

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19

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

Helen C. Knight^{*a}, Daniel T. Smith^b, David C. Knight^b & Amanda Ellison^b

^a School of Psychology, University of Sunderland, Chester Road, Sunderland,
SR1 3SD, United Kingdom

^bDepartment of Psychology, Durham University, Queen's Campus, University Boulevard,
Stockton-on-Tees, TS17 6BH, United Kingdom

* Correspondence to: helen.knight@sunderland.ac.uk Tel: +44 (0)191 5152571

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

36

37 Key Words:

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

39

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

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

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

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

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

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

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

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

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

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

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

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

144 **Assessment of Attentional Bias to Alcohol**

145 **Method**

146 **Participants**

147 124 undergraduate students in their first or second year of an Applied Psychology course at Durham
148 University (33 male; aged 18-37, M: 20.196, SD 3.328) completed an alcohol consumption
149 questionnaire (Time Line Follow Back (Sobell & Sobell, 1992)). Smoking and/or the taking of
150 prescribed or recreational drugs were exclusion criteria. Participants were asked to fill in the
151 questionnaire relating to their alcohol consumption over the past 7 days. They were then asked if this
152 was reflective of an average week, and if not, were asked to complete a section modified Time Line
153 Follow Back regarding their average alcohol consumption. Participants also checked a box to state
154 they were not nor had previously been treated for any alcohol misuse disorder. Participants were then
155 ranked from highest to lowest alcohol consumption based on total units consumed. Non-drinkers were
156 removed, along with one participant whose reported weekly alcohol consumption was above 3
157 standard deviations from the mean. Ultimately, 50 participants (12 male, aged 18-22, M: 20.08, SD:
158 1.586) with normal or corrected to normal vision and no colour blindness took part. The sample
159 consisted of the 25 heaviest and 25 lightest social drinkers. Heavy social drinkers had an average
160 weekly consumption of 56.86 units (SD: 21.409), light social drinkers had an average weekly
161 consumption of 7.984 units (SD: 4.254). These differed significantly: $t(48) = -11.196$, $p < .001$, $r =$
162 .8504. No cases of heavy or light social drinkers fell outside mean $\pm 3SD$, thus no further outliers
163 were present. All participants gave their informed consent with the approval of Durham University
164 Ethics Advisory Committee and were provided with university course credits for their time.

165 **Apparatus**

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

170 **Stimuli & Procedure**

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

185 **Results**

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

193 [Table 1 here]

194 [Figure 1 here]

195 **Fig. 1:** Pre-existing alcohol-related attentional bias in light versus heavy social drinkers. Higher d'
196 indicates increased sensitivity to change. Sensitivity is higher in heavy social drinkers than light social
197 drinkers when an alcohol-related image appears amongst neutral images. For light social drinkers,

198 sensitivity is highest when a novel neutral image appears amongst other neutral images. Error bars
199 show standard error of the mean. *Note:* ** $p < .005$, *** $p < .001$

200 **Discussion**

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

212 **Attentional Bias Inducement Task**

213 **Method**

214 The 50 participants who completed the alcohol change detection task also completed the attentional
215 bias inducement task. The apparatus was the same as that used for the alcohol change detection task.
216 The attentional bias inducement task was conducted in the same experimental session as the alcohol
217 change detection task.

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

219 A mixed design was used. Following the completion of the alcohol attentional bias experiment, all
220 participants carried out a second change detection task, after replicating the methodology used to
221 induce an attentional bias to green items in Knight et al. (2016). As with the alcohol task, the
222 Attentional Bias Inducement Task was also programmed using Borland C++ builder and presented on
223 a 19" Sony Triniton monitor with a resolution of 1024x768 pixels and a refresh rate of 100Hz using a

224 VSG ViSaGe box and custom graphics card (Cambridge Research Systems, Rochester, England).

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

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

242 [Figure 2 here]

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

246 **Results**

247 d' was entered into a 2 (Drinker: Heavy/Light) x 3 (Trial Type: Congruent Change/Incongruent
248 Change/Neutral Change) mixed factor ANOVA. No change trials were used to calculate d' , thus were
249 analysed within the ANOVA but not as an additional factor, see Table 2 for mean accuracy across all
250 types of trial. There was a significant effect of Trial Type: $F(2,96) = 11.848$, $MSE = 1.183$, $p < .001$.

251 Bonferroni-corrected pairwise comparisons revealed that d' scores in Congruent Change trials were
252 higher than Incongruent Change trials (mean difference .760, $p < .001$, $r = .783$) and Neutral Change
253 trials (mean difference .702, $p = .003$, $r = .454$) – see Fig. 3. Thus, participants were more sensitive to
254 detecting changes to green stimuli than other stimuli, suggesting a successful induced bias towards the
255 colour green. There was no effect of drinker: $F(1,48) = .812$, $MSE = 2.147$, $p = .372$ and no
256 interaction between trial and drinker: $F(2,36) = .636$, $MSE = 1.183$, $p = .465$.

257 [Table 2 here]

258 [Figure 3 here]

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

262 Discussion

263 This experiment investigated if a pre-existing attentional bias affected the procurement of an
264 additional bias by examining if heavy social drinkers are more easily biased towards a neutral
265 stimulus than light social drinkers. Evidence has been found of an equally successful inducement of
266 an attentional bias towards the colour green in both heavy and light social drinkers. Both groups
267 showed an increase in sensitivity at detecting changes to green stimuli, with a larger effect size
268 between sensitivity of detecting congruent and incongruent trials than congruent and neutral trials. If
269 those with a pre-existing attentional bias were more receptive at having additional biases induced,
270 greater sensitivity at detecting green changes in heavy social drinkers compared to light social
271 drinkers would be expected. However, our results from heavy and light social drinkers did not differ,
272 thus it can be concluded that a pre-existing attentional bias does make one more susceptible to the
273 adoption of an additional neutral bias. Nevertheless, whether this extends to additional attentional
274 biases in general remains to be determined. Moreover, as there was no main effect of drinker, nor did
275 drinker interact with trial, it can also be concluded that a potential reactivation of an alcohol
276 attentional bias caused by the first assessment of an alcohol attentional bias did not dampen the
277 development of a further attentional bias in heavy drinkers. Our previous studies have shown that an
278 induced bias can distract participants in a change blindness task in which colour is irrelevant (Knight

279 et al., 2016). A third experiment was therefore run to examine this property in heavy versus light
280 drinkers.

281 **Distractibility from an Induced Attentional Bias**

282 **Method**

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

287 **Stimuli & Procedure: Distractibility Test**

288 The fixation cross was presented for 1000ms followed by the test array consisting of a square (width
289 10.2cm) composition of four different shapes (square, circle, triangle, pentagon or trapezium: visual
290 angle: 2.5° x 2.5°) for 750ms. Again, this was masked for 100ms before reappearing until a response.
291 On 25% (120 trials) of trials a green shape was present and a different shape changed shape (Green
292 Present Change Trials), on 25% of trials a green item was present but no change occurred (Green
293 Present No-Change Trials), on 25% of trials no green item was present and a shape changed shape
294 (Green Absent Change Trials) and on 25% of trials no green item was present and no change occurred
295 (Green Absent No Change Trials). Trials were presented in a random order. Participants completed 6
296 blocks of 80 trials with a 5 minute break between each block. See Fig. 4 for an illustration of a typical
297 trial.

298 [Figure 4 here]

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

302 **Results**

303 d' was entered into a 2 (Drinker: Heavy/Light) x 2 (Trial Type: Green Present Change/Green Absent
304 Change) mixed factor ANOVA, refer to Table 3 for accuracy. There was a main effect of Trial Type:

305 $F(1,48) = 8.211$, $MSE = .106$, $p = .006$, $r = .389$. Participants had a significantly higher d' when there
306 was no green shape present (mean difference 0.187 ± 0.065). There was also an interaction between
307 Trial Type and Drinker: $F(1,48) = 7.780$, $MSE = .106$, $p = .008$, $r = .373$. Two Bonferroni-corrected
308 independent t-tests comparing heavy and light drinkers for both Trial types were conducted. There
309 was no difference between drinker groups for Green Absent trials: $t(48) = .189$, $p = .851$, however
310 there was a significant difference between groups in Green Present trials: $t(48) = -2.154$, $p = .036$, $r =$
311 $.296$. Light drinkers had lower d' scores in Green Present change trials ($M: 1.488$) than heavy social
312 drinkers ($M: 1.821$), as shown in Fig. 5.

313 [Table 3 here]

314 [Figure 5 here]

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

320 **Discussion**

321 Light social drinkers - who had no pre-existing attentional bias - were distracted away from detecting
322 changes to shapes when a green shape was also present, whereas heavy social drinkers - who had a
323 pre-existing alcohol-related attentional bias - were not. This distraction in light social drinkers
324 manifested in lower sensitivity to detect changes when an irrelevant green shape was also present.
325 Thus, light social drinkers are more distracted by induced attentional biases than heavy social
326 drinkers.

327 **General Discussion**

328 This series of experiments expanded existing findings by examining the effects of a pre-existing
329 attentional bias on behaviour in a change-detection task following the inducement of a new
330 attentional bias. No group differences on initial attentional bias inducement were found, meaning
331 that those with a pre-existing attentional bias are not more susceptible to having additional

332 attentional biases induced. However, when bias-related items were present but irrelevant, only light
333 social drinkers were distracted away from the primary task goal. Thus, having a pre-existing
334 attentional bias actually made heavy social drinkers better at ignoring previously task-relevant items
335 when they were now task-irrelevant. This could be related to more practice at controlling for an
336 attentional bias, since heavy drinkers already hold one towards alcohol which they have to control
337 daily. These control mechanisms are then utilised in the shape (distraction) experiment, meaning
338 heavy social drinkers could control for distractions caused by a further induced bias. Since light social
339 drinkers have no pre-existing attentional bias to control for in the first place, no control mechanisms
340 exist, resulting in increased distractions by their induced bias.

341 This is supported by a study that examined cocaine-related attentional bias using fMRI (Hester &
342 Garavan, 2009). Here, cocaine users who showed behaviourally low levels of an attentional bias had
343 increased activity in the right prefrontal cortex (PFC). Given the role of the right PFC – especially the
344 right Inferior Frontal Cortex – in executing control over behaviour (Aron, Robbins & Poldrack, 2014;
345 Cieslik, Mueller, Eickhoff, Langner & Eickhoff, 2015), this suggests that these cocaine users were
346 exerting higher amounts of cognitive control when completing the experimental task when
347 irrelevant cocaine-information was present. While it cannot be ascertained if the heightened PFC
348 activity resulted in more successful cognitive control, or if the development of the cognitive control
349 has resulted in heightened PFC activity, this study does highlight the potential role of PFC-dependent
350 cognitive mechanisms in controlling for irrelevant distractors; at least in certain addicted
351 populations. It is also worth noting that this corresponds with previous findings showing no
352 associated between impact of cocaine use on frontal executive regions and attention (Smith et al.,
353 2014)

354 It is interesting to note that the activation of cognitive control mechanisms appears to have occurred
355 in the current experiment despite our group of heavy social drinkers having a high mean alcohol
356 consumption rate. High rates of alcohol consumption are typically related to deficits in frontal

357 regions. Alcohol is also known to structurally affect the prefrontal cortex (Baler & Volkow, 2006).
358 Chanraud, Pitel, Pfefferbaum & Sullivan (2011) found evidence of compromised functional
359 connectivity in the posterior cingulate regions of alcoholics, and Cardenas, Studholme, Gazdzinski,
360 Durazzo & Meyerhoff (2007) discovered that recovering alcoholics display a large amount of atrophy
361 in the frontal lobe when initially entering treatment. This atrophy was partially reversible following
362 total abstinence after 8 months, but was not present in alcoholics who relapse. Moreover, in a
363 review, Baler & Volkow (2006) highlight that significant plastic adaptations occur in neurological
364 circuits relating to – among others – salience attribution and inhibitory control (Baler & Volkow,
365 2006; Tremblay & Schultz, 1999; Volkow & Fowler, 2000), suggesting that the attribution of salience
366 towards drug-related items in alcoholics may be influenced by these plastic changes that arise out of
367 dopamine responses to reward (Robinson & Berridge, 2013).

368 In our current experiment, the high alcohol consumption rate of our heavy social drinkers should
369 have at least partly inhibited the ability of the PFC to activate these control mechanisms, however
370 this does not appear to have happened. Indeed, it was our heavy, not light social drinkers who
371 displayed a better ability to control for irrelevant distractors. This could be explained in one of two
372 ways. Firstly, it is possible that this is due to a more persistent attentional bias overriding an induced
373 bias. Attentional biases are usually formed following repeated presentations of stimulus and reward
374 (Stewart, de Wit & Eikelboom, 1984; Wise & Bozarth, 1987). We have shown in a previous
375 experiment (Knight et al., 2016) that attentional biases are related to a persistent alteration of a
376 specific kind of Feature Search Mode (Folk et al., 1992; Bacon & Egeth, 1994; Leber & Egeth, 2006),
377 which gets constantly activated by environmental cues (Cosman and Vecera 2013) relying on long-
378 term memory representations (Carlisle et al., 2011). It is therefore possible that since our heavy
379 social drinkers already hold an attentional bias, their original alcohol-related attentional control
380 settings may have been re-activated when green information became explicitly irrelevant. This
381 would result in these individuals displaying low levels of distractibility towards irrelevant green
382 information because they no longer had the green-related attentional control setting activated, and

383 instead had already reverted back to their original alcohol-related control setting (Albery, Sharma,
384 Noyce, Frings & Moss, 2015).

385 Alternatively, since our heavy and light social drinkers are all undergraduate students at a top-
386 ranking UK university (Complete University Guide, 2015), our undergraduate cohort students are
387 practiced at deploying cognitive control in order to successfully complete their studies (Ostlund &
388 Balleine, 2005; Prabhakaran, Narayanan, Zhao, & Gabrieli, 2000; Ramnani & Owen, 2004; Winocur &
389 Moscovitch, 1990). The current findings might therefore be specific to this population of participants
390 (Alloway & Alloway, 2010; Blair, Gamson, Thorne, & Baker, 2005). Years of education - independent
391 from age – is related to both cognitive and neural development, with strong associations found
392 between educational attainment and cognitive control (Noble, Korgaonkar, Grieve & Brickman,
393 2013). Educational attainment is either not controlled for in investigations of attentional bias in
394 addiction or the sample is dominated by low levels of education (George et al., 2004; Goldstein et
395 al., 2004; Goldstein & Volkow, 2011). Moreover, the plastic changes to frontal regions in alcoholics
396 discussed above are not present in social drinkers (Chanraud et al., 2011; Desmond et al., 2003;
397 Thompson et al., 2004; Yuan et al., 2009), thus in non-addicted samples (of which our group of heavy
398 social drinkers are), PFC function is not yet disrupted. Repeating the current study with a non-
399 university sample may yield different findings, shedding some light on the issue.

400 It is also unlikely that the findings of the current study are due to bottom-up, automatic mechanisms
401 which have been acquired during the procurement of the arbitrary attentional bias. Firstly, the
402 inducement of an attentional bias task showed no differences in behaviour between heavy versus
403 light social drinkers, suggesting an equally successful inducement of the attentional bias. We know
404 from a previous study that these induced biases are persistent (Knight et al., 2016). Thus, behaviour
405 in the distractibility task is related to controlling for irrelevant distractors caused by an induced bias,
406 not the attentional bias dissipating in one group. If the mechanisms for controlling for distractors
407 was bottom-up and automatic in nature, we would expect to see the same pattern of behaviour in

408 all groups. The fact that heavy social drinkers behaved observably different than light social drinkers
409 is suggestive of a top-down process which has been acquired or developed in our heavy drinking
410 sample but is not present or as well-practiced in our light drinkers.

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

422 Nevertheless, the discussed findings so suggest that when an individual first develops an attentional
423 bias, bias-related information is preferentially processed and has a measurable, behavioural effect.
424 This reflects the findings of light social drinkers in the present study (and those in Knight et al.,
425 2016). Once an individual has had such an attentional bias for a period of time – and is required to
426 ignore potential distractions from it in order to perform optimally day-to-day – there is a
427 requirement for cognitive control to occur. Neurobiologically, this would require the PFC due to the
428 established links between the PFC and higher level reflective processes such as working memory,
429 executive functioning and cognitive control – those processes necessary for internally preventing a
430 pre-potent response (Adams et al., 1993; Cummings, 1993; Stuss & Alexander, 2000; Sullivan,
431 Rosenbloom & Pfefferbaum, 2000; Uekermann & Daum, 2008; Crews & Boettiger, 2009; Groman,
432 James & Jentsch, 2009). In individuals with no prefrontal atrophy caused by an addiction they are

433 able to utilise this. Continued alcohol use which disrupts PFC functionality would disrupt the ability
434 of the PFC to exert this level of control, resulting in findings usually observed in addicted populations
435 (George et al., 2004; Goldstein et al., 2004; Goldstein & Volkow, 2011). Specifically training cognitive
436 control mechanisms or otherwise improving prefrontal activation in addicts could greatly improve
437 their ability to ignore irrelevant bias-related information.

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

448 In conclusion, it would seem that the possession of one attentional bias does not mean that other
449 biases are more readily acquired. However, in a sub-addiction population, the cognitive processes
450 used to control task-irrelevant distractions caused by pre-existing attentional biases can then be
451 utilised to control for distractions caused by subsequent biases. Thus, pre-existing attentional biases
452 seem to infer an advantage when dealing with possible distractions by caused by subsequent
453 induced biases. This may be due to the sample of participants used in the current experiment being
454 well-practiced at deploying cognitive control strategies. However, as alcohol detrimentally affects
455 the function of frontal brain regions in the long term (Ratti, Bo, Giardini & Soragna, 2002; George,
456 Potts, Kothman, Martin & Mukundan, 2004; Medina et al., 2008), one speculative implication could

457 be that addiction may be mediated by a decreased ability to control for irrelevant substance related
458 information thereby manifesting the established behavioural consequences of addiction.

459

- 461 Adams, K. M., Gilman, S., Koeppe, R. A., Kluin, K. J., Brunberg, J. A., Dede, D., . . . Kroll, P. D.
462 (1993). Neuropsychological Deficits Are Correlated with Frontal Hypometabolism in
463 Positron Emission Tomography Studies of Older Alcoholic Patients. *Alcoholism-Clinical and*
464 *Experimental Research*, 17(2), 205-210. doi: DOI 10.1111/j.1530-0277.1993.tb00750.x
- 465 Albery, I. P., Sharma, D., Noyce, S., Frings, D., & Moss, A. C. (2015). Testing a frequency of
466 exposure hypothesis in attentional bias for alcohol-related stimuli amongst social drinkers.
467 *Addictive Behaviors Reports*, 1, 68-72.
- 468 Alloway, T. P., & Alloway, R. G. (2010). Investigating the predictive roles of working memory and
469 IQ in academic attainment. *Journal of Experimental Child Psychology*, 106(1), 20-29. doi:
470 DOI 10.1016/j.jecp.2009.11.003
- 471 Aron, A.R., Robbins, T.W. & Poldrack, R.A. (2004). Inhibition and the right inferior frontal cortex.
472 *Trends in Cognitive Science*, 8(4), 170-199. doi: doi:10.1016/j.tics.2004.02.010
- 473 Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex:
474 one decade on. *Trends in cognitive sciences*, 18(4), 177-185.
- 475 Baler, R. D., & Volkow, N. D. (2006). Drug addiction: the neurobiology of disrupted self-control.
476 *Trends in Molecular Medicine*, 12(12), 559-566. doi: DOI 10.1016/j.molmed.2006.10.005
- 477 Blair, C., Gamson, D., Thorne, S., & Baker, D. (2005). Rising mean IQ: Cognitive demand of
478 mathematics education for young children, population exposure to formal schooling, and the
479 neurobiology of the prefrontal cortex. *Intelligence*, 33(1), 93-106. doi: DOI
480 10.1016/j.intell.2004.07.008
- 481 Boyer, M., & Dickerson, M. (2003). Attentional bias and addictive behaviour: automaticity in a
482 gambling-specific modified Stroop task. *Addiction*, 98(1), 61-70.
- 483 Bruce, G., & Jones, B. T. (2004). A pictorial Stroop paradigm reveals an alcohol attentional bias in
484 heavier compared to lighter social drinkers. *Journal of Psychopharmacology*, 18(4), 527-+.
485 doi: Doi 10.1177/0269881104047280
- 486 Bruce, G., & Jones, B. T. (2006). Methods, measures, and findings of attentional bias in substance
487 use, abuse, and dependence, 135-149. In Wiers, R.W. & Stacy, A.W. (2006), *Handbook of*
488 *Implicit Cognition and Addiction*, USA: SAGE Publications.
- 489 Cardenas, V. A., Studholme, C., Gazdzinski, S., Durazzo, T. C., & Meyerhoff, D. J. (2007).
490 Deformation-based morphometry of brain changes in alcohol dependence and abstinence.
491 *Neuroimage*, 34(3), 879-887. doi: DOI 10.1016/j.neuroimage.2006.10.015
- 492 Carra, G., & Johnson, S. (2009). Variations in rates of comorbid substance use in psychosis between
493 mental health settings and geographical areas in the UK. *Social Psychiatry and Psychiatric*
494 *Epidemiology*, 44(6), 429-447.
- 495 Cieslik, E. C., Mueller, V. I., Eickhoff, C. R., Langner, R., & Eickhoff, S. B. (2015). Three key
496 regions for supervisory attentional control: evidence from neuroimaging meta-analyses.
497 *Neuroscience & biobehavioral reviews*, 48, 22-34.
- 498 Complete University Guide (2015). *University League Table*, Retrieved on 20/10/2015 14:37 GMT
499 from <http://www.thecompleteuniversityguide.co.uk/league-tables/rankings>
- 500 Constantinou, N., Morgan, C. J. A., Battistella, S., O'Ryan, D., Davis, P., & Curran, H. V. (2010).
501 Attentional bias, inhibitory control and acute stress in current and former opiate addicts. *Drug*
502 *and Alcohol Dependence*, 109(1-3), 220-225. doi: DOI 10.1016/j.drugalcdep.2010.01.012
- 503 Cox, W. M., Blount, J. P., & Rozak, A. M. (2000). Alcohol abusers' and nonabusers' distraction by
504 alcohol and concern-related stimuli. *American Journal of Drug and Alcohol Abuse*, 26(3),
505 489-495. doi: Doi 10.1081/Ada-100100258
- 506 Cox, W. M., Brown, M. A., & Rowlands, L. J. (2003). The effects of alcohol cue exposure on non-
507 dependent drinkers' attentional bias for alcohol-related stimuli. *Alcohol and Alcoholism*,
508 38(1), 45-49. doi: DOI 10.1093/alcalc/agg010
- 509 Cox, W. M., Hogan, L. M., Kristian, M. R., & Race, J. H. (2002). Alcohol attentional bias as a
510 predictor of alcohol abusers' treatment outcome. *Drug and Alcohol Dependence*, 68(3), 237-
511 243. doi: Pii S0376-8716(02)00219-3

- 512 Cox, W. M., Yeates, G. N., & Regan, C. M. (1999). Effects of alcohol cues on cognitive processing in
513 heavy and light drinkers. *Drug and Alcohol Dependence*, 55(1-2), 85-89. doi: Doi
514 10.1016/S0376-8716(98)00186-0
- 515 Crews, F. T., & Boettiger, C. A. (2009). Impulsivity, frontal lobes and risk for addiction.
516 *Pharmacology Biochemistry and Behavior*, 93(3), 237-247. doi: DOI
517 10.1016/j.pbb.2009.04.018
- 518 Cummings, J. L. (1993). Frontal-Subcortical Circuits and Human-Behavior. *Archives of Neurology*,
519 50(8), 873-880.
- 520 Fadardi, J. S., & Cox, W. M. (2008). Alcohol-attentional bias and motivational structure as
521 independent predictors of social drinkers' alcohol consumption. *Drug and Alcohol*
522 *Dependence*, 97(3), 247-256. doi: DOI 10.1016/j.drugalcdep.2008.03.027
- 523 Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development,
524 causes, and consequences. *Drug and Alcohol Dependence*, 97(1-2), 1-20. doi: DOI
525 10.1016/j.drugalcdep.2008.03.030
- 526 Field, M., Mogg, K., & Bradley, B. P. (2005). Craving and cognitive biases for alcohol cues in social
527 drinkers. *Alcohol and Alcoholism*, 40(6), 504-510. doi: DOI 10.1093/alcalc/agh213
- 528 Field, M., Mogg, K., Zettler, J., & Bradley, B. P. (2004). Attentional biases for alcohol cues in heavy
529 and light social drinkers: the roles of initial orienting and maintained attention.
530 *Psychopharmacology*, 176(1), 88-93. doi: DOI 10.1007/s00213-004-1855-1
- 531 Flaudias, V., Brousse, G., de Chazeron, I., Planche, F., Brun, J., & Llorca, P. M. (2013). Treatment in
532 hospital for alcohol-dependent patients decreases attentional bias. *Neuropsychiatric Disease*
533 *and Treatment*, 9, 773-779. doi: Doi 10.2147/Ndt.S42556
- 534 Folk, C. L., Remington, R. W., & Johnston, J. C. (1992). Involuntary Covert Orienting Is Contingent
535 on Attentional Control Settings. *Journal of Experimental Psychology-Human Perception and*
536 *Performance*, 18(4), 1030-1044.
- 537 George, M. R. M., Potts, G., Kothman, D., Martin, L., & Mukundan, C. R. (2004). Frontal deficits in
538 alcoholism: An ERP study. *Brain and Cognition*, 54(3), 245-247. doi: DOI
539 10.1016/j.bandc.2004.02.025
- 540 Goldstein, R. Z., Leskovjan, A. C., Hoff, A. L., Hitzemann, R., Bashan, F., Khalsa, S. S., . . . Volkow,
541 N. D. (2004). Severity of neuropsychological impairment in cocaine and alcohol addiction:
542 association with metabolism in the prefrontal cortex. *Neuropsychologia*, 42(11), 1447-1458.
543 doi: DOI 10.1016/j.neuropsychologia.2004.04.002
- 544 Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction:
545 neuroimaging findings and clinical implications. *Nature Reviews Neuroscience*, 12(11), 652-
546 669. doi: Doi 10.1038/Nrn3119
- 547 Grant, B. F., Stinson, F. S., Hasin, D. S., Dawson, D. A., Chou, S. P., Ruan, W., & Huang, B. (2005).
548 Prevalence, correlates, and comorbidity of bipolar I disorder and axis I and II disorders:
549 results from the National Epidemiologic Survey on Alcohol and Related Conditions. *The*
550 *Journal of clinical psychiatry*.
- 551 Groman, S. M., James, A. S., & Jentsch, J. D. (2009). Poor response inhibition: At the nexus between
552 substance abuse and attention deficit/hyperactivity disorder. *Neuroscience and Biobehavioral*
553 *Reviews*, 33(5), 690-698. doi: DOI 10.1016/j.neubiorev.2008.08.008
- 554 Hester, R., & Garavan, H. (2009). Neural mechanisms underlying drug-related cue distraction in
555 active cocaine users. *Pharmacology Biochemistry and Behavior*, 93(3), 270-277. doi: DOI
556 10.1016/j.pbb.2008.12.009
- 557 Johnsen, B. H., Laberg, J. C., Cox, W. M., Vaksdal, A., & Hugdahl, K. (1994). Alcoholic Subjects'
558 Attentional Bias in the Processing of Alcohol-Related Words. *Psychology of Addictive*
559 *Behaviours*, 8(2), 5.
- 560 Jones, B. T., Bruce, G., Livingstone, S., & Reed, E. (2006). Alcohol-related attentional bias in
561 problem drinkers with the flicker change blindness paradigm. *Psychology of Addictive*
562 *Behaviors*, 20(2), 171-177. doi: Doi 10.1037/0893-164x.20.2.171
- 563 Jones, B. C., Jones, B. T., Blundell, L., & Bruce, G. (2002). Social users of alcohol and cannabis who
564 detect substance-related changes in a change blindness paradigm report higher levels of use
565 than those detecting substance-neutral changes. *Psychopharmacology*, 165(1), 93-96.

566 Jones, B. T., Jones, B. C., Smith, H., & Copley, N. (2003). A flicker paradigm for inducing change
567 blindness reveals alcohol and cannabis information processing biases in social users.
568 *Addiction*, 98(2), 235-244. doi: 270 [pii]

569 Jones, B. T., & Schulze, D. (2000). Alcohol-related words of positive affect are more accessible in
570 social drinkers' memory than are other words when sip-primed by alcohol. *Addiction*
571 *Research*, 8(3), 221-232. doi: Doi 10.3109/16066350009004422

572 Knight, H.C., Smith, D.T., Knight, D.C. & Ellison, A. (2016). Altering attentional control settings
573 causes persistent biases of visual attention. *The Quarterly Journal of Experimental*
574 *Psychology* 96(1): 129-149.

575 Lusher, J., Chandler, C., & Ball, D. (2004). Alcohol dependence and the alcohol Stroop paradigm:
576 evidence and issues. *Drug and Alcohol Dependence*, 75(3), 225-231. doi: DOI
577 10.1016/j.drugalcdep.2004.03.004

578 Macleod, C., Mathews, A., & Tata, P. (1986). Attentional Bias in Emotional Disorders. *Journal of*
579 *Abnormal Psychology*, 95(1), 15-20.

580 McAteer, A. M., Curran, D., & Hanna, D. (2015). Alcohol attention bias in adolescent social drinkers:
581 an eye tracking study. *Psychopharmacology*, 232(17), 3183-3191.

582 Medina, K. L., McQueeny, T., Nagel, B. J., Hanson, K. L., Schweinsburg, A. D., & Tapert, S. F.
583 (2008). Prefrontal cortex volumes in adolescents with alcohol use disorders: Unique gender
584 effects. *Alcoholism-Clinical and Experimental Research*, 32(3), 386-394. doi: DOI
585 10.1111/j.1530-0277.2007.00602.x

586 Montgomery, C., Field, M., Atkinson, A. M., Cole, J. C., Goudie, A. J., & Sumnall, H. R. (2010).
587 Effects of alcohol preload on attentional bias towards cocaine-related cues.
588 *Psychopharmacology*, 210(3), 365-375. doi: DOI 10.1007/s00213-010-1830-y

589 Nikolaou, K., Field, M., Critchley, H., & Duka, T. (2013). Acute Alcohol Effects on Attentional Bias
590 are Mediated by Subcortical Areas Associated with Arousal and Salience Attribution.
591 *Neuropsychopharmacology*, 38(7), 1365-1373. doi: Doi 10.1038/Npp.2013.34

592 Noble, K. G., Korgaonkar, M. S., Grieve, S. M., & Brickman, A. M. (2013). Higher education is an
593 age-independent predictor of white matter integrity and cognitive control in late adolescence.
594 *Developmental science*, 16(5), 653-664.

595 Noel, X., Colmant, M., Van der Linden, M., Bechara, A., Bullens, Q., Hanak, C., & Verbanck, P.
596 (2006). Time course of attention for alcohol cues in abstinent alcoholic patients: The role of
597 initial orienting. *Alcoholism-Clinical and Experimental Research*, 30(11), 1871-1877. doi:
598 DOI 10.1111/j.1530-0277.00224.x

599 Ostlund, S. B., & Balleine, B. W. (2005). Lesions of medial prefrontal cortex disrupt the acquisition
600 but not the expression of goal-directed learning. *Journal of Neuroscience*, 25(34), 7763-7770.
601 doi: Doi 10.1523/Jneurosci.1921-05.2005

602 Pau, C. W., Lee, T. M., & Shui-Fun, F. C. (2002). The impact of heroin on frontal executive
603 functions. *Archives of clinical neuropsychology*, 17(7), 663-670.

604 Petry, N. M., Stinson, F. S., & Grant, B. F. (2005). Comorbidity of DSM-IV pathological gambling
605 and other psychiatric disorders: results from the National Epidemiologic Survey on Alcohol
606 and Related Conditions. *The Journal of clinical psychiatry*.

607 Prabhakaran, V., Narayanan, K., Zhao, Z., & Gabrieli, J. D. E. (2000). Integration of diverse
608 information in working memory within the frontal lobe. *Nature Neuroscience*, 3(1), 85-90.
609 doi: Doi 10.1038/71156

610 Ramnani, N., & Owen, A. M. (2004). Anterior prefrontal cortex: Insights into function from anatomy
611 and neuroimaging. *Nature Reviews Neuroscience*, 5(3), 184-194. doi: Doi 10.1038/Nrn1343

612 Ratti, M. T., Bo, P., Giardini, A., & Soragna, D. (2002). Chronic alcoholism and the frontal lobe:
613 which executive functions are impaired? *Acta Neurologica Scandinavica*, 105(4), 276-281.
614 doi: DOI 10.1034/j.1600-0404.2002.0o315.x

615 Roy-Charland, A., Plamondon, A., Homeniuk, A. S., Flesch, C. A., Klein, R. M., & Stewart, S. H.
616 (2017). Attentional bias toward alcohol-related stimuli in heavy drinkers: evidence from
617 dynamic eye movement recording. *The American journal of drug and alcohol abuse*, 43(3),
618 332-340.

- 619 Ryan, F. (2002). Detected, selected, and sometimes neglected: Cognitive processing of cues in
620 addiction. *Experimental and Clinical Psychopharmacology*, 10(2), 67-76. doi: Doi
621 10.1037//1064-1297.10.2.67
- 622 Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive
623 biases and craving in heavy drinkers. *Psychopharmacology*, 197(1), 169-178. doi: DOI
624 10.1007/s00213-007-1023-5
- 625 Seeley, W.W., Menon, V., Schatzberg, A.F., Keller, J., Glover, G.H., Kenna, H., Reiss, A.L. &
626 Greicius, M.D. (2007). Dissociable intrinsic connectivity networks for salience processing
627 and executive control. *The Journal of Neuroscience*, 27(9), 2349-2356. doi:
628 10.1523/JNEUROSCI.5587-06.2007
- 629 Sharma, D., Albery, I. P., & Cook, C. (2001). Selective attentional bias to alcohol related stimuli in
630 problem drinkers and non-problem drinkers. *Addiction*, 96(2), 285-295. doi: DOI
631 10.1046/j.1360-0443.2001.96228512.x
- 632 Smith, D. G., Jones, P. S., Bullmore, E. T., Robbins, T. W., & Ersche, K. D. (2014). Enhanced
633 orbitofrontal cortex function and lack of attentional bias to cocaine cues in recreational
634 stimulant users. *Biological psychiatry*, 75(2), 124-131.
- 635 Sobell, L. C., & Sobell, M. B. (1992). Timeline Follow-Back - a Technique for Assessing Self-
636 Reported Alcohol-Consumption. *Measuring Alcohol Consumption*, 41-72-228.
- 637 Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: a conceptual view.
638 *Psychological Research-Psychologische Forschung*, 63(3-4), 289-298. doi: DOI
639 10.1007/s004269900007
- 640 Swendsen, J. D., & Merikangas, K. R. (2000). The comorbidity of depression and substance use
641 disorders. *Clinical psychology review*, 20(2), 173-189.
- 642 Sullivan, E. V., Rosenbloom, M. J., & Pfefferbaum, A. (2000). Pattern of motor and cognitive deficits
643 in detoxified alcoholic men. *Alcoholism-Clinical and Experimental Research*, 24(5), 611-621.
644 doi: DOI 10.1111/j.1530-0277.2000.tb02032.x
- 645 Townshend, J. M., & Duka, T. (2001). Attentional bias associated with alcohol cues: differences
646 between heavy and occasional social drinkers. *Psychopharmacology*, 157(1), 67-74.
- 647 Uekermann, J., & Daum, I. (2008). Social cognition in alcoholism: a link to prefrontal cortex
648 dysfunction? *Addiction*, 103(5), 726-735. doi: DOI 10.1111/j.1360-0443.2008.02157.x
- 649 Waters, H., & Green, M. W. (2003). A demonstration of attentional bias, using a novel dual task
650 paradigm, towards clinically salient material in recovering alcohol abuse patients?
651 *Psychological Medicine*, 33(3), 491-498. doi: Doi 10.1017/S0033291702007237
- 652 Winocur, G., & Moscovitch, M. (1990). Hippocampal and Prefrontal Cortex Contributions to
653 Learning and Memory - Analysis of Lesion and Aging Effects on Maze-Learning in Rats.
654 *Behavioral Neuroscience*, 104(4), 544-551. doi: Doi 10.1037/0735-7044.104.4.544
- 655 Yaxley, R. H., & Zwaan, R. A. (2005). Attentional bias affects change detection. *Psychonomic*
656 *Bulletin & Review*, 12(6), 1106-1111.

657

658

659

Tables

660 **Table 1**

661 *Mean hit/miss rate in the Alcohol Task across all types of change trial, and mean correct*
662 *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

663

664

665

666

667 **Table 2**

668 *Mean hit rate in the Attentional Bias Inducement Task across all types of trial for Heavy and Light*
 669 *social drinkers and mean correct rejection/false-alarm rates for no-change trial when a green*
 670 *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

671

672

673 **Table 3**

674 *Mean hit rate in the Distractibility Task across all types of trial for Heavy and Light social drinkers and*
 675 *mean correct rejection/false-alarm rates for no-change trial when a green stimulus was either*
 676 *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

677