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, Shackleton House, City Campus, Chester Road, Sunderland, SR1 3SD, United Kingdom

[b]Psychology Department, Upper Mountjoy, South Road, Durham University, Durham, DH1, 3LE, United Kingdom

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

Abstract

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

Key Words:

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

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

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

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

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

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

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

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

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

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

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

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

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

Assessment of Attentional Bias to Alcohol

Method

Participants

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

Apparatus

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

Stimuli & Procedure

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

Results

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

Table 1

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

Drinker	*Trial Type*	*Hit Rate*	*Miss Rate*
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

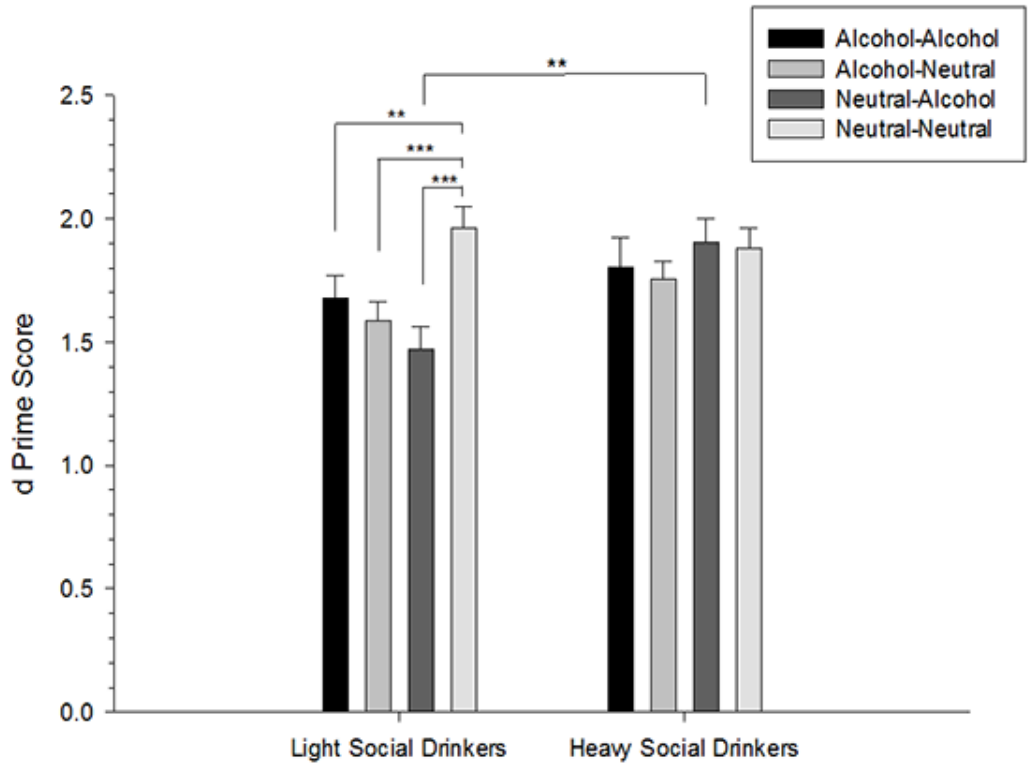

Fig. 1: Pre-existing alcohol-related attentional bias in light versus heavy social drinkers. Higher d' indicates increased sensitivity to change. Sensitivity is higher in heavy social drinkers than light social drinkers when an alcohol-related image appears amongst neutral images. For light social drinkers,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

Discussion

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

Attentional Bias Inducement Task

Method

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

Stimuli, Apparatus & Procedure: Attentional Bias Inducement Task

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

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

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

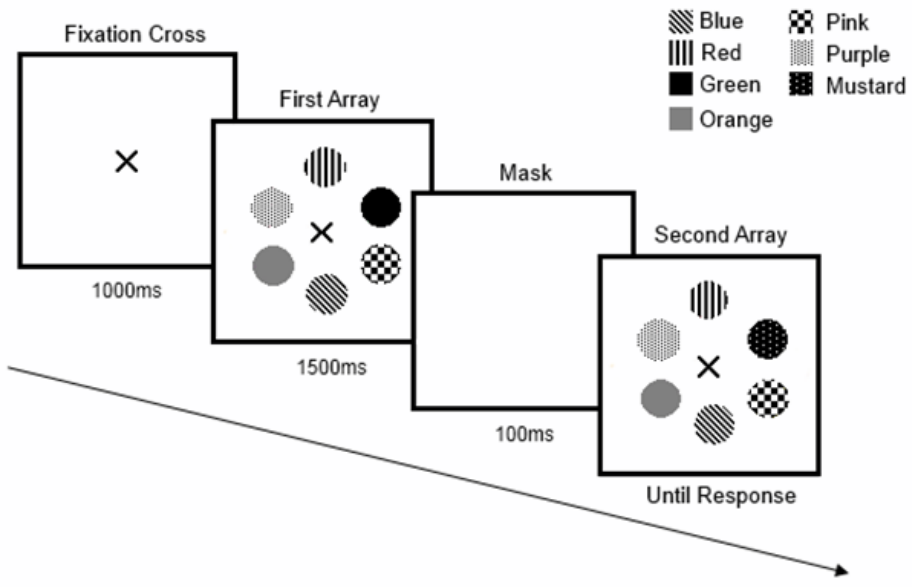

Fig 2: Procedure of Bias Experiment. A fixation cross was presented for 1000ms, followed the first array for 1500ms. This was then masked for 100ms before reappearing, where participants had to make their response as quickly but as accurately as possible, using the index finger of each hand.

Results

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

Table 2

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

Drinker	*Trial Type*	*Hit Rate*	*Miss Rate*
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

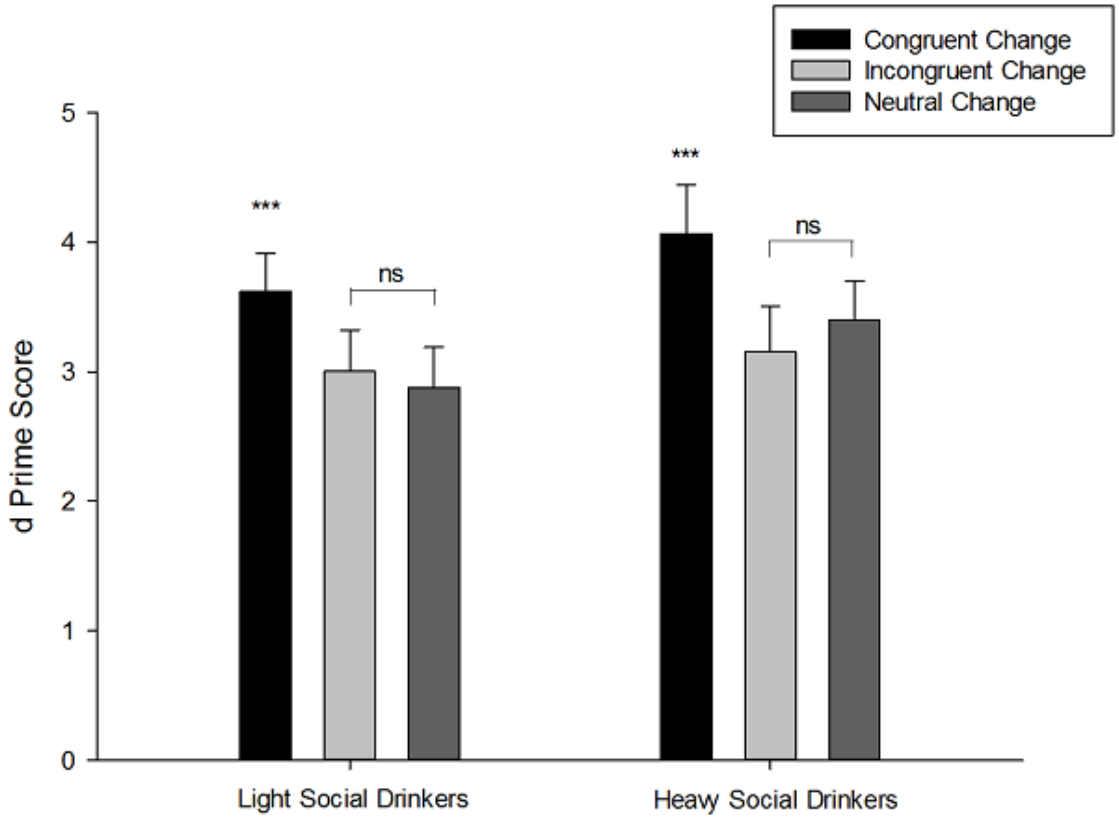

Fig. 3: Effect of induced attentional bias towards green on d' in a change detection task. Higher d' indicates greater sensitivity to change. Sensitivity is higher in Congruent Change trials than both Incongruent and Neutral change trials. *Note:* *** p<.001

Discussion

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

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

Distractibility from an Induced Attentional Bias

Method

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

Stimuli & Procedure: Distractibility Test

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

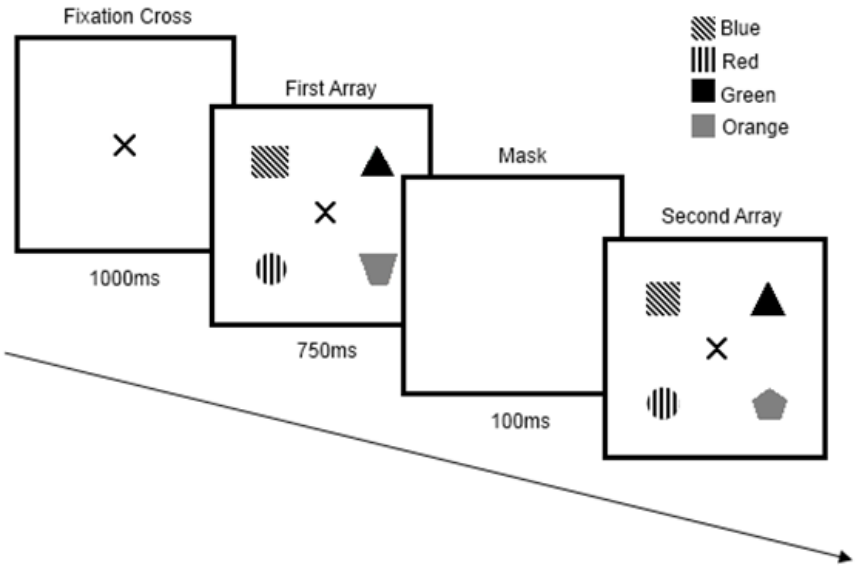

Fig. 4: Procedure of Shape Experiment. A fixation cross was presented for 1000ms, followed the first array for 750ms. This was then masked for 100ms before reappearing, where participants had to make their response as quickly but as accurately as possible, using the index finger of each hand.

Results

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

Table 3

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

Drinker	*Trial Type*	*Hit Rate*	*Miss Rate*
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

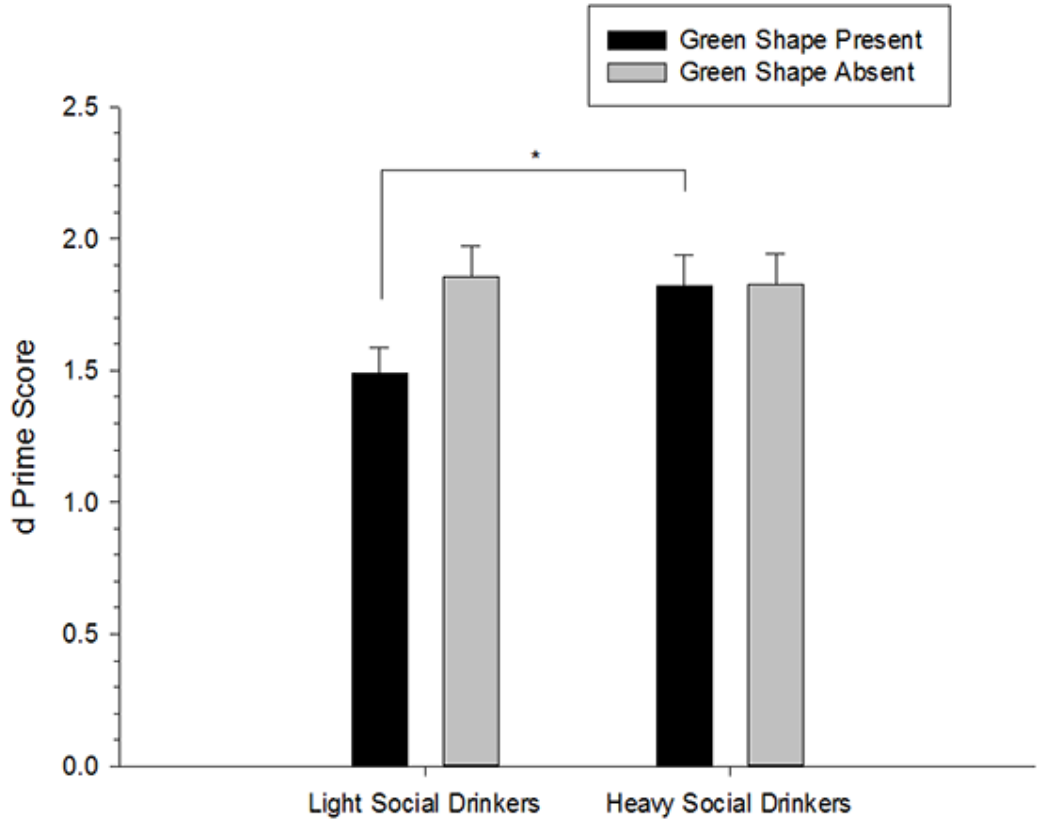

Fig. 5: Effect of the presence of a biased stimulus (a green shape) on d' when colour is task-irrelevant. Lower d' indicates decreased sensitivity to change. Light social drinkers are less sensitive at detecting changes when a green shape is present than heavy social drinkers. This suggests light social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

Discussion

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

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

General Discussion

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

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

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

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

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

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

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

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

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

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

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

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

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

References

Adams, K. M., Gilman, S., Koeppe, R. A., Kluin, K. J., Brunberg, J. A., Dede, D., . . . Kroll, P. D. (1993). Neuropsychological Deficits Are Correlated with Frontal Hypometabolism in Positron Emission Tomography Studies of Older Alcoholic Patients. *Alcoholism-Clinical and Experimental Research, 17*(2), 205-210. doi: DOI 10.1111/j.1530-0277.1993.tb00750.x

Albery, I. P., Sharma, D., Noyce, S., Frings, D., & Moss, A. C. (2015). Testing a frequency of exposure hypothesis in attentional bias for alcohol-related stimuli amongst social drinkers. Addictive Behaviors Reports, 1, 68-72.

Alloway, T. P., & Alloway, R. G. (2010). Investigating the predictive roles of working memory and IQ in academic attainment. *Journal of Experimental Child Psychology, 106*(1), 20-29. doi: DOI 10.1016/j.jecp.2009.11.003

Aron, A.R., Robbins, T.W. & Poldrack, R.A. (2004). Inhibition and the right inferior frontal cortex. *Trends in Cognitive Science, 8*(4), 170-199. doi: doi:10.1016/j.tics.2004.02.010

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: one decade on. Trends in cognitive sciences, 18(4), 177-185.

Baler, R. D., & Volkow, N. D. (2006). Drug addiction: the neurobiology of disrupted self-control. *Trends in Molecular Medicine, 12*(12), 559-566. doi: DOI 10.1016/j.molmed.2006.10.005

Blair, C., Gamson, D., Thorne, S., & Baker, D. (2005). Rising mean IQ: Cognitive demand of mathematics education for young children, population exposure to formal schooling, and the neurobiology of the prefrontal cortex. *Intelligence, 33*(1), 93-106. doi: DOI 10.1016/j.intell.2004.07.008

Boyer, M., & Dickerson, M. (2003). Attentional bias and addictive behaviour: automaticity in a gambling-specific modified Stroop task. *Addiction, 98*(1), 61-70.

Bruce, G., & Jones, B. T. (2004). A pictorial Stroop paradigm reveals an alcohol attentional bias in heavier compared to lighter social drinkers. *Journal of Psychopharmacology, 18*(4), 527-+. doi: Doi 10.1177/0269881104047280

Bruce, G., & Jones, B. T. (2006). Methods, measures, and findings of attentional bias in substance use, abuse, and dependence, 135-149. In Wiers, R.W. & Stacy, A.W. (2006), *Handbook of Implicit Cognition and Addiction*, USA: SAGE Publications.

Cardenas, V. A., Studholme, C., Gazdzinski, S., Durazzo, T. C., & Meyerhoff, D. J. (2007). Deformation-based morphometry of brain changes in alcohol dependence and abstinence. *Neuroimage, 34*(3), 879-887. doi: DOI 10.1016/j.neuroimage.2006.10.015

Carra, G., & Johnson, S. (2009). Variations in rates of comorbid substance use in psychosis between mental health settings and geographical areas in the UK. Social Psychiatry and Psychiatric Epidemiology, 44(6), 429-447.

Cieslik, E. C., Mueller, V. I., Eickhoff, C. R., Langner, R., & Eickhoff, S. B. (2015). Three key regions for supervisory attentional control: evidence from neuroimaging meta-analyses. Neuroscience & biobehavioral reviews, 48, 22-34.

Complete University Guide (2015). *University League Table*, Retrieved on 20/10/2015 14:37 GMT from http://www.thecompleteuniversityguide.co.uk/league-tables/rankings

Constantinou, N., Morgan, C. J. A., Battistella, S., O'Ryan, D., Davis, P., & Curran, H. V. (2010). Attentional bias, inhibitory control and acute stress in current and former opiate addicts. *Drug and Alcohol Dependence, 109*(1-3), 220-225. doi: DOI 10.1016/j.drugalcdep.2010.01.012

Cox, W. M., Blount, J. P., & Rozak, A. M. (2000). Alcohol abusers' and nonabusers' distraction by alcohol and concern-related stimuli. *American Journal of Drug and Alcohol Abuse, 26*(3), 489-495. doi: Doi 10.1081/Ada-100100258

Cox, W. M., Brown, M. A., & Rowlands, L. J. (2003). The effects of alcohol cue exposure on non-dependent drinkers' attentional bias for alcohol-related stimuli. *Alcohol and Alcoholism, 38*(1), 45-49. doi: DOI 10.1093/alcalc/agg010

Cox, W. M., Hogan, L. M., Kristian, M. R., & Race, J. H. (2002). Alcohol attentional bias as a predictor of alcohol abusers' treatment outcome. *Drug and Alcohol Dependence, 68*(3), 237-243. doi: Pii S0376-8716(02)00219-3

Cox, W. M., Yeates, G. N., & Regan, C. M. (1999). Effects of alcohol cues on cognitive processing in heavy and light drinkers. *Drug and Alcohol Dependence, 55*(1-2), 85-89. doi: Doi 10.1016/S0376-8716(98)00186-0

Crews, F. T., & Boettiger, C. A. (2009). Impulsivity, frontal lobes and risk for addiction. *Pharmacology Biochemistry and Behavior*, 93(3), 237-247. doi: DOI 10.1016/j.pbb.2009.04.018

Cummings, J. L. (1993). Frontal-Subcortical Circuits and Human-Behavior. *Archives of Neurology*, 50(8), 873-880.

Fadardi, J. S., & Cox, W. M. (2008). Alcohol-attentional bias and motivational structure as independent predictors of social drinkers' alcohol consumption. *Drug and Alcohol Dependence, 97*(3), 247-256. doi: DOI 10.1016/j.drugalcdep.2008.03.027

Field, M., & Cox, W. M. (2008). Attentional bias in addictive behaviors: A review of its development, causes, and consequences. *Drug and Alcohol Dependence, 97*(1-2), 1-20. doi: DOI 10.1016/j.drugalcdep.2008.03.030

Field, M., Mogg, K., & Bradley, B. P. (2005). Craving and cognitive biases for alcohol cues in social drinkers. *Alcohol and Alcoholism, 40*(6), 504-510. doi: DOI 10.1093/alcalc/agh213

Field, M., Mogg, K., Zetteler, J., & Bradley, B. P. (2004). Attentional biases for alcohol cues in heavy and light social drinkers: the roles of initial orienting and maintained attention. *Psychopharmacology, 176*(1), 88-93. doi: DOI 10.1007/s00213-004-1855-1

Flaudias, V., Brousse, G., de Chazeron, I., Planche, F., Brun, J., & Llorca, P. M. (2013). Treatment in hospital for alcohol-dependent patients decreases attentional bias. *Neuropsychiatric Disease and Treatment, 9*, 773-779. doi: Doi 10.2147/Ndt.S42556

Folk, C. L., Remington, R. W., & Johnston, J. C. (1992). Involuntary Covert Orienting Is Contingent on Attentional Control Settings. *Journal of Experimental Psychology-Human Perception and Performance, 18*(4), 1030-1044.

George, M. R. M., Potts, G., Kothman, D., Martin, L., & Mukundan, C. R. (2004). Frontal deficits in alcoholism: An ERP study. *Brain and Cognition, 54*(3), 245-247. doi: DOI 10.1016/j.bandc.2004.02.025

Goldstein, R. Z., Leskovjan, A. C., Hoff, A. L., Hitzemann, R., Bashan, F., Khalsa, S. S., . . . Volkow, N. D. (2004). Severity of neuropsychological impairment in cocaine and alcohol addiction: association with metabolism in the prefrontal cortex. *Neuropsychologia, 42*(11), 1447-1458. doi: DOI 10.1016/j.neuropsychologia.2004.04.002

Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nature Reviews Neuroscience, 12*(11), 652-669. doi: Doi 10.1038/Nrn3119

Grant, B. F., Stinson, F. S., Hasin, D. S., Dawson, D. A., Chou, S. P., Ruan, W., & Huang, B. (2005). Prevalence, correlates, and comorbidity of bipolar I disorder and axis I and II disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. The Journal of clinical psychiatry.

Groman, S. M., James, A. S., & Jentsch, J. D. (2009). Poor response inhibition: At the nexus between substance abuse and attention deficit/hyperactivity disorder. *Neuroscience and Biobehavioral Reviews*, 33(5), 690-698. doi: DOI 10.1016/j.neubiorev.2008.08.008

Hester, R., & Garavan, H. (2009). Neural mechanisms underlying drug-related cue distraction in active cocaine users. *Pharmacology Biochemistry and Behavior, 93*(3), 270-277. doi: DOI 10.1016/j.pbb.2008.12.009

Johnsen, B. H., Laberg, J. C., Cox, W. M., Vaksdal, A., & Hugdahl, K. (1994). Alcoholic Subjects' Attentional Bias in the Processing of Alcohol-Related Words. *Psychology of Addictive Behaviours, 8*(2), 5.

Jones, B. T., Bruce, G., Livingstone, S., & Reed, E. (2006). Alcohol-related attentional bias in problem drinkers with the flicker change blindness paradigm. *Psychology of Addictive Behaviors, 20*(2), 171-177. doi: Doi 10.1037/0893-164x.20.2.171

Jones, B. C., Jones, B. T., Blundell, L., & Bruce, G. (2002). Social users of alcohol and cannabis who detect substance-related changes in a change blindness paradigm report higher levels of use than those detecting substance-neutral changes. Psychopharmacology, 165(1), 93-96.

578 Jones, B. T., Jones, B. C., Smith, H., & Copley, N. (2003). A flicker paradigm for inducing change
579 blindness reveals alcohol and cannabis information processing biases in social users.
580 *Addiction, 98*(2), 235-244. doi: 270 [pii]
581 Jones, B. T., & Schulze, D. (2000). Alcohol-related words of positive affect are more accessible in
582 social drinkers' memory than are other words when sip-primed by alcohol. *Addiction*
583 *Research, 8*(3), 221-232. doi: Doi 10.3109/16066350009004422
584 Knight, H.C., Smith, D.T., Knight, D.C. & Ellison, A. (2016). Altering attentional control settings
585 causes persistent biases of visual attention. *The Quarterly Journal of Experimental*
586 *Psychology* 96(1): 129-149.
587 Lusher, J., Chandler, C., & Ball, D. (2004). Alcohol dependence and the alcohol Stroop paradigm:
588 evidence and issues. *Drug and Alcohol Dependence, 75*(3), 225-231. doi: DOI
589 10.1016/j.drugalcdep.2004.03.004
590 Macleod, C., Mathews, A., & Tata, P. (1986). Attentional Bias in Emotional Disorders. *Journal of*
591 *Abnormal Psychology, 95*(1), 15-20.
592 McAteer, A. M., Curran, D., & Hanna, D. (2015). Alcohol attention bias in adolescent social drinkers:
593 an eye tracking study. Psychopharmacology, 232(17), 3183-3191.
594 Medina, K. L., McQueeny, T., Nagel, B. J., Hanson, K. L., Schweinsburg, A. D., & Tapert, S. F.
595 (2008). Prefrontal cortex volumes in adolescents with alcohol use disorders: Unique gender
596 effects. *Alcoholism-Clinical and Experimental Research, 32*(3), 386-394. doi: DOI
597 10.1111/j.1530-0277.2007.00602.x
598 Montgomery, C., Field, M., Atkinson, A. M., Cole, J. C., Goudie, A. J., & Sumnall, H. R. (2010).
599 Effects of alcohol preload on attentional bias towards cocaine-related cues.
600 *Psychopharmacology, 210*(3), 365-375. doi: DOI 10.1007/s00213-010-1830-y
601 Nikolaou, K., Field, M., Critchley, H., & Duka, T. (2013). Acute Alcohol Effects on Attentional Bias
602 are Mediated by Subcortical Areas Associated with Arousal and Salience Attribution.
603 *Neuropsychopharmacology, 38*(7), 1365-1373. doi: Doi 10.1038/Npp.2013.34
604 Noble, K. G., Korgaonkar, M. S., Grieve, S. M., & Brickman, A. M. (2013). Higher education is an
605 age-independent predictor of white matter integrity and cognitive control in late adolescence.
606 Developmental science, 16(5), 653-664.
607 Noel, X., Colmant, M., Van der Linden, M., Bechara, A., Bullens, Q., Hanak, C., & Verbanck, P.
608 (2006). Time course of attention for alcohol cues in abstinent alcoholic patients: The role of
609 initial orienting. *Alcoholism-Clinical and Experimental Research, 30*(11), 1871-1877. doi:
610 DOI 10.1111/j.1530-0277.00224.x
611 Ostlund, S. B., & Balleine, B. W. (2005). Lesions of medial prefrontal cortex disrupt the acquisition
612 but not the expression of goal-directed learning. *Journal of Neuroscience, 25*(34), 7763-7770.
613 doi: Doi 10.1523/Jneurosci.1921-05.2005
614 Pau, C. W., Lee, T. M., & Shui-Fun, F. C. (2002). The impact of heroin on frontal executive
615 functions. Archives of clinical neuropsychology, 17(7), 663-670.
616 Petry, N. M., Stinson, F. S., & Grant, B. F. (2005). Comorbidity of DSM-IV pathological gambling
617 and other psychiatric disorders: results from the National Epidemiologic Survey on Alcohol
618 and Related Conditions. The Journal of clinical psychiatry.
619 Prabhakaran, V., Narayanan, K., Zhao, Z., & Gabrieli, J. D. E. (2000). Integration of diverse
620 information in working memory within the frontal lobe. *Nature Neuroscience, 3*(1), 85-90.
621 doi: Doi 10.1038/71156
622 Ramnani, N., & Owen, A. M. (2004). Anterior prefrontal cortex: Insights into function from anatomy
623 and neuroimaging. *Nature Reviews Neuroscience, 5*(3), 184-194. doi: Doi 10.1038/Nrn1343
624 Ratti, M. T., Bo, P., Giardini, A., & Soragna, D. (2002). Chronic alcoholism and the frontal lobe:
625 which executive functions are impaired? *Acta Neurologica Scandinavica*, 105(4), 276-281.
626 doi: DOI 10.1034/j.1600-0404.2002.0o315.x
627 Roy-Charland, A., Plamondon, A., Homeniuk, A. S., Flesch, C. A., Klein, R. M., & Stewart, S. H.
628 (2017). Attentional bias toward alcohol-related stimuli in heavy drinkers: evidence from
629 dynamic eye movement recording. The American journal of drug and alcohol abuse, 43(3),
630 332-340.

Ryan, F. (2002). Detected, selected, and sometimes neglected: Cognitive processing of cues in addiction. *Experimental and Clinical Psychopharmacology, 10*(2), 67-76. doi: Doi 10.1037//1064-1297.10.2.67

Schoenmakers, T., Wiers, R. W., & Field, M. (2008). Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology, 197*(1), 169-178. doi: DOI 10.1007/s00213-007-1023-5

Seeley, W.W., Menon, V., Schatzberg, A.F., Keller, J., Glover, G.H., Kenna, H., Reiss, A.L. & Greicius, M.D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *The Journal of Neuroscience, 27*(9), 2349-2356. doi: 10.1523/JNEUROSCI.5587-06.2007

Sharma, D., Albery, I. P., & Cook, C. (2001). Selective attentional bias to alcohol related stimuli in problem drinkers and non-problem drinkers. *Addiction, 96*(2), 285-295. doi: DOI 10.1046/j.1360-0443.2001.96228512.x

Smith, D. G., Jones, P. S., Bullmore, E. T., Robbins, T. W., & Ersche, K. D. (2014). Enhanced orbitofrontal cortex function and lack of attentional bias to cocaine cues in recreational stimulant users. Biological psychiatry, 75(2), 124-131.

Sobell, L. C., & Sobell, M. B. (1992). Timeline Follow-Back - a Technique for Assessing Self-Reported Alcohol-Consumption. *Measuring Alcohol Consumption*, 41-72-228.

Stetter, F., Ackermann, K., Bizer, A., Straube, E. R., & Mann, K. (1995). Effects of disease-related cues in alcoholic inpatients: Results of a controlled "alcohol stroop" study. *Alcoholism: Clinical and Experimental Research*, 19, 593-599

Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: a conceptual view. *Psychological Research-Psychologische Forschung*, 63(3-4), 289-298. doi: DOI 10.1007/s004269900007

Swendsen, J. D., & Merikangas, K. R. (2000). The comorbidity of depression and substance use disorders. Clinical psychology review, 20(2), 173-189.

Sullivan, E. V., Rosenbloom, M. J., & Pfefferbaum, A. (2000). Pattern of motor and cognitive deficits in detoxified alcoholic men. *Alcoholism-Clinical and Experimental Research*, 24(5), 611-621. doi: DOI 10.1111/j.1530-0277.2000.tb02032.x

Townshend, J. M., & Duka, T. (2001). Attentional bias associated with alcohol cues: differences between heavy and occasional social drinkers. *Psychopharmacology, 157*(1), 67-74.

Uekermann, J., & Daum, I. (2008). Social cognition in alcoholism: a link to prefrontal cortex dysfunction? *Addiction*, 103(5), 726-735. doi: DOI 10.1111/j.1360-0443.2008.02157.x

Waters, H., & Green, M. W. (2003). A demonstration of attentional bias, using a novel dual task paradigm, towards clinically salient material in recovering alcohol abuse patients? *Psychological Medicine, 33*(3), 491-498. doi: Doi 10.1017/S0033291702007237

Winocur, G., & Moscovitch, M. (1990). Hippocampal and Prefrontal Cortex Contributions to Learning and Memory - Analysis of Lesion and Aging Effects on Maze-Learning in Rats. *Behavioral Neuroscience, 104*(4), 544-551. doi: Doi 10.1037/0735-7044.104.4.544

Yaxley, R. H., & Zwaan, R. A. (2005). Attentional bias affects change detection. *Psychonomic Bulletin & Review, 12*(6), 1106-1111.