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1 Light social drinkers are more distracted by irrelevant information from an induced
2 attentional bias than heavy social drinkers

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20 It is well established that alcoholics and heavy social drinkers show a bias of attention towards
21 alcohol-related items. Previous research suggests that there is a shared foundation of attentional bias,
22 which is linked to attentional control settings. Specifically, attentional bias relates to a persistent
23 selection of a Feature Search Mode which prioritises attentional bias-related information for selection
24 and processing. However, no research has yet examined the effect of pre-existing biases on the
25 development of an additional attentional bias. This paper seeks to discover how pre-existing biases
26 affect the formation of a new, additional attentional bias. 25 heavy and 25 light social drinkers, with
27 and without a pre-existing bias to alcohol related items respectively, had an attentional bias towards
28 the colour green induced via an information sheet. They then completed a series of one-shot change
29 detection tasks. In the critical task, green items were present but task-irrelevant. Irrelevant green items
30 caused significantly more interference for light than heavy social drinkers. This somewhat counter
31 intuitive result is likely due to heavy drinkers having more experience in exerting cognitive control
32 over attentional biases, something not previously observed in investigations of the effects of holding
33 an attentional bias. Our findings demonstrate for the first time that an established attentional bias
34 significantly modulates future behaviour.

35

36 Key Words:

37 Attentional bias, social drinkers, cognitive bias, change detection, distraction

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39 Attentional bias is a phenomenon wherein certain items are preferentially processed at the cost of
40 others (Macleod, Mathews, & Tata, 1986). It is commonly studied in relation to addiction (Field &
41 Cox, 2008), where the development of addictive behaviours is consistently found to coincide with the
42 development of an attentional bias towards addiction-related stimuli (Boyer & Dickerson, 2003;
43 Constantinou et al., 2010; Jones, Jones, Smith, & Copley, 2003; Lusher, Chandler, & Ball, 2004;
44 Townshend & Duka, 2001; Yaxley & Zwaan, 2005). These biases appear to be causally linked to
45 addictive behaviours. For example, a larger reduction in alcohol-related attentional bias during
46 treatment is related to continued abstinence of alcohol consumption following release from
47 rehabilitation centres (Cox, Hogan, Kristian, & Race, 2002; Flaudias et al., 2013).

48 Much of what is known about attentional biases stems from research comparing substance abusers
49 and addicted populations with healthy controls across a variety of paradigms, such as the modified
50 Stroop (Lusher et al., 2004; Sharma, Albery, & Cook, 2001), dot probe (Noel et al., 2006) and dual
51 task paradigms (Waters & Green, 2003). These investigations have established that people who are
52 dependent on or abuse alcohol have consistently faster reaction times towards task-relevant alcohol-
53 related cues – i.e., in a flicker induced change blindness task where there is an alcohol-related change
54 between two images (Jones, Bruce, Livingstone, & Reed, 2006; Jones, Jones, Blundell & Bruce,
55 2002), and slower reaction times when alcohol-related cues interfere with task goals – i.e. in a Stroop
56 colour-naming task where alcohol-related content distracts from the primary goal of naming colours
57 (Cox, Blount, & Rozak, 2000; Johnsen, Laberg, Cox, Vaksdal, & Hugdahl, 1994) than control
58 participants. These studies have yielded valuable data on how attentional biases manifest in addicted
59 and at-risk individuals. However, despite this, there are some methodological issues regarding the
60 samples used in these investigations and the legitimacy by which these findings can be attributed to
61 social drinkers.

62 Specifically, the use of alcoholics is problematic because of neurophysiological differences between
63 addicts and the healthy population (Baler & Volkow, 2006; Cardenas, Studholme, Gazdzinski,
64 Durazzo, & Meyerhoff, 2007; George, Potts, Kothman, Martin, & Mukundan, 2004; Goldstein &
65 Volkow, 2011; Medina et al., 2008; Thompson et al., 2004). Long term alcohol abuse is related to a

66 detrimental effect on brain structures relating to cognitive control and executive function such as the
67 prefrontal cortex (George et al., 2004; Goldstein & Volkow, 2011; Medina et al., 2008). Thus,
68 observed differences in attention between abusers and healthy controls may be due to damage to
69 essential neural networks. It should be noted that this has been examined in some studies, with
70 differences in reaction time on attention-demanding tasks between inpatient alcoholics and matched
71 controls only occurring when stimuli were alcohol-related, suggesting a specific issue with addiction-
72 related information processing (Johnsen, Laberg, Cox, Vaksdal & Hugdahl, 1994; Stetter,
73 Ackermann, Bizer, Straube & Mann, 1995). Furthermore, the impact on frontal executive regions of
74 other drugs of abuse – specifically cocaine and heroin – has been investigated, finding no evidence of
75 an associated impact on attention (Pau, Lee & Chan, 2001; Smith, Jones, Bullmore, Robbins &
76 Ersche, 2014). Nevertheless, if the cause of the behavioural differences in addicted populations is due
77 to differences in the brain, the findings observed within these populations cannot be compared to
78 healthy, social-drinking controls. Furthermore, the experimental and control groups both across and
79 sometimes between studies are rarely well matched for age, educational attainment, working memory
80 capacity and methodologies (Goldstein et al., 2004).

81 Many studies have addressed these issues by comparing heavy and light social drinkers from
82 university samples. Some of these investigations have found group differences between heavy and
83 light social drinkers using alcohol Stroop (Fadardi & Cox, 2008), pictorial Stroop (Bruce & Jones,
84 2004) and flicker induced change blindness tasks (Jones et al., 2002). Although these findings
85 sometimes mirror those found in addicted populations, these differences are not always observed. For
86 example, Sharma et al. (2001) compared three groups of drinkers on a modified Stroop task; problem
87 (where excessive drinking has a negative impact on day-to-day life), heavy (where alcohol
88 consumption does not impact day-to-day life) and light. While a Stroop effect was found in problem
89 compared to heavy and light social drinkers, there was no difference between the heavy and light
90 social drinkers.

91 Other research focuses on individual differences. Field et al. (2011) investigated the link between
92 alcohol consumption and expectancy to receive alcohol in an eye-tracking task. Here, heavy and light

93 social drinkers were informed of the probability of receiving an alcoholic drink following each trial.
94 Heavy social drinkers displayed an attentional bias regardless of expectation (analysed via eye
95 movements to alcohol-related cues), however only the 100% expectation condition produced this
96 effect in light social drinkers. Another study found that only social drinkers with high levels of
97 alcohol craving showed evidence of increased approach towards alcohol-related cues in a dot probe
98 task (Field, Mogg, & Bradley, 2005). These results suggest individual differences in subjective
99 craving play a key role in alcohol-related attentional biases, but not necessarily in alcohol
100 consumption levels for social drinkers.

101 Finally, alcohol preload before testing increases attentional bias towards both alcohol- (B. T. Jones &
102 Schulze, 2000; Schoenmakers, Wiers, & Field, 2008) and cocaine-related items (Montgomery et al.,
103 2010). Similar results were found when participants were primed by an alcoholic or placebo drink,
104 then asked to perform an Eriksen Flanker task superimposed on either a neutral or alcohol-related
105 background, while being scanned via fMRI (Nikolaou et al., 2013). While a high dose of alcohol
106 reduced overall neural activity (and activity in both medial and dorsal PFCs), a low dose of alcohol
107 increased latency when the flanker task was completed on alcohol-related backgrounds, suggesting it
108 had caused an increase in alcohol-related attentional bias.

109 Taken together, these findings suggest that previous methodologies, with the possible exception of the
110 dot probe paradigm (Field, Mogg, Zetteler, & Bradley, 2004; Townshend & Duka, 2001), are not
111 sensitive enough to detect group differences in attentional bias changes related to alcohol
112 consumption habits. Nevertheless, while the dot probe paradigm is a more direct measure of the locus
113 of attention than the Stroop or Dual Task paradigms, it is still not a direct measure of attentional
114 orienting, and hence of attentional bias though it does suggest an alcohol-related attentional bias in
115 heavy social drinkers over light social drinkers.

116 Previously, it has been found that it is possible to induce an attentional bias towards an arbitrary
117 stimulus - a particular colour - in a group of healthy participants who were provided with a single
118 information sheet about the experiment. The bias was sustained for at least two weeks and affected

119 behaviour when bias-related items were both relevant and irrelevant to task demands (Knight, Smith,
120 Knight & Ellison, 2016). The paradigm used was also a more direct measure of attentional orienting,
121 since it allowed for the calculation of sensitivity to detect bias-related incidents free from emotional
122 and neuropharmacological confounds. These findings therefore suggest that there is a cognitive
123 foundation of attentional biases, and that these biases can be present and observed in a normative
124 sample (Folk, Remington, & Johnston, 1992). However, the potential relationship between a pre-
125 existing attentional bias and the procurement of an additional attentional bias has not yet been
126 examined. This is important, since those who already possess an attentional bias also must already
127 currently use the neural network involved in this bias. This paper therefore seeks to examine
128 attentional bias in non-addicted individuals further by examining induced biases in a sub-clinical
129 population who are already biased to an emotive stimulus – heavy social drinkers with an alcohol-
130 related attentional bias.

131 The current experiment has two parts; one examining initial inducement of an arbitrary attentional
132 bias, and one examining the effects of the bias when it becomes task-irrelevant. Our first experimental
133 question is therefore: Does a pre-existing attentional bias affect the adoption of an additional bias
134 when attending to induced-bias-related items is behaviourally advantageous? Past research would
135 suggest that this should be equally successful in all participants. In a previous study, we have found
136 that a single information sheet is sufficient to induce a robust and persistent attentional bias towards
137 green stimuli (Knight et al., 2016), mirroring similar results using smoking-related stimuli in non-
138 smokers (Yaxely & Zwaan, 2005). Our second experimental question is: Are heavy or light social
139 drinkers more distracted by their induced arbitrary biases when bias-related stimuli are task-
140 irrelevant? Given that heavy social drinkers hold a pre-existing attentional bias towards alcohol, it is
141 possible that this sample may be even further distracted by irrelevant induced bias-related stimuli.
142 However, given the exploratory nature of this research question, this is purely speculative.

143

144

145 **Assessment of Attentional Bias to Alcohol**

146 **Method**

147 **Participants**

148 124 undergraduate students in their first or second year of an Applied Psychology course at Durham
149 University (33 male; aged 18-37, M: 20.196, SD 3.328) completed an alcohol consumption
150 questionnaire (Time Line Follow Back (Sobell & Sobell, 1992)). Smoking and/or the taking of
151 prescribed or recreational drugs were exclusion criteria. Participants were asked to fill in the
152 questionnaire relating to their alcohol consumption over the past 7 days. They were then asked if this
153 was reflective of an average week, and if not, were asked to complete a section modified Time Line
154 Follow Back regarding their average alcohol consumption. Participants also checked a box to state
155 they were not nor had previously been treated for any alcohol misuse disorder. Participants were then
156 ranked from highest to lowest alcohol consumption based on total units consumed. Non-drinkers were
157 removed, along with one participant whose reported weekly alcohol consumption was above 3
158 standard deviations from the mean. Ultimately, 50 participants (12 male, aged 18-22, M: 20.08, SD:
159 1.586) with normal or corrected to normal vision and no colour blindness took part. The sample
160 consisted of the 25 heaviest and 25 lightest social drinkers. Heavy social drinkers had an average
161 weekly consumption of 56.86 units (SD: 21.409), light social drinkers had an average weekly
162 consumption of 7.984 units (SD: 4.254). These differed significantly: $t(48) = -11.196$, $p < .001$, $r =$
163 $.8504$. No cases of heavy or light social drinkers fell outside mean $\pm 3SD$, thus no further outliers
164 were present. All participants gave their informed consent with the approval of Durham University
165 Ethics Advisory Committee and were provided with university course credits for their time.

166 **Apparatus**

167 All experimental stimuli were programmed in C++ using Borland C++ builder and produced via a
168 ViSaGe box and custom graphics card (Cambridge Research Systems, Rochester, England). They
169 were displayed using a 19" Sony Triniton monitor with a resolution of 1024x768 and a refresh rate of
170 100Hz. Responses were collected via a custom-made parallel-port two-button button box.

171 **Stimuli & Procedure**

172 A white fixation cross situated in the center of a black screen (0.704 x 0.704° visual angle) was
173 presented for 1000ms, followed by a square test array (width 10.2cm) comprising four different
174 images of either alcohol-related or neutral images (visual angle: 2° x 2.5°) for 750ms. This was
175 masked via a blank screen for 100ms before reappearing. Stimuli remained present until a response
176 was made. On 20% of trials, all images were originally alcohol-related and one changed into a
177 different alcohol-related image (Alcohol-Alcohol Trials), on 20% of trials all images were originally
178 alcohol-related and one changed into a neutral image (Alcohol-Neutral Trials), on 20% of trials all
179 images were originally neutral and one changed into an alcohol-related image (Neutral-Alcohol
180 Trials), on 20% of trials all images were originally neutral and one changed into a neutral image
181 (Neutral-Neutral Trials). On the final 20% of trials no change occurred (No Change Trials). There
182 were 225 trials in total split into three blocks. Participants were asked to detect whether a change had
183 occurred as quickly but accurately as possible. Perceived Change trials were reported by pressing the
184 right-hand button on a custom-made parallel-port two-button button box. Perceived No-Change trials
185 were reported by selecting the left-hand button.

186 **Results**

187 Sensitivity measured via d' was entered into a 2 (Drinker: Heavy/Light) x 4 (Trial Type: Alcohol-
188 Alcohol/Alcohol-Neutral/Neutral-Alcohol/Neutral-Neutral) mixed factor ANOVA. See Table 1 for
189 mean accuracy across all types of trial. There was no main effect of drinker ($F(1,48) = 1.759$, $MSE =$
190 $.183$, $p = .191$, $r = .188$), however Trial Type and Drinker interacted: $F(3,144) = 10.032$, $MSE = .056$,
191 $p < .001$, $r = .254$. Bonferroni-corrected independent t-tests comparing Heavy versus Light drinkers
192 for each trial type revealed a significant difference in Neutral-Alcohol trials: $t(48) = -3.263$, $p = .002$,
193 $r = .426$. Here, d' scores of heavy drinkers were higher by an average of .4326. See Figure 1.

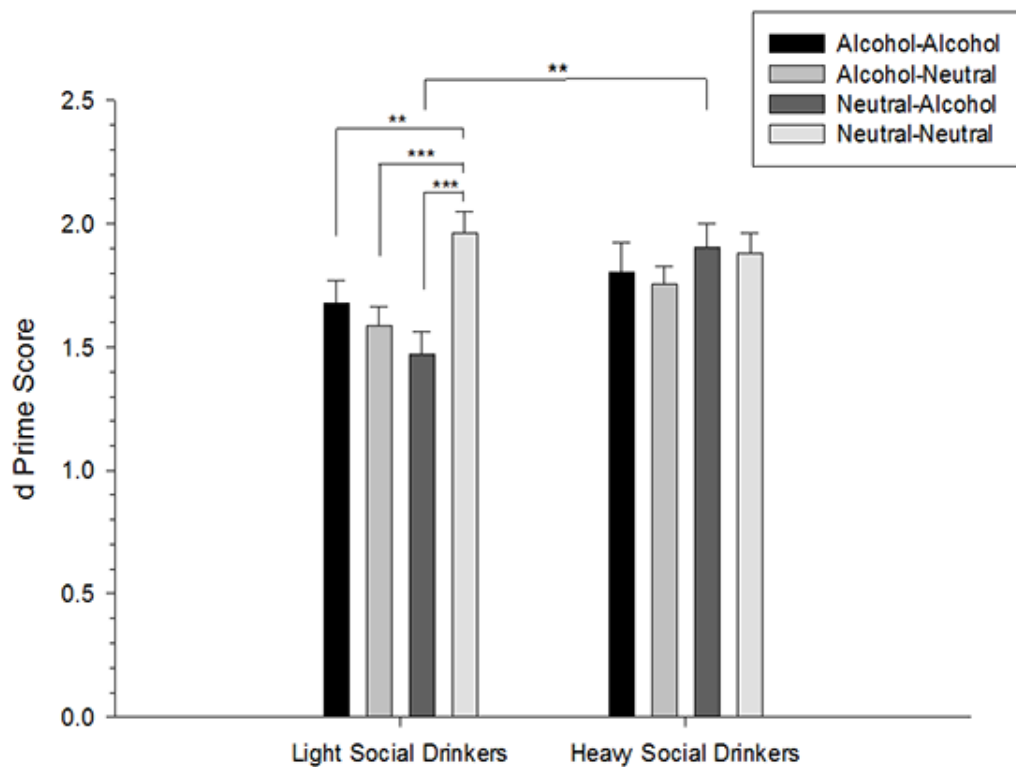
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195

196 **Table 1**

197 *Mean hit/miss rate in the Alcohol Task across all types of change trial, and mean correct*
 198 *rejection/false-alarm rates for no-change trials.*

<i>Drinker</i>	<i>Trial Type</i>	<i>Hit Rate</i>	<i>Miss Rate</i>
Light Social Drinkers	Alcohol-Alcohol	76.79	23.21
	Alcohol-Neutral	74.93	25.06
	Neutral-Alcohol	80.40	19.60
	Neutral-Neutral	78.27	21.73
	No Change	83.80	16.20
Heavy Social Drinkers	Alcohol-Alcohol	67.60	32.40
	Alcohol-Neutral	66.67	33.33
	Neutral-Alcohol	60.80	39.20
	Neutral-Neutral	77.07	22.93
	No Change	86.60	13.40



199 **Fig. 1:** Pre-existing alcohol-related attentional bias in light versus heavy social drinkers. Higher d'
 200 indicates increased sensitivity to change. Sensitivity is higher in heavy social drinkers than light social
 201 drinkers when an alcohol-related image appears amongst neutral images. For light social drinkers,
 202 sensitivity is highest when a novel neutral image appears amongst other neutral images. Error bars show
 203 standard error of the mean. *Note:* ** $p < .005$, *** $p < .001$
 204

205 **Discussion**

206 Heavy drinkers' attention was captured by the novel alcohol-related item, increasing their ability to
207 accurately detect the appearance of a novel, alcohol-related item. This result is consistent with the
208 conclusion that heavy social drinkers hold a pre-existing attentional bias towards alcohol-related
209 items. Consistent with previous studies, this increase in sensitivity was not observed in light social
210 drinkers (Field et al., 2004; Jones et al., 2003; Townshend & Duka, 2001), suggesting no alcohol-
211 related attentional bias in our light social drinkers. Furthermore, the group difference between our
212 heavy and light social drinkers, and the observation that not only did light social drinkers do not react
213 when a novel alcohol-related item appears, but they were most sensitive at spotting novel neutral
214 items appearing suggests that this task did not also induce an alcohol attentional bias in our light
215 social drinkers. Therefore, it can be concluded that our samples are valid for addressing our
216 experimental questions.

217 **Attentional Bias Inducement Task**

218 **Method**

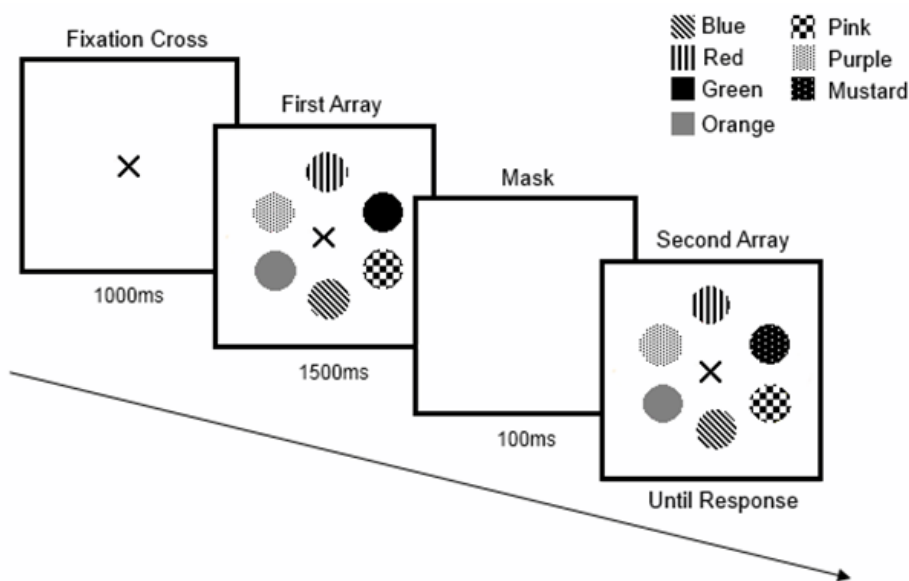
219 The 50 participants who completed the alcohol change detection task also completed the attentional
220 bias inducement task. The apparatus was the same as that used for the alcohol change detection task.
221 The attentional bias inducement task was conducted in the same experimental session as the alcohol
222 change detection task.

223 **Stimuli, Apparatus & Procedure: Attentional Bias Inducement Task**

224 A mixed design was used. Following the completion of the alcohol attentional bias experiment, all
225 participants carried out a second change detection task, after replicating the methodology used to
226 induce an attentional bias to green items in Knight et al. (2016). As with the alcohol task, the
227 Attentional Bias Inducement Task was also programmed using Borland C++ builder and presented on
228 a 19" Sony Triniton monitor with a resolution of 1024x768 pixels and a refresh rate of 100Hz using a
229 VSG ViSaGe box and custom graphics card (Cambridge Research Systems, Rochester, England).

230 To induce the attentional bias towards green, information and consent forms were used which
 231 informed participants that they were carrying out an experiment investigating how the human visual
 232 system perceives and processes the colour green, and used the word *green* several times. A white
 233 fixation cross situated in the centre of a black screen (0.704 x 0.704° visual angle) preceded the test
 234 array consisting of a circular (radius 5.1cm) composition of six circles (2.5° x 2.5° visual angle) each
 235 of which was one of 8 different equiluminous colours (green, red, blue, pink, purple, grey, mustard
 236 or orange, all 34 cd/m2). The mask was a black screen.

237 The white fixation cross was presented for 100ms followed by the initial stimulus array for 1500ms.
 238 The presentation time of the initial array differed from the alcohol change detection task and was
 239 proportional to the number of stimuli presented to avoid ceiling effects. This array was masked by a
 240 blank screen for 100ms before reappearing until a response was made. On 25% (45 trials) of trials a
 241 green item was present and changed colour (Congruent Change Trials), on 25% of trials a green item
 242 was present in the display but a different item changed colour (Incongruent Change Trials), on 25% of
 243 trials no green item was present and one of the objects changed colour (Neutral Change Trials) and on
 244 25% of trials a green item was present but no change occurred (No Change Trials). Trials were
 245 presented in a random order. See Figure 2 for an illustration of a typical trial. Participants completed 3
 246 blocks of 60 trials with a 5 minute break between each block.



248 **Fig 2:** Procedure of Bias Experiment. A fixation cross was presented for 1000ms, followed the first
 249 array for 1500ms. This was then masked for 100ms before reappearing, where participants had to make
 250 their response as quickly but as accurately as possible, using the index finger of each hand.

251 **Results**

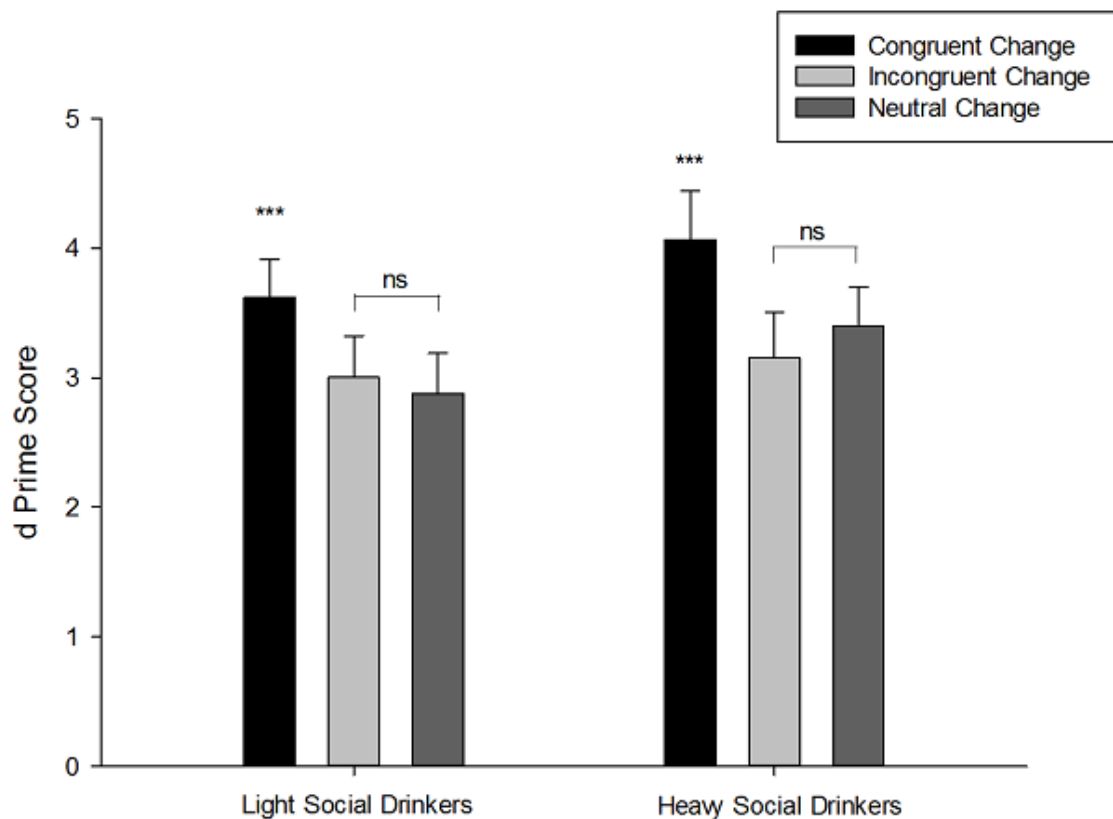
252 d' was entered into a 2 (Drinker: Heavy/Light) x 3 (Trial Type: Congruent Change/Incongruent
 253 Change/Neutral Change) mixed factor ANOVA. No change trials were used to calculate d' , thus were
 254 analysed within the ANOVA but not as an additional factor, see Table 2 for mean accuracy across all
 255 types of trial. There was a significant effect of Trial Type: $F(2,96) = 11.848$, $MSE = 1.183$, $p < .001$.
 256 Bonferroni-corrected pairwise comparisons revealed that d' scores in Congruent Change trials were
 257 higher than Incongruent Change trials (mean difference .760, $p < .001$, $r = .783$) and Neutral Change
 258 trials (mean difference .702, $p = .003$, $r = .454$) – see Fig. 3. Thus, participants were more sensitive to
 259 detecting changes to green stimuli than other stimuli, suggesting a successful induced bias towards the
 260 colour green. There was no effect of drinker: $F(1,48) = .812$, $MSE = 2.147$, $p = .372$ and no
 261 interaction between trial and drinker: $F(2,36) = .636$, $MSE = 1.183$, $p = .465$.

262 **Table 2**

263 *Mean hit rate in the Attentional Bias Inducement Task across all types of trial for Heavy and Light*
 264 *social drinkers and mean correct rejection/false-alarm rates for no-change trial when a green*
 265 *stimulus was either present or absent*

<i>Drinker</i>	<i>Trial Type</i>	<i>Hit Rate</i>	<i>Miss Rate</i>
Light Social Drinkers	Congruent Change	89.24	10.76
	Incongruent Change	75.64	24.36
	Neutral Change	75.65	24.35
	No Change (green present)	92.74	7.26
	No Change (green absent)	92.94	7.06
Heavy Social Drinkers	Congruent Change	88.27	11.73
	Incongruent Change	65.51	34.49
	Neutral Change	70.04	29.96
	No Change (green present)	94.25	5.75
	No Change (green absent)	94.87	5.13

266



267 **Fig. 3:** Effect of induced attentional bias towards green on d' in a change detection task. Higher d'
 268 indicates greater sensitivity to change. Sensitivity is higher in Congruent Change trials than both
 269 Incongruent and Neutral change trials. *Note:* *** $p < .001$
 270

271 **Discussion**

272 This experiment investigated if a pre-existing attentional bias affected the procurement of an
 273 additional bias by examining if heavy social drinkers are more easily biased towards a neutral
 274 stimulus than light social drinkers. Evidence has been found of an equally successful inducement of
 275 an attentional bias towards the colour green in both heavy and light social drinkers. Both groups
 276 showed an increase in sensitivity at detecting changes to green stimuli, with a larger effect size
 277 between sensitivity of detecting congruent and incongruent trials than congruent and neutral trials. If
 278 those with a pre-existing attentional bias were more receptive at having additional biases induced,
 279 greater sensitivity at detecting green changes in heavy social drinkers compared to light social
 280 drinkers would be expected. However, our results from heavy and light social drinkers did not differ,
 281 thus it can be concluded that a pre-existing attentional bias does make one more susceptible to the

282 adoption of an additional neutral bias. Nevertheless, whether this extends to additional attentional
283 biases in general remains to be determined. Moreover, as there was no main effect of drinker, nor did
284 drinker interact with trial, it can also be concluded that a potential reactivation of an alcohol
285 attentional bias caused by the first assessment of an alcohol attentional bias did not dampen the
286 development of a further attentional bias in heavy drinkers. Our previous studies have shown that an
287 induced bias can distract participants in a change blindness task in which colour is irrelevant (Knight
288 et al., 2016). A third experiment was therefore run to examine this property in heavy versus light
289 drinkers.

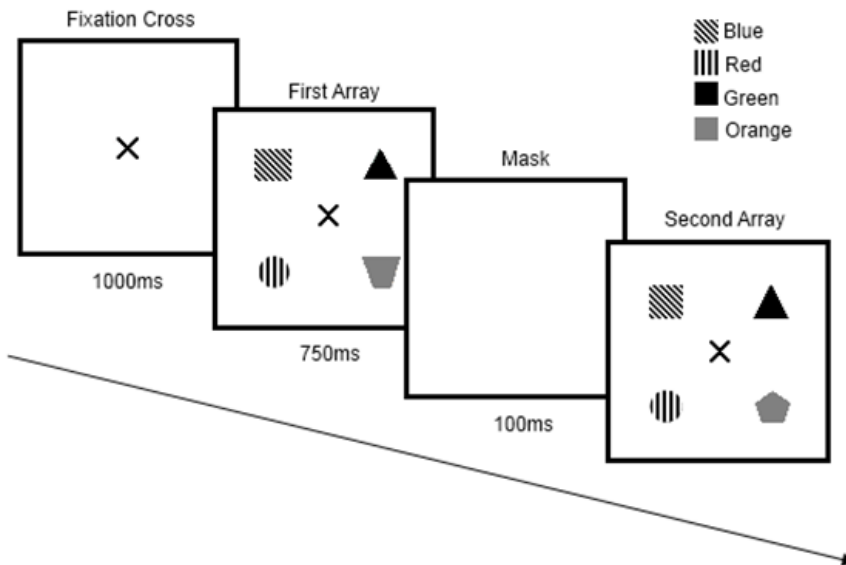
290 **Distractibility from an Induced Attentional Bias**

291 **Method**

292 The same 50 participants completed a third change detection task in the same experimental session. In
293 this case, participants were tasked with detecting changes in shape only – rendering colour irrelevant
294 to the task - and the change never occurred to any green item, rendering the colour green even more
295 irrelevant. Participants and apparatus were the same as those used for previous inducement tasks.

296 **Stimuli & Procedure: Distractibility Test**

297 The fixation cross was presented for 1000ms followed by the test array consisting of a square (width
298 10.2cm) composition of four different shapes (square, circle, triangle, pentagon or trapezium: visual
299 angle: $2.5^\circ \times 2.5^\circ$) for 750ms. Again, this was masked for 100ms before reappearing until a response.
300 On 25% (120 trials) of trials a green shape was present and a different shape changed shape (Green
301 Present Change Trials), on 25% of trials a green item was present but no change occurred (Green
302 Present No-Change Trials), on 25% of trials no green item was present and a shape changed shape
303 (Green Absent Change Trials) and on 25% of trials no green item was present and no change occurred
304 (Green Absent No Change Trials). Trials were presented in a random order. Participants completed 6
305 blocks of 80 trials with a 5 minute break between each block. See Fig. 4 for an illustration of a typical
306 trial.



307

308 **Fig. 4:** Procedure of Shape Experiment. A fixation cross was presented for 1000ms, followed the first
 309 array for 750ms. This was then masked for 100ms before reappearing, where participants had to make
 310 their response as quickly but as accurately as possible, using the index finger of each hand.

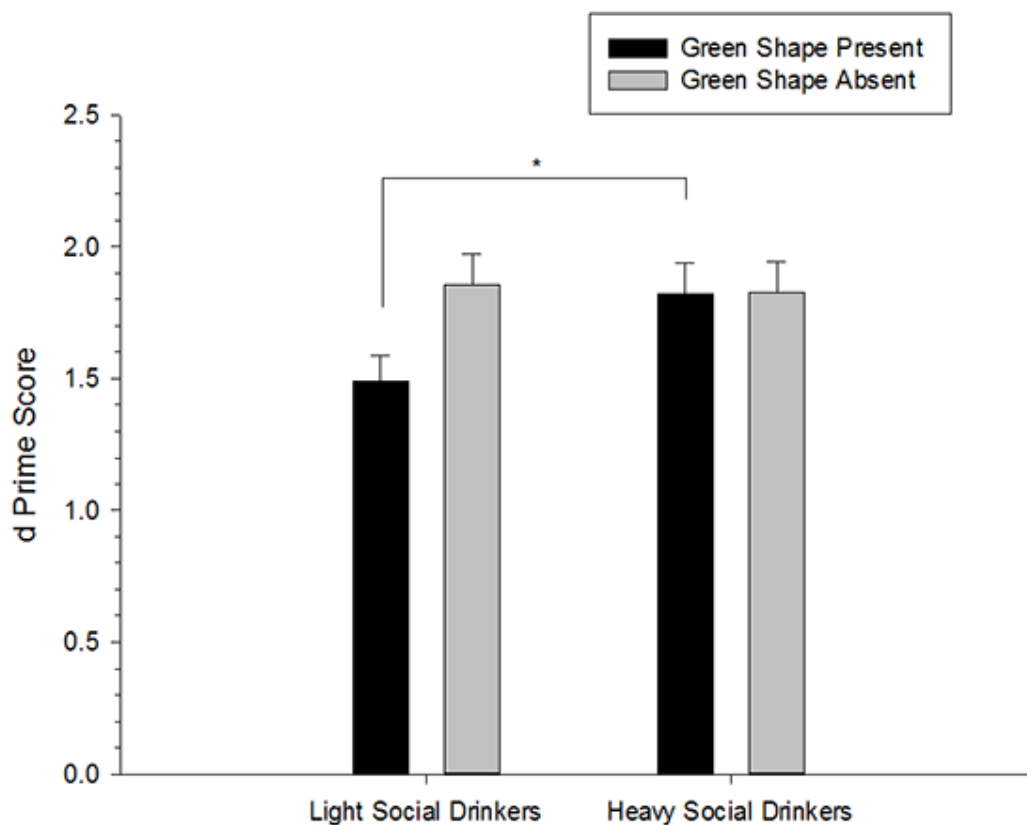
311 **Results**

312 d' was entered into a 2 (Drinker: Heavy/Light) x 2 (Trial Type: Green Present Change/Green Absent
 313 Change) mixed factor ANOVA, refer to Table 3 for accuracy. There was a main effect of Trial Type:
 314 $F(1,48) = 8.211$, $MSE = .106$, $p = .006$, $r = .389$. Participants had a significantly higher d' when there
 315 was no green shape present (mean difference 0.187 ± 0.065). There was also an interaction between
 316 Trial Type and Drinker: $F(1,48) = 7.780$, $MSE = .106$, $p = .008$, $r = .373$. Two Bonferroni-corrected
 317 independent t-tests comparing heavy and light drinkers for both Trial types were conducted. There
 318 was no difference between drinker groups for Green Absent trials: $t(48) = .189$, $p = .851$, however
 319 there was a significant difference between groups in Green Present trials: $t(48) = -2.154$, $p = .036$, $r =$
 320 $.296$. Light drinkers had lower d' scores in Green Present change trials (M: 1.488) than heavy social
 321 drinkers (M: 1.821), as shown in Fig. 5.

322 **Table 3**

323 *Mean hit rate in the Distractibility Task across all types of trial for Heavy and Light social drinkers and*
 324 *mean correct rejection/false-alarm rates for no-change trial when a green stimulus was either*
 325 *present or absent*

<i>Drinker</i>	<i>Trial Type</i>	<i>Hit Rate</i>	<i>Miss Rate</i>
Light Social Drinkers	Bias Present Change	58.88	41.12
	Bias Present No Change	90.27	9.73
	Bias Absent Change	72.14	27.86
	Bias Absent No Change	87.06	12.94
Heavy Social Drinkers	Bias Present Change	71.28	28.72
	Bias Present No Change	86.66	13.34
	Bias Absent Change	75.71	24.29
	Bias Absent No Change	84.30	15.70



326
327 **Fig. 5:** Effect of the presence of a biased stimulus (a green shape) on d' when colour is task-
328 irrelevant. Lower d' indicates decreased sensitivity to change. Light social drinkers are less sensitive
329 at detecting changes when a green shape is present than heavy social drinkers. This suggests light
330 social drinkers are more distracted by the green shape – since it never changes – than heavy social
331 drinkers. *Note:* * p<.05

332 **Discussion**

333 Light social drinkers - who had no pre-existing attentional bias - were distracted away from detecting
334 changes to shapes when a green shape was also present, whereas heavy social drinkers - who had a

335 pre-existing alcohol-related attentional bias - were not. This distraction in light social drinkers
336 manifested in lower sensitivity to detect changes when an irrelevant green shape was also present.
337 Thus, light social drinkers are more distracted by induced attentional biases than heavy social
338 drinkers.

339 **General Discussion**

340 This series of experiments expanded existing findings by examining the effects of a pre-existing
341 attentional bias on behaviour in a change-detection task following the inducement of a new
342 attentional bias. No group differences on initial attentional bias inducement were found, meaning
343 that those with a pre-existing attentional bias are not more susceptible to having additional
344 attentional biases induced. However, when bias-related items were present but irrelevant, only light
345 social drinkers were distracted away from the primary task goal. Thus, having a pre-existing
346 attentional bias actually made heavy social drinkers better at ignoring previously task-relevant items
347 when they were now task-irrelevant. This could be related to more practice at controlling for an
348 attentional bias, since heavy drinkers already hold one towards alcohol which they have to control
349 daily. These control mechanisms are then utilised in the shape (distraction) experiment, meaning
350 heavy social drinkers could control for distractions caused by a further induced bias. Since light social
351 drinkers have no pre-existing attentional bias to control for in the first place, no control mechanisms
352 exist, resulting in increased distractions by their induced bias.

353 This is supported by a study that examined cocaine-related attentional bias using fMRI (Hester &
354 Garavan, 2009). Here, cocaine users who showed behaviourally low levels of an attentional bias had
355 increased activity in the right prefrontal cortex (PFC). Given the role of the right PFC – especially the
356 right Inferior Frontal Cortex – in executing control over behaviour (Aron, Robbins & Poldrack, 2014;
357 Cieslik, Mueller, Eickhoff, Langner & Eickhoff, 2015), this suggests that these cocaine users were
358 exerting higher amounts of cognitive control when completing the experimental task when
359 irrelevant cocaine-information was present. While it cannot be ascertained if the heightened PFC

360 activity resulted in more successful cognitive control, or if the development of the cognitive control
361 has resulted in heightened PFC activity, this study does highlight the potential role of PCF-dependent
362 cognitive mechanisms in controlling for irrelevant distractors; at least in certain addicted
363 populations. It is also worth noting that this corresponds with previous findings showing no
364 associated between impact of cocaine use on frontal executive regions and attention (Smith et al.,
365 2014)

366 It is interesting to note that the activation of cognitive control mechanisms appears to have occurred
367 in the current experiment despite our group of heavy social drinkers having a high mean alcohol
368 consumption rate. High rates of alcohol consumption are typically related to deficits in frontal
369 regions. Alcohol is also known to structurally affect the prefrontal cortex (Baler & Volkow, 2006).
370 Chanraud, Pitel, Pfefferbaum & Sullivan (2011) found evidence of compromised functional
371 connectivity in the posterior cingulate regions of alcoholics, and Cardenas, Studholme, Gazdzinski,
372 Durazzo & Meyerhoff (2007) discovered that recovering alcoholics display a large amount of atrophy
373 in the frontal lobe when initially entering treatment. This atrophy was partially reversible following
374 total abstinence after 8 months, but was not present in alcoholics who relapse. Moreover, in a
375 review, Baler & Volkow (2006) highlight that significant plastic adaptations occur in neurological
376 circuits relating to – among others – salience attribution and inhibitory control (Baler & Volkow,
377 2006; Tremblay & Schultz, 1999; Volkow & Fowler, 2000), suggesting that the attribution of salience
378 towards drug-related items in alcoholics may be influenced by these plastic changes that arise out of
379 dopamine responses to reward (Robinson & Berridge, 2013).

380 In our current experiment, the high alcohol consumption rate of our heavy social drinkers should
381 have at least partly inhibited the ability of the PFC to activate these control mechanisms, however
382 this does not appear to have happened. Indeed, it was our heavy, not light social drinkers who
383 displayed a better ability to control for irrelevant distractors. This could be explained in one of two
384 ways. Firstly, it is possible that this is due to a more persistent attentional bias overriding an induced

385 bias. Attentional biases are usually formed following repeated presentations of stimulus and reward
386 (Stewart, de Wit & Eikelboom, 1984; Wise & Bozarth, 1987). We have shown in a previous
387 experiment (Knight et al., 2016) that attentional biases are related to a persistent alteration of a
388 specific kind of Feature Search Mode (Folk et al., 1992; Bacon & Egeth, 1994; Leber & Egeth, 2006),
389 which gets constantly activated by environmental cues (Cosman and Vecera 2013) relying on long-
390 term memory representations (Carlisle et al., 2011). It is therefore possible that since our heavy
391 social drinkers already hold an attentional bias, their original alcohol-related attentional control
392 settings may have been re-activated when green information became explicitly irrelevant. This
393 would result in these individuals displaying low levels of distractibility towards irrelevant green
394 information because they no longer had the green-related attentional control setting activated, and
395 instead had already reverted back to their original alcohol-related control setting (Albery, Sharma,
396 Noyce, Frings & Moss, 2015).

397 Alternatively, since our heavy and light social drinkers are all undergraduate students at a top-
398 ranking UK university (Complete University Guide, 2015), our undergraduate cohort students are
399 practiced at deploying cognitive control in order to successfully complete their studies (Ostlund &
400 Balleine, 2005; Prabhakaran, Narayanan, Zhao, & Gabrieli, 2000; Ramnani & Owen, 2004; Winocur &
401 Moscovitch, 1990). The current findings might therefore be specific to this population of participants
402 (Alloway & Alloway, 2010; Blair, Gamson, Thorne, & Baker, 2005). Years of education - independent
403 from age – is related to both cognitive and neural development, with strong associations found
404 between educational attainment and cognitive control (Noble, Korgaonkar, Grieve & Brickman,
405 2013). Educational attainment is either not controlled for in investigations of attentional bias in
406 addiction or the sample is dominated by low levels of education (George et al., 2004; Goldstein et
407 al., 2004; Goldstein & Volkow, 2011). Moreover, the plastic changes to frontal regions in alcoholics
408 discussed above are not present in social drinkers (Chanraud et al., 2011; Desmond et al., 2003;
409 Thompson et al., 2004; Yuan et al., 2009), thus in non-addicted samples (of which our group of heavy

410 social drinkers are), PFC function is not yet disrupted. Repeating the current study with a non-
411 university sample may yield different findings, shedding some light on the issue.

412 It is also unlikely that the findings of the current study are due to bottom-up, automatic mechanisms
413 which have been acquired during the procurement of the arbitrary attentional bias. Firstly, the
414 inducement of an attentional bias task showed no differences in behaviour between heavy versus
415 light social drinkers, suggesting an equally successful inducement of the attentional bias. We know
416 from a previous study that these induced biases are persistent (Knight et al., 2016). Thus, behaviour
417 in the distractibility task is related to controlling for irrelevant distractors caused by an induced bias,
418 not the attentional bias dissipating in one group. If the mechanisms for controlling for distractors
419 was bottom-up and automatic in nature, we would expect to see the same pattern of behaviour in
420 all groups. The fact that heavy social drinkers behaved observably different than light social drinkers
421 is suggestive of a top-down process which has been acquired or developed in our heavy drinking
422 sample but is not present or as well-practiced in our light drinkers.

423 It should be noted that while we took every effort to not include participants who had previously or
424 were currently suffering from an alcohol use disorder, we did not specifically screen for any
425 additional diagnosis of other mental health conditions. It is known that there is a high comorbidity of
426 addiction and other mental illnesses (Carrá & Johnson, 2009), such as anxiety (Petry, Stinson &
427 Grant, 2005), depression (Swendsen & Merikangas, 2000) and bipolar disorder (Grant et al., 2005).
428 The wording on our demographic information sheet also asked participants if they were taking any
429 “prescribed or non-prescribed medications”. This therefore should have screened for participants
430 who were currently receiving pharmacological treatment for a range of mental health conditions,
431 however individuals who were diagnosed but not on medication would still have been included.
432 Collecting this data would have provided a useful insight into the additional clinical relevance of our
433 findings, and is something that future studies on this topic should seek to do.

434 Nevertheless, the discussed findings so suggest that when an individual first develops an attentional
435 bias, bias-related information is preferentially processed and has a measurable, behavioural effect.
436 This reflects the findings of light social drinkers in the present study (and those in Knight et al.,
437 2016). Once an individual has had such an attentional bias for a period of time – and is required to
438 ignore potential distractions from it in order to perform optimally day-to-day – there is a
439 requirement for cognitive control to occur. Neurobiologically, this would require the PFC due to the
440 established links between the PFC and higher level reflective processes such as working memory,
441 executive functioning and cognitive control – those processes necessary for internally preventing a
442 pre-potent response (Adams et al., 1993; Cummings, 1993; Stuss & Alexander, 2000; Sullivan,
443 Rosenbloom & Pfefferbaum, 2000; Uekermann & Daum, 2008; Crews & Boettiger, 2009; Groman,
444 James & Jentsch, 2009). In individuals with no prefrontal atrophy caused by an addiction they are
445 able to utilise this. Continued alcohol use which disrupts PFC functionality would disrupt the ability
446 of the PFC to exert this level of control, resulting in findings usually observed in addicted populations
447 (George et al., 2004; Goldstein et al., 2004; Goldstein & Volkow, 2011). Specifically training cognitive
448 control mechanisms or otherwise improving prefrontal activation in addicts could greatly improve
449 their ability to ignore irrelevant bias-related information.

450 Our current findings also expand our previous work on inducing attentional biases in healthy
451 participants by discovering sub-group differences in the overall induced bias effect. When the
452 general population is split into heavy and light social drinkers, it is only for light social drinkers that
453 the distractibility of the biased item when task-irrelevant is found. This shows sub-group differences
454 in attentional bias between heavy and light social drinkers, clarifying previous inconsistent findings
455 (Cox, Brown, & Rowlands, 2003; Cox, Yeates, & Regan, 1999; Sharma et al., 2001), while supporting
456 more recent examinations of attentional bias via eye-movements (McAteer, Curran & Hanna, 2015;
457 Roy-Charland et al., 2017). Put together, these stress the value of using more direct (eye-movement
458 data) and sensitive (signal detection theory) measurements to measure subtle changes in attentional
459 state.

460 In conclusion, it would seem that the possession of one attentional bias does not mean that other
461 biases are more readily acquired. However, in a sub-addiction population, the cognitive processes
462 used to control task-irrelevant distractions caused by pre-existing attentional biases can then be
463 utilised to control for distractions caused by subsequent biases. Thus, pre-existing attentional biases
464 seem to infer an advantage when dealing with possible distractions by caused by subsequent
465 induced biases. This may be due to the sample of participants used in the current experiment being
466 well-practiced at deploying cognitive control strategies. However, as alcohol detrimentally affects
467 the function of frontal brain regions in the long term (Ratti, Bo, Giardini & Soragna, 2002; George,
468 Potts, Kothman, Martin & Mukundan, 2004; Medina et al., 2008), one speculative implication could
469 be that addiction may be mediated by a decreased ability to control for irrelevant substance related
470 information thereby manifesting the established behavioural consequences of addiction.

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