sensitive to experimental manipulations of the motivation to drink (e.g. Jones et al., 2011). Participants were asked to taste the two drinks and rate them on four continua (unpleasant-pleasant, tasteless-strong tasting, bitter-sweet, flat-gassy) using 100 mm visual analogue scales. Participants were informed that they were allowed to drink as much of each drink as they wished in order to make accurate ratings. At the end of the session, the volume of each drink consumed was recorded. Informal debriefing indicated that none of the participants were aware that the beer was non-alcoholic.

Procedure

Testing sessions took place between 12 pm and 6 pm in a laboratory in the School of Psychology. Participants were asked to consume a high-carbohydrate, low-fat meal the night before and a light meal (e.g. a sandwich) an hour before each experimental session. Participants were also asked to avoid drinking alcoholic drinks before each session, and to avoid heavy drinking the night before each session. All participants provided a zero breath alcohol reading before each session (Lion Alcometer 500, Lion Laboratories, Barry, UK). Participants initially attended the laboratory for a familiarisation session (cf. Weafer and Fillmore, 2008) in which they completed a questionnaire battery (demographics, timeline followback and AUDIT) before the cognitive test battery. These data are not reported here as the purpose of the familiarisation session was simply to enable participants to complete the tasks while sober.

During experimental sessions, participants initially provided a breath sample then immediately completed the DAQ and SIS. Drinks were then administered (alcohol and placebo administered double blind, control unblinded), and participants were instructed to consume the drink within 10 minutes, before a 10-minute absorption period in which participants were provided with magazines to read. This method of alcohol administration yields a peak blood alcohol content (BAC) approximately 65 minutes after consumption of 0.65 g/kg alcohol, therefore all cognitive tasks were completed during the ascending limb of the blood alcohol curve (Fillmore and Vogel-Sprott, 1998).

After the absorption period, participants completed further DAQ and SIS and provided a breath alcohol sample for a second experimenter. Participants then completed a battery of cognitive tasks, including the SRC task and COWAT as described above.
Participants also completed a Cued Go/No-Go task (Weafer and Fillmore, 2008), a delay discounting task (Du et al., 2002) and a visual probe task with eye movement monitoring (Schoenmakers et al., 2008). Results from these latter tasks are not reported here as they were not significantly affected by alcohol or placebo administration (details are available from the authors on request).

The entire cognitive test battery took approximately 40 minutes to complete. Upon completion of the tasks, participants completed an additional DAQ and SIS and provided a final breath alcohol sample before completing a short questionnaire in which they estimated the number of pub measures of vodka (one measure = 25mL vodka) that were in the drink. Finally, participants completed the taste test procedure.

Participants were advised to remain in the laboratory until their BAC had declined to 0.39g/100mL (approximately half the UK drive limit). At the end of the final session, participants were fully debriefed before being discharged.

Results

Sample characteristics

The mean age of the sample was 21.03 (±4.11) years. With regard to drinking behaviour, the mean number of units (1 unit = 8g alcohol) consumed in the week prior to enrolling in the study was 39.00 (±17.29), with males (46.95 ± 16.43) consuming more units than females (33.97 ± 16.25). Mean AUDIT scores were above the cut-off for hazardous drinking (8+) for the sample as a whole (16.06 ± 5.32), and for males (17.33 ± 5.31) and females (15.26 ± 5.31).

Perceived alcohol content, breath alcohol concentration, subjective intoxication and craving

Perceived alcohol content. A three-way repeated measures analysis of variance (ANOVA) was used to assess the estimated number of alcohol units in the drinks consumed (alcohol, placebo and control). A significant main effect of session was found (F(2,60) = 229.08, p < .001, ηp² = .88). Participants estimated that they consumed more alcohol in the alcohol session (5.23) compared to the placebo session (2.26; t(30) = 10.49, p < .001) and placebo session compared to the control session (0.00; t(30) = 10.45, p < .001).

Breath alcohol concentration. All participants had a BAC of 0g/100mL when assessed at the beginning of all sessions. In the placebo and control sessions all other BAC readings were 0mg%.

In the alcohol session, the mean BAC was 0.89 g/100mL (±0.16) at the post-drink assessment and this increased to 0.96 g/100mL (±0.11) at the end of the session (although this increase was not statistically significant, t(30) = 1.53, p > .1). There were no gender differences in BAC post-drink (t(29) = -0.30, p > .1), or at the end of the session (t(29) = -0.57, p > .1).

Subjective Intoxication Scales (SIS). Self-report ratings for ‘lightheaded’ and ‘contented’ increased following administration of both placebo and alcohol, although increases were larger after alcohol compared to placebo. Ratings of ‘alert’ decreased following both placebo and alcohol, with no difference between the two. There were no changes in any of the SIS scales following administration of the control drink, and there were no changes in ratings of ‘irritability’, ‘relaxed’ or ‘stimulated’ following any of the drinks. Descriptive and inferential statistics are not shown, but are available on request.

Subjective alcohol craving. A 3 × 3 repeated measures ANOVA was used to check differences in mean DAQ scores between sessions (alcohol, placebo and control) at the three time points within test sessions (pre-drink, post-drink and end of session) (see Figure 1).

There was a significant session by time interaction (F(4,120) = 8.13, p < .0001, ηp² = .21). After alcohol, post hoc comparisons revealed a significant increase in craving between pre-drink and post-drink (t(30) = -3.74, p < .001) and between pre-drink and end of session (t(30) = -4.61, p < .001), although post-drink and end of session did not differ from each other (p > .1). For placebo, craving increased between pre-drink and post-drink (t(30) = -2.30, p < .05), but craving then declined from post-drink to the end of session (t(30) = 2.09, p < .05); pre-drink and end of session did not differ (p > .1). There were no changes in craving after administration of the control drink (p > .1). Contrasts between different drinks revealed that craving was significantly higher post-drink after alcohol compared to placebo (t(30) = 2.63, p < .025) and after placebo compared to the control drink (t(30) = 2.73, p < .01). End of session craving was also significantly higher after alcohol compared to placebo (t(30) = 5.88, p < .001) and after placebo compared to the control drink (t(30) = 2.42, p < .05).

Cognitive tasks

Automatic alcohol approach tendencies. A repeated measures ANOVA was used to analyse reaction times, with session (alcohol, placebo, control) and task block (approach alcohol, avoid alcohol) as within subjects factors (see Figure 2).

The interaction between session and block was significant (F(2,60) = 3.59, p < .05, ηp² = .11). Planned comparisons revealed that reaction times in the approach alcohol block were faster than
reaction times in the avoid alcohol block after alcohol and placebo drinks (alcohol; $t(30) = -3.59, p < .001$; placebo; $t(30) = -3.89, p < .001$); there was also a trend in this direction after the control drink ($t(30) = -1.66, p < .06$). To investigate the interaction, the strength of automatic alcohol approach tendencies was calculated separately for each session by subtracting reaction times during the approach alcohol block from reaction times during the avoid alcohol block, such that higher scores indicate speeded approach elicited by alcohol-related cues. Paired samples t-tests revealed that automatic alcohol approach tendencies were greater after both alcohol ($t(30) = 1.89, p < .05$) and placebo drink ($t(30) = 2.48, p < .001$), compared to the control drink. However, there was no difference between alcohol and placebo ($t(30) = -0.66, p > .1$).

**Phonemic fluency.** A repeated measures ANOVA was used to analyse the number of switches between word clusters, with session (alcohol, placebo, control) as the within subjects factor (see Figure 3).

There was a significant main effect of session ($F(2,60) = 14.41, p < .001, \eta_p^2 = .32$), as participants made significantly fewer switches after alcohol compared to both placebo ($t(30) = -3.74, p < .001$) and control drinks ($t(30) = -5.42, p < .001$), which is indicative of impaired executive functioning after alcohol. There was no significant difference between placebo and control ($t(30) = -1.33, p > .1$).

**Beer consumption during taste test.** A repeated measures ANOVA was used to analyse beer consumed (as a percentage of total fluid), with session (alcohol, placebo, control) as the within subjects factor (see Figure 4).

There was a significant main effect of session ($F(2,60) = 12.62, p < .001, \eta_p^2 = 0.30$). Participants consumed significantly more beer after alcohol compared to both placebo ($t(30) = 4.46, p < .001$) and control drinks ($t(30) = 3.95, p < .001$). However, the amount of beer consumed did not differ between placebo and control ($t(30) = 1.26, p > .1$).

**Discussion**

In the current study we contrasted the effects of 0.65 g/kg alcohol with placebo and control drinks on automatic approach tendencies elicited by alcohol-related stimuli, executive cognitive functioning, self-reported craving and ad-lib beer consumption. Results indicated that automatic approach tendencies elicited by alcohol-related cues were strengthened after placebo compared to the control drink, but alcohol did not significantly increase approach tendencies beyond the increase seen under placebo. In contrast, alcohol significantly impaired executive functioning and increased beer consumption, relative to both placebo and control drinks, but placebo and control drinks did not differ from each other. Finally, self-reported craving was elevated after both alcohol and placebo administration, with more pronounced increases after alcohol. These results suggest that the pharmacological and anticipated effects of alcohol have differential effects on automatic alcohol approach tendencies and...
executive cognitive functioning as well as self-report versus behavioural measures of the motivation to drink alcohol.

Automatic approach tendencies elicited by alcohol-related cues (as assessed with the alcohol SRC task) were significantly larger after administration of alcohol and placebo compared to the control drink, although alcohol and placebo did not differ from each other. This finding may explain the absence of an effect of alcohol (versus placebo) on the SRC task in earlier studies (Fernie et al., 2012; Schoenmakers et al., 2008), in which a control drink was not included. We also note results from a study described by Farris and Ostafin (2008), who found that acute alcohol increased the strength of automatic alcohol approach associations, although those authors used a different task from the one used in the present study. Farris and Ostafin (2008) did not include a placebo control condition, so it is possible that their results reflect the anticipated rather than pharmacological effects of alcohol. Taken together, it seems that the anticipated effects of alcohol increase the strength of automatic approach tendencies elicited by alcohol-related cues, but the pharmacological effects of alcohol do not appear to contribute to these effects. We recommend that researchers who investigate the effects of alcohol on implicit alcohol cognitions should compare alcohol with both placebo and control beverages in an attempt to replicate and extend these findings.

Phonemic fluency, a measure of executive cognitive functioning that was assessed with the COWAT, was significantly impaired by alcohol in the present study. This is consistent with a large body of previous research demonstrating that multiple components of executive cognitive functioning, such as inhibitory control (Marczinski et al., 2005, 2007), working memory (Balodis et al., 2007) and mental set shifting (Guilford et al., 2010) are impaired by acute alcohol administration. Contrary to our hypotheses, we observed no difference in phonemic fluency after placebo and control drinks. This suggests that the effects of alcohol on phonemic fluency reflect the pharmacological rather than the anticipated effects of alcohol. Previous research has shown that if participants are explicitly instructed about the detrimental effects of acute alcohol administration on psychomotor function, then they compensate for these anticipated effects (Fillmore et al., 1994), and these compensatory responses reduce the magnitude of the pharmacological effects of alcohol (Fillmore and Vogel-Sprot, 1996). However, we did not see evidence of a drug-opposite response to the placebo, which we would expect if participants put in increased effort to compensate for anticipated alcohol-induced impairment. This indicates that without explicit instructions regarding the likely detrimental effects of alcohol on task performance, participants will not attempt to compensate for any expected impairments. Regardless, because executive cognitive function has been implicated in the loss of control over drinking (Field et al., 2010), this suggests that participants do not compensate for the effects of alcohol on the aspects of cognitive functioning that are required to control drinking.

In line with other research (e.g. Rose and Grunsell, 2008; de Wit and Chutuape, 1993) we found that alcohol administration significantly increased self-reported craving and alcohol-seeking behaviour, as measured by the taste test. This suggests the pharmacological effects rather than anticipated effects of alcohol are primarily responsible for alcohol ‘priming’ effects on the motivation to drink. However, we also found that the placebo produced a significant increase in alcohol craving, although this increase was small and transient compared to the increase seen after alcohol administration. Previous studies have generally shown small but non-significant increases in craving following placebo administration (e.g. Rose and Duka, 2006; Schoenmakers et al., 2008). In contrast to Marlatt et al.’s (1973) seminal study, we found no evidence that increased voluntary beer consumption was the result of the anticipated, rather than pharmacological, effects of alcohol. One explanation for this is that the alcohol-like effect of the placebo on self-reported craving in the current study was relatively short lived. In Marlatt et al. (1973) participants completed the taste test immediately after the placebo; in the current study participants completed the battery of cognitive tasks before completing the taste test, and at this point the placebo-induced increases in craving had dissipated.

In the current study we also assessed attentional bias, delay discounting and inhibitory control (using a cued Go/No-Go task). We found no effects of alcohol or placebo on any of these measures. Regarding attentional bias, the lack of an alcohol priming effect replicates the findings of Duka and Townsend (2004) and Miller and Fillmore (2011), who also reported no effect of 0.6–0.65 g/kg of alcohol on attentional bias, compared to placebo. Likewise, impulsive decision making, as assessed by non-experiential delay discounting, seems largely unaffected by priming doses of alcohol (e.g. Reynolds et al., 2006). The lack of a priming effect on the Cued Go/No-Go task was unexpected. A substantial body of evidence shows that moderate priming doses of alcohol cause impairment on this task (e.g. Marczinski et al., 2005). It is unclear why we failed to replicate these effects in the present study.

In summary, we demonstrated that 0.65 g/kg of alcohol led to impairments in executive cognitive functioning and increased voluntary beer consumption, but neither of these variables were affected by placebo administration, suggesting that these effects reflect the pharmacological rather than the anticipated effects of alcohol. Self-reported alcohol craving was increased following both placebo and alcohol, although the increase following placebo was short lived and smaller in magnitude than that seen following alcohol. We also found that both alcohol and placebo increased automatic alcohol approach tendencies, with no difference between alcohol and placebo, suggesting that this effect reflects the anticipated rather than pharmacological effects of alcohol. Future studies which investigate the effects of alcohol on cognitive performance and the motivation to drink should contrast the effects of alcohol with both placebo and control drinks, so that these potentially important expectancy effects do not go unnoticed.

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Conflicts of interest
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