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Title: Reduced attentional capture by reward following an acute dose of alcohol

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Abstract

Rationale

Previous research has shown that physically-salient and reward-related distractors can automatically capture attention and eye-gaze in a visual search task, even though participants are motivated to ignore these stimuli.

Objectives

To examine whether an acute, low dose of alcohol would influence involuntary attentional capture by stimuli signalling reward.

Methods

Participants were assigned to the alcohol or placebo group before completing a visual-search task. Successful identification of the target earned either a low or high monetary reward but this reward was omitted if any eye gaze was registered on the reward-signalling distractor.

Results

Participants who had consumed alcohol were significantly less likely than those in the placebo condition to have their attention captured by a distractor stimulus that signalled the availability of high reward. Analysis of saccade latencies suggested that this difference reflected a reduction in the likelihood of impulsive eye-movements following alcohol.

Conclusions

Our findings suggest that alcohol intoxication reduces the capacity to attend to information in the environment that is not directly relevant to the task at hand, consistent with alcohol myopia theory. In the current task this led to a performance benefit under alcohol, but in situations that require rapid responding to salient events the effect on behaviour would be deleterious.

Introduction

Alcohol is one of the most widely used drugs globally and its effects on mood and emotion are a commonly cited motivation for drinking (Park 2004). Perhaps surprisingly then, an influential theory of alcohol's stress-reducing and social effects asserts that alcohol does not have direct effects on emotion but that instead, alcohol affects attention and information processing, which in turn influences emotions and behaviour (Josephs and Steele 1990; Steele and Josephs 1990). According to this theory, alcohol reduces attentional capacity and thus restricts attention to the most salient and immediate cues, at the expense of other information in the environment. The implication is that intoxicated individuals will be less likely to allocate attention to competing information – an 'inoculating' effect that might be beneficial in some contexts (when task-irrelevant information could cause distraction) but detrimental in others where situations can change rapidly and fast updating is required (e.g., when driving). Consistent with this idea, Josephs and Steele (1990) demonstrated that following an acute dose of alcohol, participants reported decreased anxiety to an upcoming psychosocial stressor when their attention was focused on a simple filler task, relative to a group also given alcohol, but not given a filler task. No effect of the filler task was found in the no-alcohol group. Josephs and Steel took these findings to indicate that alcohol reduced the capacity to attend to anything beyond the immediate (filler) task, an effect termed alcohol myopia (Josephs and Steele 1990; Steele and Josephs 1990).

The theory of alcohol myopia has received support from varied laboratory studies of attention. It has been demonstrated that alcohol reduces distraction by stimuli that are competing for attention, ostensibly because attentional capacity is so reduced that there is no attentional reserve remaining to attend to stimuli that are not directly related to the primary task at hand (Erblich and Earleywine 1995; Jääskeläinen et al. 1999; Clifasefi et al. 2006; Allen et al. 2009; Harvey et al. 2018; see for reviews: Koelega 1995; Mocaiber et al. 2011). For example, Erblich and Earlywine (1995) had participants perform tests of short-term memory both when intoxicated and when sober. Only when participants were sober was memory performance impaired by verbal auditory distraction. Using the famous 'gorilla task' two studies reported increased inattention blindness following alcohol administration, such that intoxicated participants who were asked to count the number of passes made between basketball players were less likely to notice a person dressed as a gorilla saunter across the basketball court than were those who were sober (Clifasefi et al. 2006; Harvey et al. 2018). Demonstrating the potentially negative effects of narrowed attention, driving simulation studies have shown that an acute dose of alcohol reduces the capacity of drivers to attend to other stimuli in the visual scene (in a real-life setting this could be a child about to run into the road) and increases the tendency to focus on one aspect of driving such as staying in the middle of the lane (Allen et al. 2009; see for review: Koelega 1995). It should be noted that there is considerable debate in the cognitive literature as to whether alcohol *literally* narrows attentional focus (in a spatial sense) with some arguing for reduced spatial distribution of

attention (Canto-Pereira et al. 2016; Bayless and Harvey 2016) but others demonstrating that under certain task conditions, attention can be distributed across a diffuse spatial area following alcohol administration (Colflesh and Wiley 2013; Harvey 2016). However, the key assertion of alcohol myopia theory—that attentional capacity is functionally reduced following alcohol administration—seems robust. Lending further support, neuroimaging studies have reported that neural responses to rare and unpredictable events are reduced following alcohol administration, further suggesting that brain regions involved in the detection of distractors are influenced by an acute dose of alcohol (Jääskeläinen et al. 1999; Allen et al. 2009; Kenemans et al. 2010).

In the studies outlined above, distracting stimuli were physically distinct from participants' focal task. However, recent research has shown that it is not only the physical features of stimuli that determine how they compete for attentional resources; attention is also influenced by the motivational significance of stimuli – that is, their affective content or their association with rewards or punishments (see for review: Watson et al. 2019b). Consistent with the findings reviewed above, it also seems that distraction by motivationally significant distractors is reduced following alcohol administration, as demonstrated by two studies using different forms of distraction by negatively-valenced stimuli (Curtin et al. 2001; Erthal et al. 2005). In a perceptual discrimination task, participants showed reduced attentional capture by unpleasant distractor images following an alcohol dose (Erthal et al. 2005). Curtin et al., (2001) examined the effect of alcohol on the fear-potentiated startle response elicited by presentation of words that predicted delivery of electric shock (relative to words that did not). The baseline fear-potentiated startle response was similar in intoxicated and sober participants. However, under divided attention conditions—when participants were assigned the additional task of responding to the colour of the words—a reduced fear response was observed in the alcohol relative to the no alcohol group. Taken together, these studies suggest that an acute dose of alcohol reduces the resources available to process information that is not directly relevant to the task at hand, even when such information is aversive and of motivational relevance to participants.

The studies cited above suggest that alcohol renders aversive stimuli less distracting. However, it is unclear whether this effect is specific to aversive stimuli (implying a specific influence of alcohol on the aversive motivational system), or whether distraction by appetitive stimuli would also be reduced (consistent with a more general influence of alcohol on distraction by salient stimuli). Consequently, in the current study we investigated the effect of alcohol on attentional distraction by reward-associated stimuli. Beyond its theoretical significance, this question has potential practical implications. We spend our lives surrounded by reward-related stimuli (signs for fast-food restaurants, advertisements featuring attractive models, notifications of 'likes' on social media etc), and so it is important to understand how alcohol influences the extent to which such stimuli impinge on our cognition and behaviour.

In the current study, we used an eye-tracking procedure developed by Le Pelley, Pearson, Griffiths and Beesley (2015) to investigate attentional capture by reward-related stimuli in visual search. In this procedure (a variant of the additional singleton task: Theeuwes 1992, 1994), participants must search for a diamond target amongst circles on each trial (see Figure 1). Within the search display, a uniquely coloured singleton circle (termed the *distractor*) signals whether a high or low reward is available for making a rapid eye-movement (saccade) to the target diamond on that trial. For example, a blue distractor circle might signal that a rapid saccade to the target will receive high reward (500 points: points are later converted into money), whereas an orange circle signals that low reward is available (1 point). Importantly, while the colour of the distractor signals the size of reward that is available, participants are informed that looking at this distractor will result in the reward being omitted. Under these conditions it is counterproductive to look at the coloured distractors in general, and particularly counterproductive to look at the coloured distractor signalling availability of high reward. Nevertheless, the consistent finding is that participants look at the coloured distractor on many trials, and critically are more likely to look at the high-reward distractor than the low-reward distractor, even though this means they miss out on more high-value rewards. (Le Pelley et al. 2015; Failing et al. 2015; Pearson et al. 2016; Watson et al. 2019a). These findings suggest that the attention-grabbing nature of the distractors is influenced not only by their physical salience (their distinctiveness as colour-singletons), but also by their incentive salience (their status as signals of reward). The effects of both physical salience and reward on attention are rapid, being most pronounced in the fastest eye movements that participants make: that is, rapid, impulsive saccades are particularly likely to go towards salient distractors, whereas slower, more controlled saccades are more likely to go towards the target (Godijn and Theeuwes 2002; Mulckhuyse et al. 2008; Failing et al. 2015; Pearson et al. 2016).

Figure 1. Trial structure of the visual search task. Participants began each trial by fixating a central white cross inside a circle. A search display then appeared, and participants' task was to move their eyes to the diamond (the *target*) as quickly as possible. The search display could contain a colour-singleton circle (the *distractor*), and the colour of this distractor signalled the number of points available for making a rapid eye movement to the target – in this example blue signals availability of 500 points (high reward). However, if participants looked at the coloured distractor prior to looking at the target, the reward that would have been delivered on that trial was cancelled (*reward omission trials*).

In the current study we investigated how an acute dose of alcohol influences performance in this task. In fact this procedure provides a particularly interesting test of the effect of alcohol on attention, in that one can generate opposing predictions based on different interpretations of previous theorising. One interpretation sees alcohol myopia as reflecting a reduction in overall processing capacity, such that participants must devote more attentional resources to perform the main task. This leaves fewer resources for processing of other stimuli, hence rendering them less distracting. On this account, which mirrors the *load theory* of selective attention (Lavie et al. 2004; Lavie 2005) distraction by reward-related distractors should be reduced following an acute dose of alcohol (much as distraction by task-irrelevant, aversive stimuli is reduced: Curtin et al., 2001; Erthal et al., 2005). An alternative interpretation is that alcohol myopia results from attention being restricted to the most salient available cues (Steele and Josephs 1990). In previous studies, this would likely have been the task-relevant cues since these were physically salient (Curtin et al. 2001) or appeared in a predictable location allowing for proactive allocation of spatial attention (Erthal et al. 2005). By contrast, in the current study the *distractors* are highly salient, marked both by their incentive (reward-related) salience, and also by physical salience (as the only coloured item in the scene). Moreover, participants do not know where the target (the diamond shape) will

appear prior to the onset of the search display, and hence cannot usefully pre-allocate spatial attention. If the effect of alcohol is to increase the likelihood that highly salient cues will capture attention, then on this interpretation we might expect greater capture by reward-related distractors following acute alcohol. This latter pattern would also be consistent with neurobiological evidence relating to the effect of alcohol on striatal brain regions that process reward (Schreckenberger et al. 2004) and associations between reward sensitivity, impulsivity and hedonic responses to alcohol (Reynolds et al. 2006; Dougherty et al. 2008; Bidwell et al. 2013; Morris et al. 2016); taken together, this work indicates that reward signals may become more salient following acute doses of alcohol and alcohol may increase the automatic orienting of attention towards reward-related signals.

Method

Participants and Apparatus

This study was approved by the UNSW Sydney Human Research Ethics Advisory Panel (Psychology). Inclusion criteria were being at least 18 years of age, drinking alcohol at least occasionally and not scoring higher than 15 on the Alcohol Use Disorders Inventory Test (AUDIT; Babor et al. 2001). Those who met these criteria were randomly assigned to the placebo or alcohol condition, with the constraint that gender was evenly matched across the groups. Given the large effect sizes observed in studies that have examined the effects of alcohol on various cognitive processes (Hindmarch et al. 1991; Dry et al. 2012) we tested for as many days required to test 28 participants in each condition (alcohol/placebo). This gave power of .90 to detect an effect size of $f=0.4$ (critical interaction between reward level and alcohol condition in the mixed ANOVA). In total we recruited 62 participants. Three of these participants had an AUDIT score higher than 15 and were excluded from the study. One further participant (assigned to the alcohol group) was unable to consume both drinks and was excluded. Our final sample therefore comprised 58 participants, with 29 in each condition. All participants received course credit and a monetary bonus that was contingent on their performance in the visual search task ($M = \$10.09$ AUD, $SEM = \$0.48$ AUD).

Participants were tested individually using a Tobii TX300 eye-tracker (sample rate 300 Hz), mounted on a 23-inch monitor (1920 × 1280 resolution, 60 Hz refresh rate). For gaze-contingent calculations, the experiment script down-sampled gaze data to 100 Hz.

Participants' head position was stabilised using a chinrest 60 cm from the screen. Stimulus presentation was controlled by MATLAB using Psychophysics Toolbox extensions (Brainard 1997; Pelli 1997; Kleiner et al. 2007). The experiment script and raw data for the experiment reported in this article are available via the Open Science Framework at <https://osf.io/fxcpt/>.

Design

Visual search task. The visual search task was based on that used by Pearson et al. (2016). All stimuli were presented on a black background. Participants began each trial by fixating in the centre of the screen (fixation display; see Figure 1). Once participants had accumulated 300 ms of gaze dwell time inside a circle (diameter 3.0° visual angle) surrounding a central cross, the fixation display turned yellow for 300 ms, indicating that the trial was about to begin. Following a 150-ms blank interval, the search display appeared. The search display comprised a set of six shapes (each $2.3 \times 2.3^\circ$ visual angle), distributed evenly around the centre-point of the screen, with the centre of each shape at an eccentricity of 5.1° visual angle. Five shapes were circles, and one was a diamond (the target). On the majority of trials, one of the circles was coloured either blue (RGB values: 37, 141, 165) or orange (RGB: 193, 95, 30) with similar luminance (~ 24.5 cd/m²): we term this colour-singleton circle the *distractor*. All other shapes were grey (RGB: 70, 70, 70; luminance ~ 8.3 cd/m²). We also included a small number of distractor-absent trials (all shapes rendered in grey, no colour singleton) where participants could earn 10 points for correctly looking at the target.

Participants' task was to move their eyes as quickly as possible to the diamond target: a response was registered once 100 ms of gaze dwell time had accumulated within a region of interest (diameter 3.5° visual angle) centred on this target. The colour of the distractor circle present in the search display signalled the magnitude of reward that was available on each trial. For half of participants, blue was the high-reward colour and orange was the low-reward colour; for the other half of participants this mapping was reversed. If the display contained a distractor rendered in the high-reward colour, a rapid eye-movement to the diamond target earned 500 points; if the display contained a distractor in the low-reward colour (or no colour-singleton distractor), a rapid response earned only 10 points. Crucially, however, if any eye gaze was registered within a 5.1° diameter region centred on the distractor circle, the reward was cancelled (henceforth referred to as *reward omission trials*). The trial ended immediately when a response to the target was registered, or after 2000 ms (timeout). If the trial was not a reward omission trial and response time was less than 1000ms, participants saw a feedback screen indicating the number of points earned on that trials (10 or 500). If the trial was a reward omission trial, the feedback screen indicated "+0 points, you could have won 500/10 points" (as appropriate). If response time was greater than 1000 ms, feedback read "+0 points, too slow: you could have won 500/10 points". If no response was registered before the timeout, feedback read: "Too slow: please try to look at the diamond more quickly". Feedback was presented for 1500 ms and the inter-trial interval was 700 ms.

Trials were arranged in blocks of 30. Each of the first two blocks (pretraining phase; before any alcohol was administered) consisted of 13 trials with the high-reward distractor, 13 trials with the low-reward distractor, and 4 distractor-absent trials. The 12 blocks following alcohol

administration (main phase) each contained 10 trials with a high-reward distractor, 10 trials with a low-reward distractor, 4 distractor-absent trials, and 6 'both-distractor' trials in which both the high- and low-reward distractors appeared in the search display. On these both-distractor trials, participants could earn 500 points for a rapid saccade to the target, but this reward was omitted if gaze was detected on either of the coloured distractors. Trial order in each block was randomised. Target location was determined randomly on each trial. If the display contained a single coloured distractor, this distractor was randomly located one or two positions clockwise or anticlockwise from the target. On distractor-absent trials, one of the grey circles one or two positions from the target was randomly selected to play the role of a 'distractor', and gaze on this stimulus triggered an omission trial just as on trials with a coloured distractor. On both-distractor trials, both the high and low-reward distractor were positioned either one or two positions from the target, in opposite directions (i.e., if the high-reward distractor was two positions clockwise from the target, the low-reward distractor was two positions counter-clockwise from the target).

AUDIT questionnaire. The AUDIT is a widely used self-report tool, developed by the World Health Organisation, to assess alcohol use risk (Babor et al. 2001). The AUDIT consists of 10-items assessing hazardous alcohol use in the last year. Scores greater than 8 are taken to indicate hazardous drinking behaviour and scores greater than 15 to indicate a high level of alcohol-related problems (an exclusion criterion in the current study).

Behavioral inhibition system/Behavioral activation system (BIS/BAS) questionnaire. The BISBAS scale (Carver and White 1994) consists of 24 items scored on a 4-point scale ranging from "very false for me" to "very true for me" and has good reliability (Cooper et al. 2007). We focused on the subscale of reward responsiveness (example item: "When I get something I want, I feel excited and energized.") in order to verify that this relevant factor did not differ significantly between alcohol and placebo groups.

Perceived intoxication rating. Participants were asked to indicate how intoxicated they felt after consuming the drinks on a VAS scale from 0 to 100 marked with the anchors "completely sober" and "very drunk".

Breath alcohol concentration (BrAC) measurements. At various points during the experiment participants were asked to blow into a calibrated Alcolizer LE Breathalyzer (Alcolizer Technology, Australia), which is a law enforcement grade device (certified to Australian Standard 3547) used to measure breath alcohol concentration (BrAC), expressed as grams of alcohol per 210 litres of breath (g/210L). For reference, the legal BrAC limit for driving is 0.050 g/210L in Australia, and 0.080 g/210L in the United States.

Procedure

Figure 2a provides an overview of the procedure of this study. Participants first completed the AUDIT and BISBAS questionnaires. Participants scoring above 15 on the AUDIT were deemed ineligible to participate in the study.

For remaining participants, following these questionnaires a baseline BrAC measurement was taken. We then used a Beurer BF600W body composition scale to measure participants' lean body mass (kg), which would be used later to calculate alcohol dose for those in the alcohol group.

Participants then completed the pretraining phase of the visual search task, in order to familiarise them with the task in a sober state. They were informed that their task was to move their eyes to the diamond "as quickly and directly as possible", and that they would receive a monetary bonus ("typically \$8 to \$13") at the end of the experiment, depending on how many points they earned; no other information on the conversion rate between points and money was provided. Participants were then instructed on the colour-reward contingencies in the visual search task, e.g., that whenever a blue circle was present in the display, they could win 500 points for looking at the diamond, and whenever an orange circle was present, they could win 10 points. Participants were also informed that looking at the coloured distractor would result in the reward on that trial being omitted. Participants were asked to repeat these instructions to the experimenter to ensure that they understood.

After completion of the first two blocks, the experimenter mixed the drinks in front of the participant. Participants in the alcohol condition received 0.6 g of alcohol per kg of lean body mass across two drinks that each consisted of vodka (37.5% alcohol by volume), a sugar-free lemon cordial and diet tonic water in ratios of 1:1:2. This dose was intended to achieve BrAC of .05g/210L, the legal limit for driving in Australia. Participants in the placebo condition received water in place of the vodka (poured from a vodka bottle) and the rim of each cup was smeared discreetly with vodka. Participants were told to drink at a pace comfortable to them, but within 20 minutes.

The instructions of the visual search task were then repeated to participants before they completed the main phase of this task (12 blocks), with a short break after every 2 blocks. After completion of the sixth block (around 15 minutes after finishing the drinks) and the eight block (around 20 minutes after finishing the drinks) a BrAC measurement was taken.

Finally, participants completed the alcohol beliefs questionnaire, provided a final BrAC measurement, were debriefed and received the money that they had earned in the task.

Figure 2. (a) Schematic of the procedure of the experiment. Phases shown in yellow are self-report ratings; in green are the practice and main phase of the visual search task; and blue shows the point at which participants consumed alcohol or placebo according to group assignment. Points at which breath alcohol concentration (BrAC) samples were taken are indicated (BrAC1 to BrAC4). **(b)** BrAC across samples BrAC1 to BrAC4 for participants in the placebo and alcohol conditions. All participants in the placebo condition had zero BrAC at all timepoints. Error bars show standard error of the mean. **(c)** Self-reported perceived intoxication ratings taken at the end of the experiment, for participants who had earlier consumed placebo (Plac) or alcohol (Alc). Grey symbols show individual data; black lines show mean and standard error of the mean.

Data Analysis

Analysis scripts and raw data are available at <https://osf.io/fxcpt/>. Analysis of data from the main phase of the visual search task followed our established protocols (Pearson et al. 2015; Le Pelley et al. 2019; Watson et al. 2019a). We discarded data from the first two trials of each block, trials in which the search task timed out, and trials with less than 25% valid gaze location data (Table 1 shows proportions of excluded trials for alcohol and placebo groups).

The primary dependent variable for the main phase of the search task was the proportion of omission trials: trials in which participants looked at the coloured distractor (or the grey circle assigned the status of a distractor on distractor-absent trials) and hence the reward was cancelled. We were particularly interested in three different contrasts (see also Watson et al., 2019). First, comparing performance on trials with a low-reward colour-singleton distractor with distractor-absent trials isolated the effect of physical salience on attention, since these trial-types differed in whether the search display contained a colour-singleton distractor, but

had the same reward (10 points). Second, comparing performance on trials with a high-reward versus low-reward singleton distractor allowed us to isolate the effect of reward on attention: in both cases the search display featured a colour-singleton distractor (and counterbalancing of colour–reward relationships ensured that these distractors had equivalent physical salience across participants), the only difference was whether this distractor signalled availability of a high or low reward. The third contrast examined both-distractor trials, in which both the high- and low-reward distractors appeared in the search display, placing them in direct competition for attention. Comparing the proportion of both-distractor trials in which participants looked that at the high- versus the low-reward distractor provides another index of the effect of reward on attention.

Results

Demographic Information

Table 1 shows demographic information for the alcohol and placebo groups. The groups did not differ significantly on any of the demographic or questionnaire measures. The table also shows data quality indicators from the test phase of the visual search task (proportion of trials excluded from analysis due to time-outs; proportion excluded due to poor eye tracking [less than 25% valid gaze location data]; and proportion of valid gaze location samples recorded from the eye tracker). Again these did not differ significantly between groups, indicating that alcohol consumption did not influence the quality of eye-tracking data.

Table 1. Demographic information and data quality indicators for the alcohol and placebo groups.

	Alcohol (<i>n</i> = 29)	Placebo (<i>n</i> = 29)	Comparison
Gender ratio (female : male)	16 : 13	16 : 13	–
Age	19.34 (0.29)	19.97 (0.48)	<i>p</i> = .28
BMI	22.95 (0.67)	23.13 (0.68)	<i>p</i> = .85
AUDIT	6.21 (0.63)	6.59 (0.67)	<i>p</i> = .68
BISBAS Reward Responsiveness	17.62 (0.38)	17.24 (0.36)	<i>p</i> = .47
% time-out trials ^a	1.08% (.31%)	1.04% (.42%)	<i>p</i> = .94
% invalid eye-tracking trials ^b	.59% (.22%)	.38% (.13%)	<i>p</i> = .41
% valid gaze registered ^c	96.7% (.6%)	96.8% (.6%)	<i>p</i> = .92

Note: Rows other than that for gender ratio show *M* (*SEM*) for the alcohol and placebo groups. The final column shows the *p*-value resulting from an independent t-test comparing the two groups. BMI = Body mass index; AUDIT = Alcohol use disorders identification test; BISBAS = Behavioral inhibition/activation system questionnaire. ^a Percentage of trials of the test phase of the visual search task excluded from analysis (from 360 total) because no response was registered before the search display timed-out (after 2000 ms). ^b Percentage of remaining trials excluded due to less than 25% valid

gaze data. ^c Percentage of gaze samples from the eye-tracker containing valid gaze location data (following exclusions).

BrAC and Perceived Intoxication Ratings

Figure 2b shows BrAC readings across the four measurement points (BrAC1-BrAC4: see Figure 2a). All participants had a BrAC reading of 0g/210L at baseline (BrAC1). For participants who consumed the placebo drink, BrAC remained at zero throughout the experiment. For participants who consumed alcohol, BrAC increased significantly from baseline to the BrAC2 measurement, approximately 15 minutes after participants had consumed the drinks, $t(28) = 11.2$, $p < .001$, $d_z = 2.08$, and had not declined significantly by the end of the experiment (BrAC4), $t(28) = 0.22$, $p = .83$, $d_z = 0.04$.

Figure 2c shows self-reported perceived intoxication ratings, measured at the end of the experiment. Many of the participants in the placebo group reported non-zero intoxication levels ($M = 24$, $SEM = 4.1$), despite not having received any alcohol. However, perceived intoxication was significantly higher for participants in the alcohol group ($M = 58.4$, $SEM = 4.2$), $t(56) = 5.83$, $p < .001$, $d = 1.53$.

Main Phase of Visual Search Task

During the main phase of the visual search task, participants who had consumed alcohol performed significantly better than those who had consumed placebo in terms of number of points earned (alcohol $M = 67,076$, $SEM = 2,525$; placebo $M = 52,800$, $SEM = 3,865$), $t(56) = 3.09$, $p = .003$, $d = 0.81$. This meant, in turn, that alcohol participants earned a larger monetary bonus than placebo participants, since the bonus was determined by the number of points earned.

Omission trials: Effect of physical salience. Analysis of proportion of omission trials via ANOVA with factors of distractor type (single low-reward distractor vs. distractor-absent) and alcohol condition (alcohol vs. placebo) revealed a main effect of distractor type, $F(1,56) = 28.0$, $p < .001$, $\eta_p^2 = 0.33$, with omission trials more likely when the display contained a colour-singleton distractor than when it did not (Figure 3a), demonstrating an influence of physical salience on attention. The main effect of alcohol condition did not reach significance, $F(1,56) = 3.27$, $p = .076$, $\eta_p^2 = 0.06$, and there was no significant interaction, $F(1,56) = 0.54$, $p = .47$, $\eta_p^2 = 0.01$. Hence the effect of physical salience on attention did not differ significantly as a function of alcohol consumption (Figure 3b).

Figure 3. (a) Proportion of omission trials for single-distractor trials featuring a high-reward distractor, a low-reward distractor, or no colour-singleton distractor (distractor absent), for participants who had consumed placebo or alcohol. Omission trials are trials on which participants looked at the colour-singleton distractor (or the grey circle assigned the status of a distractor on distractor-absent trials) prior to looking at the target, hence causing reward omission. **(b)** Physical salience (PS) score (difference in proportion of omissions on low-reward vs. distractor-absent trials), and **(c)** reward score (difference in proportion of omissions on high- vs. low-reward distractor trials), for placebo (Plac) and alcohol (Alc) conditions. **(d)** Proportion of both-distractor trials on which participants caused reward omission by looking at the high- vs. low-reward distractor after consuming placebo or alcohol. Error bars show standard error of the mean.

Omission trials: Effect of reward. Initial analysis of the effect of reward on the proportion of omission trials in the search task considered single-distractor trials. ANOVA with factors of distractor type (high reward vs. low reward) and alcohol condition revealed a main effect of distractor type, $F(1,56) = 98.7$, $p < .001$, $\eta_p^2 = 0.64$; Figure 3a shows omission trials were more likely when the distractor signalled high reward than low reward, showing an effect of reward on attentional capture. There was also a main effect of alcohol condition, $F(1,56) = 13.0$, $p < .001$, $\eta_p^2 = 0.19$, that was qualified by a significant interaction, $F(1,56) = 9.61$, $p = .003$, $\eta_p^2 = 0.15$, with a smaller effect of reward on distraction in the alcohol condition than the placebo condition (see Figure 3c). Analysis of simple effects revealed that participants were significantly less likely to look at the high-reward distractor in the alcohol condition than the placebo condition, $t(56) = 3.64$, $p < .001$, $d = 0.96$, whereas eye movements to the low-reward distractor did not differ significantly between groups, $t(56) = 1.65$, $p = .105$, $d = 0.43$.

The key finding that the effect of reward on attention was reduced following alcohol consumption was supported by analysis of the proportion of *both-distractor* trials in which participants looked at the high- versus low-reward distractor. ANOVA revealed significant main effects of distractor type, $F(1,56) = 93.2$, $p < .001$, $\eta_p^2 = 0.62$, and alcohol condition, $F(1,56) = 10.6$, $p = .002$, $\eta_p^2 = 0.16$; critically there was also a significant interaction,

$F(1,56) = 9.25, p = .004, \eta_p^2 = 0.14$, with a smaller effect of reward on attention in the alcohol condition than the placebo condition. Analysis of simple effects revealed that alcohol consumption decreased capture by the high-reward distractor, $t(56) = 3.28, p = .002, d = 0.86$, but had no significant effect on capture by the low-reward distractor, $t(56) = 0.39, p = .70, d = 0.10$.

Saccade latency and direction. To summarise, alcohol consumption *improved* performance in the visual search task. More specifically, it seemed to result in participants being less distracted by the high-reward distractor, and hence less likely to lose out on high-value rewards. Follow-up analyses explored this effect further, by considering differences in saccade latencies: the latency between onset of the search display and the first saccadic eye-movement. A velocity-threshold identification algorithm (velocity criterion 40° visual angle per second: see Salvucci & Goldberg, 2000) was used to detect the first saccade on each trial, using the raw data from the eye-tracker (sampled at 300 Hz). Following our previous protocols (Le Pelley, Pearson, Porter, Yee & Luque, 2019; Pearson et al., 2016), trials were excluded from saccade analysis if the participant made an anticipatory saccade (saccade latency below 80 ms), if no gaze was recorded within 100 pixels of the central fixation point in the first 80 ms of the trial, or if no saccade was detected. These criteria led to exclusion of a further 12.5% of trials.

Averaged across all trials of the main phase of the visual search task, participants in the alcohol group were significantly slower to initiate a saccade following presentation of the search display ($M = 251.4$ ms, $SEM = 5.2$ ms) than those in the placebo group ($M = 229.4$ ms, $SEM = 5.8$ ms), $t(56) = 2.83, p = .006, d = 0.74$. However, this analysis provides only a coarse indication of between-group differences since it collapses across different trial types, and saccades going to different locations (e.g., towards a salient distractor or towards the target).

More focussed analyses examined data from single-distractor trials with a high-reward versus low-reward distractor, since these were the most frequent trial-types and hence provided the richest dataset. We first analysed saccade latency for trials in which participants' first saccade was in the direction of the coloured distractor (defined by the saccade vector having an angular deviation $<30^\circ$ to the left or right of that distractor; see Pearson et al., 2016): see Figure 4a. ANOVA with factors of alcohol group and distractor type revealed marginal main effects of alcohol group, $F(1,56) = 3.91, p = .053, \eta_p^2 = 0.07$, and distractor type, $F(1,56) = 3.71, p = .059, \eta_p^2 = 0.06$, with a significant interaction, $F(1,56) = 8.16, p = .006, \eta_p^2 = 0.13$. Analysis of simple effects revealed that, for participants in the placebo condition, first saccades to the distractor had lower latency when the distractor signalled high versus low reward, $t(28) = 3.39, p = .002, d_z = 0.63$, whereas latency did not differ significantly as a function of reward for the alcohol condition, $t(28) = 0.65, p = .52, d_z = 0.12$. Orthogonal contrasts showed that alcohol (relative to placebo) resulted in greater latency of

first saccades to the high-reward distractor, $t(56) = 2.80, p = .007, d = 0.74$, but had no significant effect on latency for the low-reward distractor, $t(56) = 1.15, p = .25, d = 0.30$.

Figure 4. (a) Mean latency of first saccades towards a colour-singleton distractor signalling high vs. low reward, for participants who had consumed placebo or alcohol. (b) Proportion of first saccades towards the high- vs. low-reward distractor [distractor-saccade proportion, DSP] as a function of saccade latency, for placebo and alcohol conditions. Mean first saccade latencies and DSP were calculated separately for each tertile of individual-participant saccade latency distributions using the Vincentizing procedure (Ratcliff 1979): symbols on each line represent tertiles 1, 2, and 3. Error bars show standard error of the mean. (c) Results of permutation analysis of saccade latency and DSP. Contrasts refer to a series of tests in which observed DSP for a particular group (alcohol/placebo), distractor type (high/low reward), and tertile (1 to 3) was compared to an estimate of the other group's DSP at the same saccade latency (see text for details). For example, the contrast labelled "obs

A1 vs est P1/2" should be read as "**observed** DSP for **Alcohol** condition, latency tertile **1**, versus an **estimate** of DSP for the **Placebo** condition at the same saccade latency derived from data from their tertiles **1** and **2**". The "Overall" distribution represents a combined analysis across the other four contrasts. The left part of the figure (red) relates to trials with a high-reward distractor; the right (blue) to trials with a low-reward distractor. The x-axis shows reduction in DSP following alcohol: positive values indicate less distraction (better performance) in the alcohol group relative to placebo. Each plot shows the density of 50,000 comparisons under the alternative and null hypotheses. The light-shaded region in each plot shows values between the 2.5th and 97.5th percentiles: if this shaded area lies above zero (dashed line), then the alcohol-driven reduction in DSP is significant at the $p < .05$ (two-tailed) level. The exact p -value for each contrast is shown on each plot.

A follow-up analysis examined the direction of the first saccade as a function of saccade latency, using the Vincentizing procedure (Ratcliff 1979). Specifically, we calculated the proportion of first saccades going towards the colour-singleton distractor (henceforth referred to as distractor-saccade proportion, DSP) for each tertile of each individual's saccade latency distribution separately for high- and low-reward distractors. Figure 4b shows that, for each condition, DSP was greatest for the fastest tertile of saccades, and decreased across subsequent tertiles. Moreover, the effect of reward on first-saccade direction—given by the difference in DSP for the high- vs low-reward distractor—was most pronounced for the fastest tertile of saccades in both the placebo and alcohol groups (replicating previous findings: Failing et al., 2015; Pearson et al., 2016). In general, first saccade latency was greater in the alcohol group than the placebo group (consistent with analyses reported above). Notably, DSP for the low-reward distractor in the alcohol group was consistent with the DSP observed in the placebo group for a given saccade latency (i.e., the lines for the low-reward distractor conditions appear superimposed in Figure 4b). By contrast, DSP for the high-reward distractor for the alcohol group appeared lower than that expected for the placebo group for a given latency. This would suggest that an acute dose of alcohol decreased distraction by high-reward stimuli, even after controlling for the slower saccade latencies induced by the pharmacological effects of alcohol.

In order to test whether the group differences in DSP for the high-reward distractor were statistically significant at matched saccade latencies, we analysed the Vincentized data using a form of permutation test. This test estimated DSP at a common saccade latency for each group and compared these estimated values. One such analysis used data from the first and second tertiles of the placebo condition to estimate DSP at the mean latency of the first tertile of the alcohol condition (which lay between these values: see Figure 4b). Specifically, this analysis paired each participant in the placebo group with a participant in the alcohol group (so participant PlacP1 in the placebo group was paired with participant AlcP1 in the alcohol group, participant PlacP2 with participant AlcP2, and so on). We then used the DSP for tertiles 1 and 2 of the high-reward condition for participant PlacP1 to estimate (by linear interpolation) DSP at the saccade latency of tertile 1 of the high-reward condition for participant AlcP1 (and the same for participant-pairs PlacP2/ AlcP2, PlacP3/ AlcP3 etc). We

could then compare the mean *observed* DSP at the first-tertile latency for the alcohol group with the mean *estimated* DSP for the identical latency for the placebo group. The participants within each group were then randomly shuffled, and the estimation process was repeated (so now placebo participant PlacP1 might be paired with alcohol participant AlcP17, PlacP2 with AlcP4, etc). This process was repeated 30,000 times, yielding a distribution of mean differences between the placebo and alcohol groups under the alternative hypothesis (H1) that there was a meaningful difference between groups.

Next we used a similar procedure to generate a distribution of values under the null hypothesis (H0) of no systematic difference between groups. Now for each permutation we assigned a randomly chosen half of participants to 'group 1', and the other half to 'group 2', ignoring their true alcohol/placebo group assignment. We then repeated the process of interpolation described above (so now participant PlacP1 in group 1 might be paired with PlacP24 in group 2; PlacP2 with AlcP4; AlcP1 with AlcP14; and so on), to yield a contrast between groups under the hypothesis that group labels are exchangeable.

This approach was used to create distributions of "placebo minus alcohol" DSP values for permutations under the alternative hypothesis (actual group assignments) and null hypothesis (random group assignments) for a range of contrasts at different saccade latencies (Supplementary Figure S1 shows these distributions). To analyse the resulting data, for each contrast we drew 50,000 random pairs of samples from the alternative and null distributions, and calculated the difference between them. Figure 4c shows the distribution of these difference scores for four contrasts, which reflect the reduction in DSP following alcohol; the proportion of scores above zero yields a *p*-value for the difference between groups. For the high-reward distractor, scores for the reduction in DSP under alcohol were generally positive: for a given saccade latency, participants in the alcohol condition were less likely to make a first saccade to the high-reward distractor than were participants in the placebo condition. For the low-reward distractor, scores tended to be closer to zero, indicating similar DSP for alcohol and placebo conditions when controlling for saccade latency. We also calculated an 'overall' comparison across the four contrasts for each reward type shown in Figure 4c, by calculating (for 50,000 cases) the difference in the mean of samples drawn from each of the alternative distributions versus the mean of samples from the null distributions. These latter 'main effect' analyses revealed that there was a significant reduction in DSP for the high-reward distractor for the alcohol group relative to the placebo group when controlling for saccade latency, $p = .002$, with a significant ($p = .04$) difference also observed for the individual contrast at the latency of the placebo group's second tertile. By contrast, no significant difference in DSP was observed for the low-reward distractor when controlling for saccade latency ($ps \geq .462$).

505 In the current study we examined whether an acute dose of alcohol would influence
506 attentional distraction by reward-related stimuli. In the task used here, critical distractor
507 stimuli signalled whether high or low reward was available on each trial – but attending to
508 these stimuli was counterproductive since if participants looked at the reward-signalling
509 distractor, the reward was omitted. Under these conditions, we found that the influence of
510 reward on attentional capture was significantly reduced in participants who had consumed
511 alcohol relative to a placebo group. Specifically, participants in the alcohol group were less
512 likely than those in the placebo group to look at a distractor signalling high reward, but the
513 groups did not differ significantly in distraction by a low-reward-signalling distractor. The
514 difference in distraction on high-reward trials meant that participants in the alcohol group
515 earned more points (and hence money) overall than those in the placebo group.

516 For participants in the placebo group, distraction was most evident in the most rapid eye-
517 movements following display onset (Figure 4b), replicating previous findings (Failing et al.,
518 2015; Pearson et al., 2016). That is, very rapid saccades were most likely to be directed
519 erroneously to salient distractors, suggesting that these rapid saccades often reflected an
520 ‘impulsive’ attentional response, as compared with slower, ‘controlled’ saccades which were
521 more likely to go towards the target. Notably, participants in the alcohol condition showed
522 an overall increase in saccade latencies. This increased latency did not reflect a simple delay
523 in the pattern of participants’ eye-movements: if it had, then data for the alcohol condition in
524 Figure 4b would have been a horizontal (rightward) translation of data for the placebo
525 condition. Instead, increased saccade latencies in the alcohol group were accompanied by a
526 decrease in the frequency of distraction (i.e., data in Figure 4b were translated downwards
527 and to the right). This implies that participants who had consumed alcohol were less likely to
528 make fast impulsive saccades—which tend to go to the distractor—and relatively more likely
529 to make slower, controlled saccades to the target. Consistent with the idea that alcohol-
530 driven differences in saccade behaviour did not simply reflect a blanket delay, we found that
531 these differences were particularly pronounced when the display contained a high-reward
532 distractor. Indeed, when we used permutation analyses to compare the proportion of
533 saccades to the distractor at matched saccade latencies, we found that participants in the
534 alcohol group were significantly less likely than placebo participants to be captured by the
535 high-reward distractor.

536 Our finding of reduced distraction following an acute dose of alcohol is broadly consistent
537 with the theory of alcohol myopia. More specifically, it is consistent with an interpretation of
538 this theory wherein alcohol reduces overall perceptual processing capacity (Erthal et al.,
539 2005), so that a greater proportion of available attentional resources must be allocated to
540 execute the focal task (find the diamond), leaving fewer resources for processing salient
541 distractors. This interpretation in turn follows from the *perceptual load theory* of selective

attention, which asserts that distractors will be processed only to the extent that sufficient perceptual resources are available (Lavie et al. 2004; Lavie 2005).

While a load-based interpretation of alcohol myopia theory can account for reduced distraction following alcohol consumption, it anticipates a general reduction in distraction across all types of distractors, which is at odds with our finding that the effect of alcohol was greater for the high-reward distractor than the low-reward distractor. One possibility is that this pattern reflects a floor effect: the placebo group looked at the low-reward distractor on only ~10% of trials (versus ~40% for the high-reward distractor), and hence there is less room for an alcohol-driven reduction on low-reward trials. Notably, however, we also found that the effect of alcohol on saccade latency was significantly greater for high-reward trials than low-reward trials (Figure 4a), and this cannot be so easily ascribed to a floor effect (as there was no physical limit preventing an increase in latency on low-reward trials). Taken together, our findings suggest that beyond any influence on overall processing capacity, alcohol may exert a selective effect on processing of high-reward distractors. However, the current data do not allow us to determine whether this selective effect is a consequence of reward specifically (high-reward distractors signal greater reward than low-reward distractors), or incentive salience more generally (high-reward distractors signal a more motivationally significant event) – though previous findings of an alcohol-related reduction in distraction by aversive stimuli (Curtin et al. 2001; Erthal et al. 2005) suggest support for the latter account.

As noted above, our results are broadly consistent with a load-based interpretation of alcohol myopia. By contrast, the current findings are at odds with an interpretation in which the effect of alcohol is to restrict attention to the most salient cues in the environment (Josephs and Steele 1990; Steele and Josephs 1990). Data from the placebo group show that the high-reward distractor was more salient (more attention-grabbing) than the low-reward distractor (in line with results of many previous studies using this task: e.g., Failing et al. 2015; Le Pelley et al. 2019; Pearson et al. 2015, 2016; Watson et al. 2019a). Hence if alcohol were to increase the likelihood of capture by highly salient stimuli, then we should expect alcohol to increase capture by the high-reward distractor relative to its effect on the low-reward distractor (therefore producing a performance *decrement* on high-reward trials relative to low-reward trials): the opposite of the pattern observed.

Looking beyond alcohol myopia theory, others have argued that alcohol affects response inhibition and the ability to exert top-down control over behaviour. Evidence for this idea comes primarily from studies using the go/no-go task where participants must push a key in response to a frequent go stimulus and not push a key upon presentation of an infrequent no-go stimulus. A number of studies have demonstrated that relative to a placebo control group, participants given a low dose of alcohol respond more frequently in the presence of the no-go signal, while not showing differential responding to go signals (Dry et al. 2012;

Mulvihill et al. 1997; Curtin and Fairchild 2003; Easdon et al. 2005; see for review: Schweizer and Vogel-Sprott 2008). In contrast however, findings from studies using the anti-saccade task do not fit with this idea that alcohol intoxication leads to behavioural disinhibition. In this attentional control task, a stimulus appears on one side of the screen and the participant must inhibit a reflexive saccade to that stimulus and instead make a saccade to the opposite side of the screen. A number of studies have reported that an acute alcohol dose did not lead to more failures in oculomotor inhibition (Vorstius et al. 2008; i.e. reflexive saccades to the visual stimulus; Roche and King 2010; Schmitt et al. 2013) although consistently, alcohol does slow saccade latencies (the time taken to initiate the saccade). Similarly, our current results do not fit with the idea that alcohol impairs top-down inhibitory control. We have recently demonstrated that performance in this task is sensitive to variations in top-down control: if participants' executive resources are otherwise engaged in a demanding working-memory task, then distraction by reward-related stimuli is significantly increased (Watson et al. 2019a). Hence if alcohol similarly depleted executive resources and the capacity for top-down behavioural control, then we would likewise expect participants to show increased attentional distraction by reward-related stimuli following the alcohol dose, which is the opposite to the pattern of reduced distraction that we observed. Our findings thus further call into question the idea that alcohol reduces top-down control. Instead, the current and previous findings from various tasks fit with the suggestion that alcohol reduces the capacity to attend to information from multiple sources (Steele and Josephs 1990; Fillmore and Vogel-Sprott 1997; Fillmore and Vogel-Sprott 1998; Marczinski and Fillmore 2003; George et al. 2005; Schweizer and Vogel-Sprott 2008). If attention is focused on the primary task (find the diamond, push a key in response to a go cue, saccade to the opposite side of the screen from a visual stimulus), then alcohol seems to reduce the extent to which competing information receives attentional prioritisation.

That said, we note a potential alternative interpretation of the results of the current study. It is possible that participants in the alcohol group showed less evidence of reward-related distraction because the monetary rewards were less motivating to them. Consider an extreme example: if rewards had no motivational value, then there would be no difference in the salience of a high- versus low-reward distractor, and hence no difference in the extent to which they captured attention. While this idea could potentially account for our findings, we believe it is unlikely, since prior evidence suggests that alcohol either has no effect on reward sensitivity (Fillmore and Vogel-Sprott 2000; Reynolds et al. 2006; Dougherty et al. 2008; Bidwell et al. 2013), or may even *increase* reward sensitivity (Euser et al. 2011; Bidwell et al. 2013; Lyvers et al. 2015). Future research using the current procedure could test this possibility more directly, for example by appending an operant conditioning task to measure participants' willingness to work for monetary rewards (cf., Treadway et al. 2009) as a function of alcohol consumption.

The alcohol dose used in the current study was relatively low, with breath alcohol concentrations remaining stable at around 0.050 g/210L, throughout the visual search task. This is the maximum concentration of breath alcohol legally allowed when driving in Australia; many countries have a higher limit (e.g., 0.080 g/210L in the United States). Previous studies have reported dose-dependent effects of alcohol on behavioural tasks (Dry et al. 2012; Roche and King 2010; see for review: Schweizer and Vogel-Sprott 2008) and it is possible that we would have observed different results with higher dose of alcohol. However, it is worth stressing that even at a relatively low dose the alcohol group reported feeling intoxicated, and we observed significant effects on attention and behaviour even though many of the participants would have been legally able to drive a car. In our procedure—in which attending to salient stimuli was counterproductive, since it caused omission of reward—the alcohol-driven reduction in distraction benefitted participants in the alcohol group such that they earned more points. In real-world contexts, however, the situation will typically be quite different. Presumably we have evolved an attentional system that prioritizes rapid and automatic processing of highly salient events—that is, an ‘impulsive’ system—because rapid detection of such events confers advantages in terms of maximising rewards and avoiding danger. Evidence from the current task suggests that alcohol interferes with the efficient operation of this system, reducing the efficiency of detecting and responding to these high-salience events. In situations which may require rapid changes in behaviour in response to incoming sensory information (for example, while driving) this reduction in processing efficiency could potentially have catastrophic consequences.

In summary, this study examined whether a dose of alcohol would influence distractibility by reward-related stimuli in a visual search task, when participants were motivated to ignore these stimuli. In line with alcohol myopia theory, participants assigned to the alcohol group showed reduced distraction by distractors signalling high reward (and consequently earned a larger monetary bonus) than participants in the placebo group. These results suggest that alcohol intoxication reduces the capacity to attend to information in the environment that is not directly relevant to the task at hand. In the current context, this reduced distraction conferred a performance advantage, but in many situations where attentiveness is required, reduced distraction could lead to negative outcomes.

Conflict of Interest Statement

On behalf of all authors, the corresponding author states that there is no conflict of interest.

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