THE INFLUENCE OF ALCOHOL ON BASIC MOTORIC AND COGNITIVE DISINHIBITION

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Abstract — It has been proposed that alcohol weakens control processes, which in turn supports the occurrence of disinhibited behaviours. Two studies were run, in parallel (both with 32 participants) using a between-subject design to investigate any disinhibiting effects of a moderate dose of alcohol (0.6 g/kg compared to placebo), previously found to trigger increased desire for alcohol. Disinhibiting effects were tested on basic motoric and cognitive control processes, using a go/no-go (GNG) and the Stroop task (ST) respectively. Although a higher proportion of participants wanted more alcohol under the alcohol preload (priming effect), this effect was not found to be significant. In the GNG task, correct response latency (RL) decreased from baseline \[P = 0.008\] while number of incorrect hits increased \[P = 0.030\] irrespective of treatment, indicating the formation of a habit-like response and motoric disinhibition. Although error rate did not differ between groups, an interaction occurred with regard to erroneous RL: participants under alcohol became quicker, while those under placebo became slower \[P = 0.014\]. In the ST, those preloaded with alcohol made significantly more errors \[P = 0.021\] and were quicker to complete the task \[P = 0.044\] compared with those preloaded with placebo, indicating a strong alcohol effect on cognitive disinhibition. The data suggest that a moderate dose of alcohol, which induces priming to want more alcohol, had disinhibiting effects both on a basic motoric and a cognitive inhibitory task. Thus the idea that priming may be mediated by the disinhibitory effects of alcohol is supported.

INTRODUCTION

Although Jellinek’s extreme view that loss of control can result from consumption of a single drink has lost popularity, a role for weakened self-control in addiction is still acknowledged. Previous research has indicated that alcohol supports behaviours that are normally under inhibitory control. For example, alcohol can stimulate eating in both restrained and non-restrained eaters (Polivy and Herman, 1976; Westenhoefer et al., 1994), and smoking increases during alcohol consumption (Mello et al., 1987). Continued use of a drug has also been explained via control processes: the drug—rather than the individual—comes to exert power over behavioural output, resulting in compulsive, disinhibited drug administration (Fillmore, 2003).

Disinhibition has been separated into behavioural and cognitive subtypes (Bechara et al., 1994). Johansson and Hansen (2000) have suggested that prefrontal areas may be more associated with cognitive and ventral striatal areas more with behavioural disinhibition. Motoric disinhibition may be understood in terms of a lack of control over simple habit-like behaviours that require little or only basic cognitive processing. During a go/no-go (GNG) task, participants under alcohol showed lateralized readiness potentials (a measure of brain activity associated with the preparation for action) to no-go stimuli within periods of time too short for any meaningful cognitive processing (Marinkovic et al., 2000). Such a finding suggests that alcohol can induce motoric disinhibition. Cognitive disinhibition illustrates weakening effects of alcohol of more complex, executive function systems (Finn et al., 1999).

To examine further the disinhibiting effects of alcohol two studies were run, in parallel, separating the motoric and cognitive control processes, employing a moderate dose of alcohol, previously found to induce priming in social drinkers (Rose and Duka, 2006). In addition, the two studies also included an assessment of the priming effect, on desire for alcohol, following the alcohol dose.

A popular paradigm used to test inhibitory processes is the GNG task, whereby participants must respond to certain stimuli while withholding responses to other stimuli. Although there are many types of GNG that have shown an effect of alcohol (Newman and Kosson, 1986; Helmers et al., 1995; Finn et al., 1999; LeMarquand et al., 1999; Marczynski and Fillmore, 2003), the experiment on motoric disinhibition presented here required a simple task to reduce cognitive input as much as possible. A GNG task was used based on Maguire et al.’s (2003) simple shape task, incorporating blocks of stimuli including either sequences of go-only stimuli or mixed go and no-go stimuli. The go-only blocks encourage quick and habit-like responding. fMRI readings have shown activity within frontal lobe regions during the GNG task, implicating the prefrontal cortex as an important structure in the inhibition of habit-like, basic motor responses (Maguire et al., 2003).

The study on cognitive disinhibition applied the Stroop task (ST; Stroop, 1935). The ST requires that participants first read aloud colour-name words printed on a card, giving a baseline score in terms of latency and error rate. Subsequently, participants must ignore the word and call out the ink colour the word is printed in. To perform well in the ST, it is assumed that cognitive inhibitory mechanisms are intact to deal with the cognitive conflict created by inhibiting the natural tendency of reading aloud the printed word rather than the ink the word is printed in. Any weakened performance on the ST, either in error rate or latency, represents the ‘Stroop effect’, and the greater this is the more disinhibited the person is said to be (Mintzer and Stitzer, 2002). As discussed above, control processes are believed to reside in the frontal lobe regions of the brain, and the ST is thought to show the state of executive functioning within this area, specifically the level of ‘mental’—or cognitive—control (Ratti et al., 2002). The ventromedial portion of the frontal lobes has been highlighted as more important for cognitive inhibitory control while motor...
inhibition has been linked more with the anterior cingulated cortex, an area affected by alcohol use (Bechara and Martin, 2004; Ramaekers and Kuypers, 2005).

As both disinhibition tasks used involved a visual element as well as very basic memory for instructions, a delayed matching to sample (DMS) task was employed in order to account for possible short-term memory and/or perceptual effects of alcohol, which may have hampered the interpretation of any disinhibition results if not assessed.

From the previous literature, it was hypothesized that a moderate dose of alcohol, previously shown to prime social drinkers, would weaken inhibition of basic motoric responses, leading to an increase in erroneous responding. Concerning the cognitive task, it was believed that alcohol would show less disinhibitory effects as the ST requires more complex cognitive processing to support accurate behaviour. Such an assumption was also based on previously reported data from studies that have examined the effects of alcohol on colour Stroop (Duka and Townshend, 2004).

METHODS

Study 1: Motoric disinhibition

**Design.** Conditions were equally divided by gender before participants were randomly allocated to one of two conditions: alcohol or placebo preload (between-subjects design). The GNG task involved three blocks where disinhibition, via error rate, could be measured, thus repeated measures were used, employing three (block) within-subjects factors and condition as the between-subjects factor. Response latency (RL) was also analysed using block as the within- and condition as the between-subjects factor.

The DMS task included four levels of delay. These were entered as within-subjects factors while condition was entered as the between-subjects factor. Both the number of correct responses and the RL were analysed.

**Participants.** Thirty-two participants (16 male) aged 22.00 (SEM ± 0.637) years participated in the study. Participants were recruited via email and posters advertisement and were screened for suitability through a medical assessment and questionnaire, carried out by a qualified person. All participants completed an alcohol use questionnaire (AUQ), giving an estimate of weekly alcohol consumption. Individuals who had past, or current, mental health disorders, drug dependence or abuse were not permitted to take part. Owing to the moderate dose of alcohol participants may have been required to consume during the experiment, individuals who drank less than 10 units of alcohol a week were excluded. Participants consumed an average of 33.199 (SEM ± 4.139) alcohol units per week. Participants were required not to drink alcohol the night before the experiment, take illegal drugs for at least 7 days before, and not to eat a meal of high fat content the night or morning before the study or to drink any caffeinated beverages on the day of attendance. Upon arrival, participants were given an overview of the experimental procedures. In order to counteract alcohol expectancy, participants were told that they might receive any of the following drinking ingredients: alcohol, soft drink, stimulant or sedative. An informed consent form was signed and participants were paid £10 on completion of the investigation.

**Beverage administration.** Beverages were presented on a tray laid with an absorbent towel. Alcohol was administered in a dose of 0.6 g/kg in a 500 ml solution: 90% food grade ethanol with Orangina. The alcohol dose is the equivalent to 5.25 UK units of alcohol (8 g) which can be found in approximately 2 pints of premium lager. The placebo beverage consisted of the respective volume of Orangina only, with drops of ethanol placed onto the towel. Drinks were divided into ten 50 ml aliquots, to be consumed within a structured 30 min period (3 min per aliquot).

**Blood alcohol levels.** Participants’ blood alcohol levels (BALs) were converted from breath alcohol concentrations assessed using a standard breathalyser (alcometer model S-D3M, Lmb Electronic Gmbh, Schweig, Germany). To participate, BAL had to be 0.0% before any experimental procedures commenced. BAL readings were taken at the end of the experimental session and participants were permitted to leave the laboratory when levels were below 0.04%, half the UK legal driving limit.

Measurements

**Nuffield medical questionnaire**

Health clearance was assessed and given using a medical questionnaire. This ensured participants were not taking any medication that may have either reacted badly to alcohol or could have affected results.

**Drinking habit and trait measures**

These measures were employed to assess any differences in participants’ drinking habits and expectations that might have affected the dependent variables.

**Alcohol use questionnaire (AUQ; Mehrabian and Russell, 1978).** The AUQ gives an estimation of average weekly alcohol consumption. In addition to an overall score, the AUQ gives a binge score based on consumption, speed of drinking (drinks per hour), number of intoxications in the past 6 months and the percentage of times of getting drunk when going out drinking (Townshend and Duka, 2005).

**Alcohol expectancies questionnaire (AEQ; Fromme et al., 1993).** The AEQ is a 38-item questionnaire consisting of seven factors, four positive (sociability, tension reduction, liquid courage, and sexuality) and three negative (cognitive and behavioural impairments, risk and aggression, and self-perception) for assessing expectations concerning the alcohol effects. The AEQ is based on Fromme’s Comprehensive Effects of Alcohol Questionnaire (Fromme et al., 1993) and was used to provide a picture of participants’ expectations concerning the effects of alcohol.

**Priming assessment**

After testing was complete, participants were asked to fill in a short assessment. The assessment asked whether—at any point after the preloads—they would have liked more of the preload if given the opportunity and, if so, how much more (maximum 10 aliquots). A final question was printed...
concerning whether they had wanted alcohol at any point after the preloads. This was printed on the reverse side, to avoid participants being influenced by the word ‘alcohol’.

Motoric disinhibition

Go/no-go task. Used to assess any effects of preload on participants’ ability to inhibit a response that had become habit-like through behavioural repetition.

The GNG consisted of five blocks (B) of stimuli in the following order: B1 = go and no-go (g/ng), B2 = go only, B3 = g/ng, B4 = go only, and B5 = g/ng. Each block consisted of 24 trials, in the g/ng blocks there were 12 go and 12 no-go stimuli. The stimuli consisted of blue ellipses on a black background; the shape was either presented vertically or rotated 45° clockwise. Participants were presented with a set of instructions on the computer screen, asking them to respond by pressing the spacebar once, as quickly as possible, whenever a vertical shape was presented, and not to respond whenever a rotated shape was presented. Instructions were reversed for half the participants, counterbalanced across preload type. Stimuli were presented for 2 s. However, the participants’ response terminated the stimulus presentation, with an inter-stimulus interval of 1 s. Block 1 acted as a baseline for performance while the go-only blocks were included to encourage the formation of a habit-like response to the go stimuli. The task took approximately 4 min. The GNG was performed using E-Prime software.

Perceptual & short-term recall memory

Delayed matching to sample task. Included as a measure of perceptual functioning and immediate and delayed recall memory in order to ascertain any detrimental effects of the preloads.

The participant was presented with a ‘sample’ complex visual pattern made up of four different coloured components on a touch-screen monitor. After varied intervals (simultaneous, 0, 4000 or 12 000 ms delays) four other ‘choice’ patterns are presented. The participants’ task was to press the choice which was an exact match to the sample. If the participant made a mistake, they were allowed to keep making choices until the correct pattern was chosen. To discourage strategies based on single components, all of the choice patterns had one component in common. After three practice trials there were 20 counterbalanced test trials (five simultaneous and five at each of the delays), the participant was then given the chance to rest before completing another 20 counterbalanced test trials. The DMS takes approximately 10 min to complete. The DMS was performed using CANTAB Eclipse software.

Procedure. Participants attended the laboratory for one session only. Upon arrival, participants provided a breathalyser reading (all zero), measures of height and weight, and filled in the medical questionnaire and informed consent form.

The AUQ and AEQ were completed before the 30 min structured preload phase commenced.

Approximately 22 min after the preload, participants completed the GNG and DMS tasks, the order of which was counterbalanced across participants. The two tasks took approximately 15 min to complete, thus the timing of the tasks was such that they were equally spread across the 30 min post-preload point, which was when priming and BAL was found to peak in previous research (Rose and Duka, 2006). Following the two tasks, participants were asked to fill in a short assessment regarding the preload they had consumed and whether or not they had wanted alcohol at any point during the experiment, following the preloads.

Finally, the participants gave a breathalyser reading—which had to correspond with a BAL of 0.04% or below to leave the laboratory—and were fully debriefed and compensated for their time.

Study 2: Cognitive disinhibition

Design. Equal numbers of male and female participants were randomly allocated to one of two preload conditions: alcohol or placebo (between-subjects design). The ST included two tasks: word and colour naming, both of which yielded an error and latency score. Independent t-tests were employed, entering condition as the grouping variable to assess any significant differences on these measures. The DMS was analysed as described above.

Participants. Thirty-two participants (16 male) aged 21.844 (SEM ± 0.596) years, with an average weekly alcohol consumption of 33.977 (SEM ± 4.038) took part. Participants were compensated for their time with £10. Eligibility details were identical across the two studies.

Beverage administration, BAL and measurements. As the motoric experiment described above, with the exception of the disinhibition task.

Cognitive disinhibition

Stroop task (ST; Stroop, 1935). The ST was used to assess whether cognitive disinhibition was greater following an alcohol preload relative to placebo.

The ST consisted of four colour words (blue, brown, green, and red) presented 28 times each, making a total of 112 words, matched for font and size, printed on a laminated sheet of A4 card. Each word was printed in an incongruent colour (blue, brown, green, and red), each colour occurring 28 times. Participants were instructed to face away from the experimenter, to minimize any possible distraction caused by the experimenter being in the same room. Participants were then asked to read out loud, as quickly as possible, the words printed on the card. After this task, which acted as both a familiarization of the stimuli and a baseline, participants were asked to ignore the words printed on the card and call out, as quickly as possible, the ink colour that the words were printed in. The experimenter measured errors and latency using a marking sheet. The task took approximately 4 min.

Perceptual & short-term recall memory

Delayed matching to sample task. Included and used as described above.

Procedure. The procedure followed the same course as the motoric disinhibition experiment: 23 min after the structured preload phase, participants completed the ST and the DMS task, the order of which was counterbalanced.
across participants. The two tasks took approximately 14 min to complete, thus the timing of the tasks was such that they were equally spread across the 30 min post-preload point. Following the two tasks, participants completed a short questionnaire regarding any desire they had felt for the preload and alcohol during the experiment, after they had consumed the preload.

Following this, the participants were fully debriefed, compensated and allowed to leave the laboratory once breathalyser readings indicated that BALs were 0.04% or below.

RESULTS

Study 1: Motoric disinhibition

Statistical analysis. One-way ANOVA was performed on personality characteristics, alcohol habit and expectancy measures to ensure groups were equal on these attributes.

A chi-square test was performed on the categorical data, to assess whether the number of participants wanting more of the preload or alcohol was significantly different between preload conditions.

Repeated measures were used to investigate any differences over time and between conditions on all the main dependent variables, using condition as between- and three levels of block (for GNG data) or four levels of delay (for DMS data) as within-subject factors. Main effects were investigated using bonferroni planned comparisons and interactions using contrasts, entering preload and time (when appropriate) as simple analysis.

Independent t-tests were performed on various signal detection measures which are automatically analysed using the CANTAB software.

Drinking habit and trait measures. One-way ANOVA revealed no statistically significant differences between groups in terms of alcohol habits or expectancies (Table 1).

Priming assessment. A chi-square test showed that 26.667% of those preloaded with alcohol had wanted more of the alcoholic preload, whereas 40.000% had wanted alcohol at some point during the experiment. None of the participants preloaded with placebo wanted either more of the non-alcoholic preload or alcohol (Table 2). However, the expected counts of the chi-square results did not exceed five, a requirement of the analysis if it is to be accepted (see Discussion).

Go/no-go task. GNG Blocks (B1, B3, B5)

Correct Responses: Repeated measures employing three levels of block as the within factor and condition as the between factor showed a main effect of block [F(2, 56) = 4.035, P = 0.023]. Compared with baseline, correct hit rate seemed to increase slightly. However, planned comparisons failed to pick up any effect (data not shown).

Correct RL (Fig. 1): A main effect of block [F(2, 56) = 5.296, P = 0.008] was found; bonferroni planned comparisons revealed that this difference occurred between B1 & B3 and B1 & B5; RLs decreased relative to baseline [I-J = 46.949, P = 0.016; I-J = 41.674, P = 0.073 respectively]. This suggests a robust practice effect, which made the go response habit-like.

Error Responses (Fig. 2): A significant main effect of block was found [F(2, 56) = 3.746, P = 0.030]. Planned
was found in the within-subjects contrasts \( F(2,28) = 6.840, P = 0.014 \). Planned contrasts revealed that the interaction occurred between blocks 3 and 5: error RL decreased after alcohol from block 3 to 5 but increased after placebo. Post-hoc between-subjects comparisons showed a significant difference in block 3 \( t(28) = 2.35, P = 0.026 \) but not in block 5.

Go-only blocks (B2 & B4): There were no statistically significant effects of block or differences between conditions in terms of correct responses or correct RL within the go-only blocks (data not shown).

**Delayed matching to sample**

Correct responses: A main effect of time occurred for the number of correct responses \( F(9, 84) = 10.510, P < 0.001 \). Comparisons showed that the number of correct responses during the simultaneous trials was significantly greater than those in any other delay \( 0 \text{ ms: } I-J = 0.800, P = 0.004; 4000 \text{ ms: } I-J = 0.633, P = 0.062; 12000 \text{ ms: } I-J = 1.300, P < 0.001 \). Correct responses during 4000 and 12000 ms delays also decreased \( I-J = -0.667, P = 0.085 \). There were no effects involving condition and therefore data are not shown.

Response Latency: A main effect of time was also found in relation to the RL [corrected \( F(2,28, 64.025) = 34.226, P < 0.001 \). Comparisons showed that latencies were significantly shorter during the simultaneous and 0 ms trials compared with the 4000 and 12000 ms trials [simultaneous & 4000 ms: \( I-J = -253.689, P = 0.006 \); simultaneous & 12000 ms: \( I-J = -740.808, P < 0.001 \); 0 & 4000 ms: \( I-J = -233.361, P = 0.019 \); 0 & 12000 ms: \( I-J = -720.479, P < 0.001 \). Latencies during the 4000 and 12000 ms trials also increased significantly \( I-J = -487.119, P < 0.001 \). There were no effects involving condition and therefore data are not shown.

Sensitivity to errors: Participants’ sensitivity to errors regardless of error tendency showed no difference between conditions \( P = 0.706 \). A high score (ranging from -1 to 1) denotes errors occurring after errors, participant scores averaged 0.591, thus sensitivity was not substantial [alcohol: mean = 0.573 (SEM ± 0.053); placebo: mean = 0.6100 (SEM ± 0.086)].

Strength of error effects: Strength of trace required to elicit an error, that is, the strength of the emotional reaction to an error needed to generate a subsequent, immediate error, was not different between groups \( P = 0.338 \). Scores range from -1 to 1. Low scores indicate participants are making numerous errors, whereas high scores illustrate that errors are only more likely immediately after an error. Participants’ scores illustrate that the strength of error effects was not great

<table>
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<tr>
<th>Factor</th>
<th>Alcohol</th>
<th>Placebo</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol units per week</td>
<td>31.946 (4.648)</td>
<td>36.009 (6.726)</td>
<td>0.623</td>
</tr>
<tr>
<td>per week</td>
<td>14.60 – 68.86</td>
<td>13.78 – 81.89</td>
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</tr>
<tr>
<td>AUQ score</td>
<td>56.076 (6.588)</td>
<td>72.470 (10.825)</td>
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<tr>
<td>Binge score</td>
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<td>37.040 (6.406)</td>
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<tr>
<td>Incorrect response latency (ms)</td>
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<td></td>
<td></td>
</tr>
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<td>0</td>
<td>Alcohol</td>
<td>Placebo</td>
<td>P value</td>
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<tr>
<td></td>
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<td></td>
<td>3.5</td>
<td>4.5</td>
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</tbody>
</table>

**Study 2**

Table 3. Average number of alcohol units per week, AUQ score, binge score and ratings for the seven different factors derived from the alcohol expectancy questionnaire (AEQ). Values are expressed as mean (±SEM) and range where appropriate. Statistical analysis showed no differences between preload groups on these factors, with the exception of sociability expectations: those in the alcohol preload group showed a higher expectation that alcohol would make them sociable.

Table 3. Average number of alcohol units per week, AUQ score, binge score and ratings for the seven different factors derived from the alcohol expectancy questionnaire (AEQ). Values are expressed as mean (±SEM) and range where appropriate. Statistical analysis showed no differences between preload groups on these factors, with the exception of sociability expectations: those in the alcohol preload group showed a higher expectation that alcohol would make them sociable.
showed no difference between conditions. Data were not shown.

There were no effects involving condition and therefore 
P from errors, shown by mean (SEM ±) position between −1

and 1, 1 denoting high sensitivity [alcohol: mean = −0.476, (SEM ±0.053); placebo: mean = −0.683, (SEM ±0.149)]

Study 2: Cognitive disinhibition

Statistical analysis. Analysis mirrored that of the motoric study with the exception of the disinhibition task: independent t-tests were used to assess whether performance on the ST differed across conditions in terms of error rate and latency. Main effects were investigated using bonferroni planned comparisons and interactions with contrasts, entering preload type and time — when appropriate — as simple analysis.

Drinking habit and trait measures. One-way ANOVA showed that participants only differed on the AEO factor of sociability; participants preloaded with alcohol held the expectation that alcohol would make them feel more sociable than those preloaded with placebo (Table 3).

 Priming assessment (Table 4). Of participants preloaded with alcohol 31.250% wanted more of the preload during the experiment and 43.750% wanted alcohol. None of the participants preloaded with placebo wanted any more of the preload or alcohol during the experiment. Although the chi-square analysis yielded some significant results, assumptions were not met, as in the motoric disinhibition task.

Stroop task. Word Stroop: Independent t-tests showed that there was no statistical difference between conditions on error rate or on latency to complete the task (data not shown).

Colour Stroop: Independent t-tests showed that those preloaded with alcohol made statistically more errors [t(30) = 2.444, P = 0.021; see Fig. 4(a)] and completed the task in significantly less time [t(30) = −2.107, P = 0.044; see Fig. 4(b)] than those preloaded with placebo.

Delayed matching to sample. Correct responses: A trend for a main effect of delay occurred [F(3, 90) = 2.205, P = 0.093], correct responses decreasing with delay.

Correct RL: A main effect of delay occurred [corrected F(1.416, 42.467) = 5.588, P = 0.014]; comparisons highlighted differences between simultaneous and 4000 ms & 12000 ms as well as a trend for 0 ms [I-J = −328.580, P < 0.001; I-J = −534.449, P = 0.020; I-J = −194.401, P = 0.065 respectively]. Latency increased as a function of delay. There were no effects involving condition and therefore data were not shown.

Sensitivity to errors: Participants’ sensitivity to errors showed no difference between conditions [F = 0.615]. Little sensitivity was found in terms of errors following directly on from errors, shown by mean (SEM ±) position between −1

and 1.

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<th>Number of measures (mean ±SEM range)</th>
<th>Wanted alcohol (out of 15)</th>
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<tr>
<td>Alcohol</td>
<td>5</td>
<td>1.500 (0.626) 3–8</td>
<td>7</td>
</tr>
<tr>
<td>Placebo</td>
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</table>

Table 4. Number of participants wanting more of the preload, the number of measures they wanted, and number of participants wanting alcohol during the experiment.

Fig. 4. (a) Error rate (mean ± SEM) in the colour Stroop task for alcohol and placebo preload. Number of errors were significantly more after the alcohol, relative to the placebo, preload. (b) Completion latency (seconds, mean ± SEM) in the colour Stroop task for alcohol and placebo preload. Those preloaded with alcohol completed the colour ST in less time than those preloaded with placebo.

DISCUSSION

Two studies were run in parallel to assess the relative influence that a moderate dose of alcohol, previously found to trigger desire for alcohol (Rose and Duka, 2006), had on basic motoric and cognitive inhibition processes. It was found that the alcohol preload affected aspects of the motoric processes tested in the GNG task and clearly impaired inhibitory
cognitive processes during the ST. However, the priming assessment analysis (i.e. increase in wanting alcohol) did not reveal statistically significant results in either of the two studies in contrast to the previous study (Rose and Duka, 2006). Crossover analysis using chi-square was applied for the categorical data of responding ‘yes’ or ‘no’ to wanting alcohol at any point during the experiments. To accept chi-square results, the expected counts of the analysis must exceed five in both conditions and this was not the case, as no participant preloaded with placebo responded with ‘yes’ to having wanted alcohol at any point. This contrasts the 40.00% and 43.75% of the subjects after alcohol preload reporting ‘yes’ in the motoric and cognitive experiments, respectively. In addition, within Rose and Duka’s (2006) previous experiment, the imagery procedure used required more in-depth processing from the participant, whilst the current priming assessment simply asked participants if they had wanted more of the preload or alcohol at any point after the preload consumption. It is possible that the former technique was more successful in gauging participants’ alcohol desire. However, due to the importance of the timing of the experimental tasks, it was inappropriate to include an imagery scenario and response task in these studies.

The disinhibition tasks required a minimal amount of memory to hold task instructions and of perceptual functioning to perceive the visual stimuli. To assess any detrimental alcohol effects on these processes, which may have affected the results, a DMS task was included. Results showed a tendency for error rate and RL to increase as a function of delay but alcohol had no effect. Thus, disinhibition findings cannot be accounted for by impairments in basic memory or perceptual functioning.

The GNG task was successful in producing a habit-like motoric response to go stimuli, as shown by the significant decrease in response latencies across blocks (Berlin and Bohlin, 2002). Error rates increased also from baseline to blocks 3 and 5 (this increase was found significant only for block 3) implying the paradigm was successful in producing a level of disinhibition. However, alcohol did not have an effect on the error rate. This finding contradicts previous work, which has shown that, even during fairly simple tasks requiring a degree of motoric inhibition, alcohol induces greater levels of disinhibition, measured by increased error rates (Fillmore and Vogel-Sprott, 1995; Fillmore, 2004). Although alcohol did not affect the occurrence of errors during no-go stimuli, it had a differential effect on the latency to an error compared to placebo. As the habit response was established, alcohol participants became faster whereas placebo participants became slower. It is interesting to note that in a previous study in which motoric disinhibition in the absence of cognitive processing was tested, an alcohol preload only affected reaction time (alcohol decreased reaction time) while leaving accuracy unaffected (Marinkovic et al., 2000). However, alcohol dose at any point in Marinkovic et al.’s (2000) study was slightly lower than in the present study (0.4 g/kg of 100% and 0.6 g/kg of 90% proof ethanol, respectively).

These findings, taken together, raise the question of whether changes in error rate, latency, or in both, provide the most reliable account for the findings with regard to disinhibitory effects of alcohol in the literature. If RL, then Marinkovic et al.’s (2000) findings correspond to the current findings in the motoric task. Indeed, the only differential effect of preload occurred with regard to the latencies to commit an error between blocks 3 and 5: latency decreased within the alcohol preload but increased within the placebo preload. It is interesting that this differential effect occurred between blocks 3 and 5, after participants had progressed in the habit training (blocks 2 and 4). It is possible that after the placebo preload, participants became more wary of the task, perhaps owing to the previous experience of having to inhibit responses suddenly after the habit-forming in blocks 2 and 4. In contrast, alcohol-preloaded participants became quicker, indicating that they were not influenced by past experience. Lack of using prior knowledge to act appropriately indicates that alcohol did indeed weaken certain processes likely to be used in self-control. Thus, it can be suggested that in accordance with Marinkovic et al.’s (2000) data, a moderate dose of alcohol has basic motor disinhibiting effects.

Fillmore and Rush (2001) after the preload argued that alcohol may affect both the behavioural activation system (BAS) and the behavioural inhibition system (BIS). It is difficult to suggest from our data whether the effects of alcohol on the motoric task is mediated by the BAS, BIS, or both. However, as the effects were seen on latencies not on error commission during the no-go condition, it could be suggested that a priming dose of alcohol may have a greater effect on active, approach systems like BAS. Nevertheless, further studies should address this question more directly.

With regard to the ST, alcohol increased error rates and decreased latencies to complete the task. Most studies looking at Stroop effects in the context of drug- or pathology-related behaviours have used a modified version, which includes words relevant to the particular populations being assessed—food-related for eating disorders (Johansson and Hansen, 2000), smoking-related for smokers (Munafo et al., 2003) and alcohol-related for alcoholics (Duka and Townshend, 2004). In our study we have used the classical ST, which includes colour words. Our intention in the present studies was to examine general basic effects of alcohol on motoric and cognitive processes outside cognitive or emotional processes related to alcohol drinking.

The key finding from the second experiment was that those preload with alcohol made more errors and completed the colour-naming task in less time than those preload with placebo. Previous literature looking at the Stroop effect, focusing on increases in error rate or latency to complete the task, has been somewhat mixed concerning the effects of alcohol on the classic ST: while Gustafson and Kallmen (1990) found that a dose of 1 ml/kg of alcohol (100%) led to a more pronounced Stroop effect, Duka and Townshend (2004) failed to show an increase in disinhibition following preloads of 0.3 g/kg and 0.6 g/kg doses of alcohol, although alcohol did increase disinhibition on an alcohol modified ST.

The findings of Study 2 suggest that a dose of 0.6 g/kg of alcohol does increase cognitive disinhibition. The current finding that the ST was completed in less time after alcohol relative to placebo is unusual. One possible explanation may come from a finding by Fillmore and Rush (2001) that, after alcohol, participants would trade accuracy for speed during a stop-signal task, even when good performance—assessed on both accuracy and speed—was rewarded. Although no reward was offered during the current research for completing
the ST, it is possible that completing the task served as a reward. Alternatively, even in the absence of reward, alcohol may influence behaviour, so that speed is preferred over more careful actions indicating an increase in impulsive use of alcohol. These are tentative explanations and the reason for this finding remains unclear.

The current findings fit in with the growing amount of evidence implicating alcohol as having attenuating effects on inhibition. The importance of such an effect may be wide-ranging, not only has alcohol been linked with acts of impulsivity, including aggression, but disinhibition may also support the continuation of drinking once it is initiated and thus may play a role in the occurrence of binge drinking, a drinking pattern which has numerous negative consequences (Townshend and Duka, 2005). Theoretically, the findings fit in with several models including that of Volkow et al. (2004), which provides an interesting insight into compulsive drug-taking incorporating reward, memory, and lack of control.

The research aimed to explore the relative influence that a dose of alcohol, previously found to prime desire for alcohol, had on basic motoric and cognitive control processes, a distinction that has been made by previous researchers (Bechara et al., 1994). Alcohol showed disinhibitory effects in both the motoric and the cognitive task. Although it is unlikely that motoric and cognitive inhibitory processes are mutually exclusive categories, the present research allowed the study of two basic control mechanisms before looking into more complex processes, for instance alcohol-related processes, which may further elucidate how disinhibition may contribute to the maintenance of alcohol-related behaviours.

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