The effects of alcohol consumption on attentional bias and impulsivity

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**ABSTRACT**

Impulsivity, a multi-dimensional construct, has been extensively explored within addiction research; with increased alcohol consumption repeatedly linked to heightened impulsivity. Similarly, greater attentional bias towards alcohol-related stimuli, commanding both initial and maintenance of attention, has been consistently reported in more accustomed alcohol drinkers. However, due to the lack of research combining these facets of addiction, the purpose of the present study was to explore the relationship between attentional bias and impulsivity; providing a more comprehensive understanding of alcohol-drinking in a sample of light and heavy drinkers ($N = 44$). Fundamentally, maintained attentional biases towards alcohol-related stimuli, measured using a visual dot-probe task, were significantly and positively correlated with trait impulsivity, using the Barratt Impulsivity Scale. Both maintenance and initial biases in alcohol-attention were positively correlated with state impulsivity of impaired inhibitory control in a ‘stop’ task; but were not correlated with impulsive choice in a delayed discounting paradigm. Furthermore, significantly increased craving was reported following alcohol cue-exposure in the visual dot-probe. Thus, supporting the notion that highly impulsive individuals are more susceptible to relapse due to biased attention towards alcohol-related cues; which elicits increased craving. Ultimately, knowledge of this relationship is essential to further inform effective rehabilitation interventions within addiction treatment; bringing this under-researched, complex relationship into the arena of literature.
Introduction

Alcohol consumption is a popular social phenomenon of increasing interest in current Western society. Recent statistics denote the problems associated with the United Kingdom’s excessive consumption and escalating addiction, suggesting that one in 25 adults are considered alcohol dependent (Department of Health, 2005). Alcohol intoxication has been linked to sexual risk-taking (Rehm, Shield, Joharchi & Shuper, 2012); risky driving behaviour (Transport Statistics, 2009); increased aggression (Duke et al., 2011) and most shockingly, contributes to approximately 33,000 deaths annually (Academy of Medical Sciences, 2004). Such implications of alcohol misuse are estimated to annually cost Britain £1.7 billion in health services and £6.4 billion due to loss of productivity in the workplace (Cabinet Office, 2003). This research clearly indicates that increased alcohol consumption has detrimental effects at both an interpersonal and societal level (Duke et al., 2011). Exploring the cognitive and motivational processes implicated in alcohol-drinking is essential to reduce the outlined problems.

Addiction theories have combined psychological and neurobiological concepts to explain the complexities of addiction. Robinson and Berridge (2000; 2003) in their theory of incentive sensitisation, postulate that global alterations occur within the nucleus accumbens (and related circuitry) following substance use, which mediates the psychological process of incentive salience. Such neural circuits become hypersensitised to the effects of the drug and associated stimuli, creating increased neurotransmission, particularly in the dopamine pathway. This hypersensitisation is argued to be the central underpinning of this theory; leading to a ‘pathological wanting’ and intense craving in the presence of drugs and drug-related cues, especially with repeated exposure/pairing. These cues then act as a powerful ‘conditioned incentive’ which grab an accustomed user’s attention becoming increasingly salient; termed ‘incentive salience attribution’ (Robinson & Berridge, 2003). Consequently, such cues can effectively trigger relapse, particularly as neuroadaptations have been found to remain altered long after drug cessation (Castner & Goldman-Rakic, 1999); rendering dependent users particularly vulnerable in their presence. This excessive ‘wanting’ induced motivational state, linked to the dysfunction of prefrontal cortical systems, is deemed fundamental in the transition from voluntary to involuntary drug-seeking and taking behaviour (Robinson & Berridge, 2000).

Within the literature, prominent links have been made between Robinson and Berridge’s theory of incentive sensitisation and the concept of attentional bias (Franken, 2003), described as: ‘the presence of triggering stimuli which may occur in the absence of awareness’ (Townshend & Duka, 2001, p.67). Numerous paradigms have been used to measure attentional bias; but predominantly feature the ‘stroop’ and ‘visual dot-probe’ task. In addiction paradigms, the stroop task involves the rapid colour naming of drug-related and neutral words; requiring participants to ignore word meaning and attend only to the colour ink they are presented in. Research argues that substance users (including heavy drinkers) are generally slower at naming drug-related, rather than neutral words, due to the incentive salience attached to the word’s meaning; capturing and distracting attention away from the primary task (Murphy & Garavan, 2011). However critics argue that the stroop task is an ambiguous measure of attentional bias, with suggestions that
slower colour naming could be plausibly explained by other factors, including: the emotionality of words presented (confounding emotional salience with alcohol salience); cognitive load or interference caused by suppressing word meaning (Klein, 2007). Similarly, Bauer and Cox (1998) found that alcohol-related words were distracting to both abusers and non-abusers. Subsequently, the elaboration of clearer measures has been highlighted as a necessary future direction within the field (MacKillop, 2006), with focus being placed on the visual dot-probe task (Field & Eastwood, 2005; Townshend & Duka, 2001). This paradigm centres on the assumption that individuals will respond faster to the probe which appeared in the visuospatial location that they were attending to, immediately prior to responding. Therefore if individuals respond faster to probes that replace alcohol-related images, compared to neutral images, then it is inferred that their visual-attention was directed at this cued-stimulus at the moment of image offset. Thus alcohol attentional bias is assumed (Field et al., 2010).

Fundamentally, the visual dot-probe allows for different levels of attention to be investigated. Spatial attention research has indicated that presentation times of over 150ms are required to enable individuals to disengage, or shift attention, from one simple cue to another (Theeuwes, 2005). Therefore presenting paired images for durations of 200ms allows for a brief attentional shift between both images, whereas longer durations allow for multiple shifts of attention. The duration between the onset of the cue and the response-stimulus, often termed ‘stimulus onset asynchrony’ (SOA; Chanon et al., 2010), determines whether biases in ‘initial orientation of attention’ (explored using short SOA’s) or ‘maintenance of attention’ (long SOA’s) are being examined. Research using the dot-probe has demonstrated greater attentional biases at the longest SOA’s of 500ms and 2000ms, in heavy drinkers compared to light drinkers; but not at SOA’s of 200ms (Field Mogg, Zetteler & Bradely, 2004; Townshend & Duka, 2001). This implies that maintenance of attention is the primary operating process in heavy drinkers; suggesting that the mere exposure of such stimuli may not create biased attraction, but rather the preoccupation with alcohol-related stimuli (Field & Eastwood, 2005). These findings highlight that even non-dependent individuals can demonstrate sensitivity to alcohol-related cues; challenging the notion that only accustomed substance users, with hypersensitised prefrontal reward pathways, experience this salience attribution (Robinson & Berridge, 2000). This further implies that initial experimentation can potentially lead to dependence with continued alcohol use.

Interestingly, Field et al., (2004) demonstrated that subjective alcohol craving was correlated with attentional bias at 2000ms across all participants- highlighting an association between craving and the continued attention towards such cues. Attentional bias is suggested to modulate subjective craving, having a mutually excitatory association (Franken, 2003) in which biases evoke further increases in craving within a positive feedback loop (Field & Powell, 2007). An assumption supported by Field and Eastwood (2005) who suggest a causal bi-directional relationship; contributing to escalated alcohol-craving and seeking behaviours in the presence of such cues. This has important implications as it demonstrates that attentional biases may play a vital role in the maintenance of substance use and relapse. Field, Kiernan, Eastwood and Child (2008) explored this concept when determining whether alcohol-related cues could elicit approach behaviours in light and heavy drinkers. Findings indicated that heavy drinkers were quicker to approach
alcohol-related images, compared to light drinkers, and that this bias was strongly associated with subjective alcohol-craving. Additionally, Field and Eastwood (2005) established that an ‘attend group’ of heavy social drinkers, trained to direct attention towards alcohol-related stimuli, demonstrated a significant increase in: attentional bias to alcohol-related cues, the urge to drink and the amount of alcohol consumed in a ‘taste testing’ session; in comparison to an ‘avoid group’. Both findings demonstrate that experimental manipulation of alcohol attentional bias can elicit craving, which in turn influenced individuals’ motivation to drink and increased the likelihood of consumption. This implies that heavy drinkers would be less likely to abstain from alcohol as they are more susceptible to attentional biases and increased craving, supporting the assumption that this relationship is mutually excitatory. This research positions subjective craving as an important factor to explore alongside attentional bias; with research consistently demonstrating increased craving following cue-exposure (MacKillop, 2006). Furthermore, craving measures (Bohn, Krahn & Staehler, 1995) have been demonstrated to be crucial predictors of relapse and therefore further investigation is necessary to improve clinical alcohol treatments (Flannery, Poole, Gallop & Volpicelli, 2003).

As well as reactivity to cued-stimuli; impulsivity is also considered a key concept in the understandings of addiction; with increased substance use repeatedly linked to heightened impulsivity across both state and trait measures (Field et al., 2010). Impulsivity, construed as a multi-faceted construct (Patton et al., 1995) has been defined as: ‘the predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or others’ (Moeller et al., 2001, p.1784). Trait measures of impulsivity, derived from personality research, include the well-established Barratt Impulsivity Scale (BIS-11; Patton et al., 1995) a self-report questionnaire exploring impulsivity along three core components: ‘motor’, ‘attention’ and ‘non-planning’ impulsiveness. The BIS-11 has been used in a diverse range of clinical populations, particularly substance use (Stanford et al., 2009), demonstrating greater impulsivity in alcohol dependent (Bjork, Hommer, Grant & Danube, 2004) and alcoholic populations (Dom, D’haene, Hulstijn & Sabbe, 2006).

Furthermore, Olmstead (2006) outlined two main processes implicated in impulsivity: ‘impulsive choice’, characterised by a lack of regard for negative future consequences, and ‘impaired inhibitory control’, characterised primarily by impulsive motor responses. Several approaches have been developed to explore these outlined facets of ‘state’ impulsivity, including ‘delay discounting’ and ‘stop’ paradigms. The laboratory paradigm ‘delay discounting’ investigates the tendency to discount larger delayed rewards in preference of smaller immediate rewards; with greater discounting indicative of increased impulsive choice (Johnson & Bickel, 2002). This is relative to that of a real-life situation whereby an individual chooses an immediate drug of choice over a drug-free lifestyle (Yi, Mitchell & Bickel, 2010). Johnson and Bickel (2002) explored this paradigm using monetary rewards in a computerised choice procedure; where a smaller, immediate monetary reward is pitted against a larger, delayed reward ranging from one day to 25 years.

Using this paradigm, Vuchinich and Simpson (1998) established that heavy and problem drinkers showered greater impulsive choice by discounting delayed rewards more than light drinkers. Similarly, Bjork et al. (2004) uncovered a higher rate of
delayed discounting in abstinent alcohol-dependent patients, compared to controls. Furthermore, Petry (2001) compared discounting within current and abstinent alcoholics, compared to controls, and found that active alcoholics discounted future outcomes more than controls; with abstinent alcoholics falling between the two. Exploring impulsivity across the spectrum of alcohol status, as these studies do, supports the notion that impulsivity increases with more experienced substance use, and is highest whilst in active addiction compared to abstinence. However, it is important to note that such differences have not been consistently observed. Dom, D’haene, Hulstijn, & Sabbe (2006) found little difference in monetary reward discounting between ‘late onset alcoholics’ and non-alcoholic controls. This was linked to the lack of controlling for cigarette use; unreported in both Dom et al., 2006 and Bjork et al., 2004. Smoking has been argued to confound results, obscuring whether heightened discounting is due to the effects of alcohol use or current cigarette smoking (Madden & Bickel, 2010). Consequently, the present study stipulates the recruitment of non-smoking participants.

Inhibitory control, often explored using the ‘Stop-IT’ task (Verbruggen & Logan, 2008), has been defined as: ‘the ability to inhibit a motor response that has already been initiated’ (Field et al., 2010, pg.1347). Within this task, reaction times to stimuli are recorded, along with the active inhibition of responses when an auditory stop signal is produced. More impulsive individuals, with impaired inhibitory control, are assumed to be unable to tolerate long stop signal delays (SSD) - the delay between the presentation of a stimulus and a stop signal. Therefore shortened SSD’s and prolonged stop signal response times (SSRT) are considered indicative of response inhibition deficits (Logan, Schachar & Tannock, 1997). Such deficits are deemed characteristic of substance abuse; with prolonged SSRT’s demonstrated in chronic: cocaine, methamphetamine and alcohol-dependent users, compared to controls (Verbruggen & Logan, 2008). Furthermore increased SSRT’s in high risk adolescents have been argued to later predict alcoholism; inferring that this may reflect differences occurring pre-substance abuse (Nigg et al., 2006). Unfortunately, these paradigms cannot establish whether impulsive individuals are more inclined to engage in substance use; or if impulsiveness is an effect of continued substance use.

Recent research has begun to explore attentional bias in conjunction with impulsivity; with increased levels of both concepts being ascertained in cocaine dependent individuals (Liu et al., 2011) and heavy/problem drinkers (Murphy and Garavan, 2011; Field, Christiansen, Cole & Goudie, 2007). Murphy and Garavan (2011) ascertained that increased attentional bias (during an alcohol stroop task); impaired inhibitory control and impulsive decision making were significant predictors of Audit scores for student problem drinkers; whom were twice as likely to have elevated levels than non-problem drinkers. Impulsivity measures were significantly correlated with alcohol attentional bias across the sample, indicating a cognitive profile whereby such deficits mediated the transition from early-drinking towards maintained problem-drinking. Moreover, Field et al. (2007) observed delayed discounting of hypothetical alcohol and monetary rewards and increased attentional bias (via an alcohol stroop task) in adolescent heavy drinkers- alongside the additional measure of subjective craving. In all participants delay discounting, attentional bias, alcohol craving and alcohol consumption were moderately correlated with one another. These findings support the notion that more impulsive individuals demonstrate
greater attentional bias, leading to greater cue-induced craving; consistent with the assumptions of incentive sensitisation (Robinson & Berridge, 2003). However due to the previously outlined limitations of the stroop task, these studies may benefit from replacing this measure of attentional bias in order to clarify and ground these arguments within reliable measures. Therefore the dot-probe task (Field et al., 2010) may prove a more effective measure within the present study, allowing for the exploration of both initial and maintenance of attention by presenting images for short (200ms) and long (2000ms) SOA’s.

Collectively, such research furthers the notion that attentional bias, impulsivity and subjective craving, combined, could offer a more coherent account of the complex relationship of processes underpinning alcohol-drinking behaviour. Continued research exploring the interlinking relationship between these facets of addiction is needed (Field & Cox, 2008), particularly in non-clinical samples (Murphy & Garavan, 2011) to draw attention to this under-researched area. Knowledge of such concepts is imperative to help inform alcohol interventions. On this basis, the aims of the present study were to establish whether an inter-play between impulsivity and attentional bias exists, along with the presence of cue-induced subjective craving, in a non-dependent sample of light and heavy drinkers. Consequently, it was hypothesised that heavy drinkers (males consuming >21 units and women >14 units weekly) would demonstrate greater attentional bias to alcohol-related stimuli as well as greater levels of impulsivity, as measured by the BIS-11, stop and delay discounting tasks, in comparison to light drinkers (<10 units). A direction supported by the outlined research. It was also hypothesised that there would be a significant, positive relationship between these concepts; postulating that as levels of impulsivity increase so does attentional biases to alcohol-related stimuli. Finally, subjective craving was investigated (using the Alcohol Urge Questionnaire); hypothesising that exposure to alcohol-related stimuli would increase reports of subjective craving, producing a greater increase in heavy drinkers, as postulated by Field et al. (2008).

Methodology

Design

The between groups’ independent variable (IV) of drinking status, having two levels: heavy and light drinker, was used throughout.

Impulsivity

Between groups designs were used to explore whether heavy drinkers demonstrated greater impulsivity, compared to light drinkers, across all impulsivity measures. The IV was drinking status; however the dependent variable (DV) differed according to the measure used. In the BIS-11 the DVs were the scores achieved on each of the three subscales: attention, motor and non-planning as well as total combined scores. In the delay discounting task the DV was the $k$-value of impulsive choice, produced for each participant. In the stop task: SSRT and SSD (measured in milliseconds) and the probability of responding on stop signal trials, were the key DVs used.
Attentional Bias

It was hypothesised that heavy drinkers would demonstrate greater attentional bias to alcohol-related stimuli compared to light drinkers, commanding both initial (200ms SOA) and maintenance of attention (2000ms SOA). This was examined using a 2 X 2 mixed design with one between groups IV of drinking status and one within groups IV of image type (neutral and alcohol-related). The DV was the reaction times (ms) to the stimuli presented at either 200ms SOA or 2000ms SOA.

Subjective Craving

It was hypothesised that exposure to alcohol-related stimuli would increase reports of subjective craving; producing a greater increase in heavy drinkers. This was assessed using a 2 X 2 mixed design with one between groups IV of drinking status, and one within groups IV of Alcohol Urge Questionnaire administration (AUQ), again with two levels: before and after cue exposure in the visual dot-probe. The DV measured was the scores achieved on the AUQ.

Associations

Finally, a correlational design was employed to explore the hypothesis that there would be a significant, positive relationship between attentional bias and impulsivity. Attentional bias scores were calculated for this design by measuring the difference in reaction times when the dot-probe was in the location of the neutral cue, to that of the alcohol-related cue. This bias was calculated for both SOA’s at 200ms and 2000ms and correlated with the following measures: mean scores of BIS attention, BIS motor, BIS non-planning, BIS total; SSD, SSRT, probability of responding on stop signal trials and k-values.

Participants

Forty four participants, including 18 males and 26 females, were recruited according to their drinking status through advertisements placed around Leeds Metropolitan University. The sample consisted of 17 light drinkers ($M = 21.47$, $SD = 3.56$) and 22 heavy drinkers ($M = 19.82$, $SD = 1.4$) all aged between 18 and 30 ($M = 20$). Light drinkers were recruited if they consumed <10 units of alcohol per week. Heavy drinkers were differentiated according to gender, recruiting males who consumed >21 units of alcohol weekly, and females >14 units weekly. This corresponds with the upper limits recommended by UK NHS guidelines (NHS Information Centre, 2010). Five participants did not fulfil such criteria and therefore were not assigned a status, but were still included in correlational analyses investigating the relationship between impulsivity and attentional bias. Undergraduate Psychology students received participant pool credits for taking part. Eligible participants included non-smokers with normal/corrected to normal colour vision that reporting drinking at least one unit of alcohol weekly. This inclusion criteria avoided the potential confound with nicotine addiction, which has already been well established to impact on measures of impulsivity (e.g. Dom et al., 2006). Those who described themselves, or anyone close to them, as having experienced difficulties in relation to alcohol were kindly asked not to partake.
Materials

Questionnaires

Demographic information was determined via a 4-item questionnaire, establishing each participants’ age, gender, smoking status and when they last consumed alcohol (appendix A).

Barratt Impulsivity Scale (BIS-11; Patton, Stanford & Barratt, 1995)

The BIS-11, a 30-item self-report questionnaire, was used to assess participants’ levels of trait impulsivity across three subscales (appendix B). These subscales included: ‘attention impulsiveness’ consisting of eight items (e.g. ‘I don’t pay attention’); ‘motor impulsiveness’ comprised of eleven items (e.g. ‘I do things without thinking’) and finally eleven items of ‘non-planning impulsiveness’ (e.g. ‘I plan tasks carefully’). Participants responded to all items using a 4-point Likert scale from ‘rarely’ to ‘always’. Eleven items were reversed scored during coding. Written directions were detailed above the questionnaire, instructing participants to answer promptly and honestly. Chronbach’s alpha (α = .81) indicated a high level of internal consistency within the present study; consistent with Stanford et al. (2009) reporting a similar Chronbach’s alpha (α = .83) as well as high levels of test-retest reliability and convergent validity.

Alcohol Urge Questionnaire (AUQ; Bohn, Krahn & Staeehler, 1995)

Alcohol craving levels were assessed using the AUQ (appendix C) with written permission granted by Dr Michael Bohn (appendix D). Written instructions appeared above the questionnaire, informing participants to answer with regard to their current feelings, whilst clarifying that questions referred to drinks containing alcohol. The questionnaire consisted of eight items including: ‘I crave a drink right now’ which were rated on a 7-point Likert scale from ‘strongly agree’ to ‘strongly disagree’. Two items were reversed scored during coding. Again a high level of internal consistency was demonstrated in the present study (Chronbach’s alpha = .908). This is consistent with previous research including Bohn et al., (1995) α = .9; Drummond and Phillips (2002) α = .93 and MacKillop (2006) α = .86.

Timeline Follow Back Procedure (TLFB; Sobell & Sobell, 1992)

Participants’ alcohol consumption was assessed via a four-week retrospective Timeline Follow back procedure, highlighting key dates, in which participants were asked to plot the units of alcohol they consumed (appendix E). An alcohol unit guide, produced using published NHS guidelines, was given to aid participants in their efforts to calculate their consumption (appendix F). The TLFB has been argued to be a reliable measure of alcohol consumption with good re-test reliability (Field & Powell, 2007) and high convergent and discriminant validity (Fals-Stewart et al., 2000).
Computerised Tasks

Visual Dot-Probe Task

A visual dot-probe task (as used by Field et al., 2004, 2011) was generated using E-Prime, version 1, on a 15-inch computer to investigate alcohol attentional bias. Instructions were presented on-screen in white, Arial, size 14 font on a black background (appendix G), directing participants to respond as quickly as possible to the upward or downward arrows presented, using the corresponding keys on the keyboard.

Each participant was presented with 40 picture pairs (appendix H). Each pair consisted of one alcohol-related image (e.g. individuals drinking alcohol) adjacent to one neutral image (e.g. individuals drinking water). Additionally a further 20 picture pairs of furniture were used as fillers, chosen for their non-descript content (appendix H). Images were closely matched for content, complexity, brightness and colour saturation (Field & Eastwood, 2005) and displayed against a black background; as were all instructions, fixation points and probes. Furthermore, images were digitalised and formatted (bitmap) to measure 100x125mm and positioned 60mm apart when visible on-screen. Picture pairs were presented for short and long SOA’s of 200ms and 2000ms measuring initial and maintenance of attention.

At the beginning of each trial a white central fixation cross was presented for 500ms to relocate focus to the centre of the screen. A 250ms delay then followed before the presentation of the picture pair. Once the picture pairs had been removed they were immediately replaced by the presentation of a dot-probe, either a white ‘upward’ or ‘downward’ arrow measuring 30mm on-screen, positioned in the centre of the space previously occupied by one of the images. Participants were instructed to indicate the location of the dot-probe, as quickly and accurately as possible using the corresponding ‘up’ and ‘down’ response arrows. Once a response had been made, the dot-probe was removed and participants were presented with a black screen for 1000ms; followed by the next trial.

Each of the picture pairs were presented four times, twice on the left and twice on the right of the screen at 200ms and 2000ms, allowing for every combination of picture and dot-probe to be generated- with the probe appearing at equal frequency in the location of the neutral and alcohol-related image. This totalled 240 trials, comprised of 80 filler and 160 critical trials. E-prime randomised the presentation order for each participant. All participants’ completed one practise block of 16 trials at the beginning of the experiment using four of the filler pairs. This was followed by two experimental blocks which were separated by a compulsory one minute break.

Stop Task (Logan, Schachar, & Tannock, 1997)

The computerised ‘Stop-IT’ task, ran using Windows XP, was used to measure behavioural disinhibition. Instructions were presented on-screen in white, Arial size 14 font on a black background (appendix I) in which participants were asked to either respond to the stimulus presented on screen, or actively inhibit a response to this stimulus when an auditory tone was produced. The researcher emphasised that participants should not wait for an auditory tone, but respond to stimuli as quickly as
possible. The stimuli presented were a series of squares and circles. When a square was presented participants were required to press the left response key using their left index finger and when a circle; the right response key using their right index finger. On 25% of trials an auditory stop tone was produced, after a variable stop signal delay (SSD), whereby participants were required to actively inhibit their response. The SSD was initially set at 250ms, but continually readjusted via a tracking procedure; increasing by 50ms after successful inhibition and decreasing by 50ms after unsuccessful inhibition; to obtain a 0.5 probability of stopping. All participants completed a practise block of 32 trials followed by four experimental blocks of 64 trials; each separated by a 10 second break in which feedback on response times were presented to participants.

As response inhibition is modelled as a race between the opposing demands of a go and stop process; SSRT is calculated as the ‘finishing time’ of this process minus SSD (Logan et al., 1997). Essentially, when SSD increases, the probability of responding on stop signal trials, p(r/s), also increases (Verbruggen & Logan, 2008). Therefore impulsive individuals are unable to tolerate long SSD’s and demonstrate prolonged SSRT’s. These key dependent variables were extracted from the ‘Stop-IT’ output.

Delay Discounting Task (Johnson & Bickel, 2002)

A computerised delay discounting task was used to measure impulsive choice. On-screen instructions, presented in size 14 black font on a grey background (appendix J), outlined that participants were required to choose between an immediate or delayed hypothetical monetary reward, for example: ‘Would you prefer £660 right away or wait one day and receive £1000?’ (Appendix K). Using the computer mouse to respond, participants chose between: the left command button, ‘option 1’, which displayed an immediate reward or the right command button, ‘option 2’, displaying the delayed reward set consistently at £1000. Participants were presented with numerous hypothetical options in seven experimental blocks of different time increments: one day, one week, one month, six months, one year, five years and 25 years. A circular icon positioned above the command buttons remained in the centre of the screen throughout; displaying as green when the response commands could be registered, but turning red for two seconds once a selection had been made. This disabled the command buttons, forcing participants to wait and consider the next options thoughtfully.

In this paradigm, the magnitude of the smaller, immediate reward was adjusted until the subjective value of both rewards was approximately equal; termed the indifference point for that delay. These indifference points were used to calculate the rate at which the delayed rewards were discounted, termed the ‘free parameter: k’, in which higher k-values demonstrated more rapid discounting of delayed rewards; inferring increased impulsive choice (Murphy & Garavan, 2011). When applying the exponential model, the value of the delayed reward is exponentially discounted, in equal proportion, so that with each delay there is a set decrease in reward value (Kirby, 1997). This can be expressed in the following equation:

\[ V = Ae^{-kD} \]
In figure (1): V is the current discounted value of the delayed reward, termed the indifference point; A symbolises the amount of the delayed reward; k denotes the rate of discounting (an empirically derived constant, scaled according to the degree of discounting) and D is the delay of the reward (Kirby, 1997). These indifference points can then be plotted on a graph for each participant, allowing the calculation of the k and R² value (denoting goodness of fit) to be deduced.

Procedure

As alcohol is known to impact on reaction times (Anderson et al., 2011; Siliquini et al., 2011), participants were asked not to consume alcohol 24 hours prior to testing. All participants were tested individually, between 10am and 5pm, in a designated laboratory at Leeds Metropolitan University. One researcher accompanied all participants during testing. All participants read an information sheet (appendix L) detailing the procedure involved and confidentiality regulations in place. Each individual was assigned a participant identification number, noted at the top of the consent form (appendix M) which participants signed before the study commenced.

Once seated, 1m away from the computer monitor, participants completed the demographic questionnaire, followed by the BIS-11. All participants then completed the computerised ‘Stop’ and ‘Delay Discounting’ task, which were counterbalanced across participants, to counteract order effects. The AUQ was then administered to each participant, prior to alcohol-cue exposure. Following this, participants were then seated on a different computer to complete the visual dot-probe. Hand positioning on this task was counterbalanced across participants in attempt to avoid reaction time differences due to dominant hand response. Therefore 50% of participants responded to the ‘upward’ arrow using their right index finger and the ‘downward’ arrow with their left index finger, and 50% vice versa. Participants then completed the AUQ for the second time, determining craving post-cue exposure. The TLFB was completed at the end of the procedure, to avoid demand characteristics or preoccupation with reported alcohol consumption whilst completing the tasks. Following the completion of the study all participants were thanked and given a full written debrief (appendix N). This explained the concept behind the study; reminded participants of their entitlement to withdraw their data and outlined the relevant support sources available. The study took each participant approximately 50 minutes to complete and was granted Ethical approval from the School of Psychology’s ethics committee at Leeds Metropolitan University (appendix O).

Data Analysis

The general assumptions of normality distribution and homogeneity of variance were explored for each variable using Shapiro Wilk and Levene’s tests, respectively. Non-normal data, where possible, was Log10 transformed.

Trait and State Impulsivity

Mean scores were calculated for trait impulsivity BIS-11 subscale scores, and state impulsivity measures including: delay discounting k-values and stop task task measures
of SSRT, SSD and p(r/s). These were then analysed by Independent groups t-tests to determine whether differences were apparent in terms of drinking status.

**Attentional Bias**

The dot-probe task was analysed by calculating the mean reaction times (RTs), on critical trials, for each probe position (replacing neutral or alcohol-related images) and at the duration in which it occurred (i.e. at 200ms or 2000ms). ‘Bias scores’ were then established by subtracting RTs to probes that replaced alcohol-related images from RTs to probes replacing neutral images; whereby positive scores were indicative of alcohol attentional bias (Field & Eastwood, 2005). Trials in which participants reaction times were >2000ms, and trials with errors, were removed from analysis (Field & Eastwood, 2005). Consequently 4.09% of data was removed. Two 2x2 mixed ANOVA’s, with a between factor of drinking status and within a factor of image type, at SOA’s of 200ms and 2000ms were conducted; exploring the effects of drinking status on reaction times to alcohol-related and neutral stimuli.

**Subjective Craving**

Subjective craving was explored by calculating total AUQ scores for the first and the second administration, following exposure to alcohol-related stimuli. This was analysed using a 2x2 mixed ANOVA with a between factor of drinking status and a within factor of administration time; exploring differences in craving with regard to drinking status.

**Associations**

Spearman’s rho and Pearson’s correlations were then implemented to explore the relationship between attentional ‘bias scores’ and mean trait and state impulsivity measures, as described.

**Results**

Normality and homogeneity of variance tests were carried out on all variables, and were assumed if the significance level of the Shapiro-Wilk and the Levene’s test, respectively, was > 0.05 (Miles & Shevlin, 2001). Homogeneity of variance was not violated and data that was non-normal was transformed using a Log10 transformation. Where non-normal data was unsuccessfully transformed (e.g. BIS-11 attention subscale; reaction times to neutral stimuli and AUQ 2nd administration scores) equivalent non-parametric analyses were performed, or ANOVA’s were proceeded with, due to their robust nature against violations of normality (Field, 2009).
Trait Measures of Impulsivity: Difference Between Heavy and Light Drinkers

BIS-11
Descriptive statistics for the total and subscale scores of the BIS-11 are presented in Figure 1.

Figure 1: Mean scores for BIS-11 subscales for heavy and light drinkers (error bars demonstrating SD of the mean)
* Significantly different from light drinkers at .05 level

Figure 1 indicates that heavy drinkers scored marginally higher than light drinkers across all subscales and total scores. Error bars further suggest that variances in scores were greater for light drinkers compared to heavy drinkers. An Independent samples t-test was performed on mean BIS-11 total scores, motor and non-planning subscales and a Mann-Whitney U on the attention subscale.

On average heavy drinkers had greater total BIS-11 scores ($M = 68.73$, $SD = 11.39$) compared to light drinkers ($M = 64.41$, $SD = 15.89$), but this difference was not significant ($t(37) = 1.24$, $p > .05$). Greater motor impulsivity was reported in heavy drinkers ($M = 25$, $SD = 4.88$) compared to light drinkers ($M = 24$, $SD = 5.33$). Again, this difference was not statistically significant ($t(37) = 0.67$, $p > .05$). Heavy drinkers demonstrated greater non-planning impulsivity ($M = 23.53$, $SD = 7.5$), although this difference was not statistically significant ($t(37) = 0.68$, $p > .05$). Heavy drinkers reported greater attention impulsiveness ($M = 18.77$, $SD = 3.19$) compared to light drinkers ($M = 16.88$, $SD = 4.6$). A Mann-Whitney U test revealed that this difference was statistically significant ($U = 120.5$, $p < .05$).
State Measures of Impulsivity: Differences Between Heavy and Light Drinkers

Delay Discounting

Mean $k$-values and standard deviations for both drinking status conditions are displayed in Table 1.

Table 1
Means and standard deviations for $k$-values according to drinking status

<table>
<thead>
<tr>
<th></th>
<th>Delay discounting $k$-values</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Light drinkers</td>
<td>.33</td>
<td>.24</td>
</tr>
<tr>
<td>Heavy drinkers</td>
<td>.34</td>
<td>.20</td>
</tr>
</tbody>
</table>

Table 1 demonstrates that heavy drinkers had marginally higher $k$-values than light drinkers; indicative of increased discounting of delayed rewards. Standard deviations indicate a large variance in $k$-values of impulsive choice in both groups; although slightly greater in light drinkers. An independent groups $t$-test revealed that this difference was non-significant $t(37) = .03$, $p > .05$.

Mean $R^2$ values for heavy ($M = .80$, $SD = .12$) and light drinkers: ($M = .70$, $SD = .13$) demonstrate a relatively poor fit to data, particularly in light drinkers. Furthermore, a statistically significant difference in $R^2$ values between light and heavy drinkers was established ($t(37) = 2.42$, $p < .05$) which indicates that the model did not fit the data equally across groups.

Stop Task

Means and standard deviations of SSRT’s and SSD’s are presented in Table 2.

Table 2
Means and standard deviations for stop task variables according to drinking status

<table>
<thead>
<tr>
<th></th>
<th>SSD</th>
<th>SSRT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Light drinkers</td>
<td>273.36</td>
<td>157.33</td>
</tr>
<tr>
<td>Heavy drinkers</td>
<td>273.30</td>
<td>132.21</td>
</tr>
</tbody>
</table>

Table 2 outlines that light drinkers had greater SSRT’s and marginally greater SSD’s than heavy drinkers; suggesting that light drinkers could tolerate longer delays between stimuli and stop signals, but also had prolonged reaction times. Standard deviations demonstrate that variances in reaction times across both stop-variables were greater for light drinkers compared to heavy drinkers. The difference between heavy drinkers and light drinkers mean SSRT’s was not statistically significant $t(37) = -1.12$, $p > .05$. Similarly, heavy drinkers marginally lower mean SSD’s, compared to
light drinkers, was not significantly different $t(37) = -.001, p > .05$. This refutes the experimental hypothesis.

The mean probability of responding on stop signal trials are depicted below in Table 3.

**Table 3**
**Means and standard deviations for the probability of responding on stop signal trials in light and heavy drinkers**

<table>
<thead>
<tr>
<th></th>
<th>Mean probability of responding on stop signals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
</tr>
<tr>
<td>Light drinkers</td>
<td>53.72</td>
</tr>
<tr>
<td>Heavy drinkers</td>
<td>53.40</td>
</tr>
</tbody>
</table>

As illustrated in Table 3, heavy drinkers mean probability of responding on stop signals was marginally lower than light drinkers, indicating that heavy drinkers were only fractionally more likely to inhibit a response on stop-signal trials. An Independent samples Mann-Whitney $U$ Test revealed that this difference was not significant ($U = 201, p > .05$).

**Attentional Bias: Differences Between Heavy and Light Drinkers**

**Visual Dot-Probe**

Means and standard deviations of reaction times (RT) to visual dot-probe stimuli, at their allotted exposures, are shown below in Table 4.

**Table 4**
**Means and standard deviations of RTs to dot-probes according to drinking status at 200ms and 2000ms**

<table>
<thead>
<tr>
<th></th>
<th>RT to neutral stimuli at 200 SOA</th>
<th>RT to alcohol stimuli at 200 SOA</th>
<th>RT to neutral stimuli at 2000 SOA</th>
<th>RT to alcohol stimuli at 2000 SOA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>Light drinkers</td>
<td>596.98</td>
<td>90.14</td>
<td>599.16</td>
<td>92.33</td>
</tr>
<tr>
<td>Heavy drinkers</td>
<td>562.08</td>
<td>54.15</td>
<td>567.26</td>
<td>59.31</td>
</tr>
<tr>
<td>Total sample</td>
<td>577.29</td>
<td>73.14</td>
<td>581.17</td>
<td>76.09</td>
</tr>
</tbody>
</table>

As presented in Table 4, heavy drinkers demonstrated faster reaction times across both SOA’s (200ms and 2000ms) and to both image types (neutral and alcohol) compared to light drinkers. Standard deviations demonstrate consistently greater variances in light drinkers' reaction times at both SOA’s and image types. However, mean total reaction times across the entire sample indicate that responses to neutral
stimuli were marginally faster than to alcohol-related stimuli at SOA 200ms; but slower at 2000ms.

Reaction times in the visual dot-probe task were analysed using two, 2x2 mixed design ANOVA’s, one performed at SOA 200ms and one at 2000ms, exploring drinking status and image type. At SOA’s of 200ms, a non-significant main effect for image type $F(1, 37) = 1.05, p > .05, \eta^2 = .17$ was revealed. There was no main effect of drinking status ($F(1, 37) = 2.02, p > .05, \eta^2 = .23$) and no significant interaction between image and drinking status ($F(1, 37) = .17, p > .05, \eta^2 = .07$). In the 2000ms exposure, no significant main effect for image type was established ($F(1, 37) = .13, p > .05, \eta^2 = .06$). Additionally, no significant main effect was found for drinking status ($F(1, 37) = 3.92, p > .05, \eta^2 = .31$). However this effect was approaching significance ($p = .055$) implying that heavy drinkers were demonstrating faster reaction times to stimuli presented, with $\eta^2$ revealing a medium effect size (Cohen, 1988). No significant interaction effect for image and drinking status was established ($F(1, 37) = 1.49, p > .05, \eta^2 = .2$).

**Subjective Craving**

Mean AUQ scores for both drinking status conditions are illustrated in Figure 2.

![Figure 2: Mean AUQ scores before and after attentional bias cue exposure, according to drinking status (SD presented as error bars)](image)

** Significant main effect of drinking status at .01 level

As illustrated in Figure 2, mean AUQ scores increased from the first admission to the second admission, post alcohol-cue exposure, in both light and heavy drinkers. Variability in the data appears comparable in heavy drinkers but not light drinkers. Additionally, heavy drinkers generally reported greater AUQ scores both before ($M = 24, SD = 9.02$) and after cue exposure ($M = 28.68, SD = 10.1$), compared to light
drinkers, before ($M = 14.94, SD = 5.89$) and after ($M = 20.29, SD = 13.26$) alcohol-related exposure.

A 2 X 2 ANOVA was performed, comparing drinking status and AUQ scores between the first and second administration. Analysis revealed a significant main effect of administration time ($F(1, 37) = 13.11, p < .01$) with a large effect size (eta squared = .51), indicating that scores were significantly higher on the second AUQ administration after exposure to alcohol-related images, than before exposure. The main effect of drinking status was statistically significant $F(1, 37) = 9.22, p < .01$, eta squared = .45 whereby heavy drinkers demonstrated significantly increased AUQ scores, of a medium effect size (Cohen, 1988). There was not a significant interaction between administrations of the AUQ and drinking status $F(1, 37) = .06, p > .05$, eta squared = .04.

**Correlational Analyses**

Attentional bias scores were calculated for both exposure durations (200ms and 2000ms). Shapiro Wilk analyses revealed that ‘bias score 200ms’ was normally distributed whereas ‘bias score 2000ms’ was not. Therefore both Pearson’s and Spearman’s Rho correlations were used to explore the relationship between attentional bias and the measures of impulsivity, as illustrated below. Parametric analyses were run only on the data which did not violate normality assumptions.

**Table 5**

<table>
<thead>
<tr>
<th></th>
<th>Attention Motor</th>
<th>Non-planning Motor</th>
<th>BIS-11 Totals</th>
<th>Bias score 200ms</th>
<th>Bias score 2000ms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attention</strong></td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Motor</strong></td>
<td>.621**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Non-planning</strong></td>
<td>.549**</td>
<td>.59**</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>BIS-11 Totals</strong></td>
<td>.785**</td>
<td>.853**</td>
<td>.871**</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Bias score 200ms</strong></td>
<td>.021</td>
<td>.119</td>
<td>-.038</td>
<td>-.031</td>
<td>-</td>
</tr>
<tr>
<td><strong>Bias score 2000ms</strong></td>
<td>.439**</td>
<td>.356**</td>
<td>.256*</td>
<td>.367**</td>
<td>.097</td>
</tr>
</tbody>
</table>

* Spearman’s Rho correlation, significant at .05 level, ** Spearman’s Rho correlation, significant at .01 level
Bias scores at SOA 200ms did not significantly correlate with any of the BIS-11 subscales. However bias scores at SOA 2000ms significantly correlated with total BIS-11 scores and all subscales: attention, motor and non-planning. Each demonstrating a positive correlation; indicating that attentional biases increase with impulsivity levels, when exposed to images for 2000ms.

The correlation coefficients between bias scores and delay discounting k-values and stop task variables (SSD, SSRT and probability of responding) are displayed in Table 6.

**Table 6**
Correlation matrix of delay discounting and stop task variables (state measures of impulsivity) with attentional bias scores at SOA’s of 200ms and 2000ms

<table>
<thead>
<tr>
<th></th>
<th>SSD</th>
<th>SSRT</th>
<th>Probability of responding on stop signal trials</th>
<th>k-value</th>
<th>Bias Score 200ms</th>
<th>Bias Score 2000ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSD</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>SSRT</td>
<td>-.487**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Probability of responding on stop signal trials</td>
<td>-.736**</td>
<td>.402**</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>k-value</td>
<td>.047</td>
<td>-.103</td>
<td>-.179</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bias Score 200ms</td>
<td>-.305*</td>
<td>.271*</td>
<td>.419**</td>
<td>.165</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Bias Score 2000ms</td>
<td>-.034</td>
<td>.283*</td>
<td>.046</td>
<td>-.161</td>
<td>.097</td>
<td>-</td>
</tr>
</tbody>
</table>

* Spearman’s Rho correlation, significant at .05 level, ** Spearman’s Rho correlation, significant at .01 level, • Pearson’s correlation, significant at .05 level

Bias scores at 200ms significantly correlated with SSD and the probability of responding on stop signal trials. A medium, negative correlation with SSD indicates that the greater the attentional bias score at 200ms the smaller the SSD i.e. the more impulsive the individual. A significant positive correlation with ‘probability of responding’ suggests that the higher the probability of responding on stop signal
trials (i.e. the more impulsive an individual) the greater the attentional bias score towards alcohol-related images at 200ms.

Additionally, bias scores showed a significant positive correlation with SSRT at both 200ms and 2000ms. This suggests that increased attentional bias for alcohol-related images, at both exposures, is associated with increases in SSRT; indicative of impaired inhibitory control. No significant correlations were found between bias scores and delay discounting k-values.

Discussion

The present findings demonstrate that heavy drinkers were not statistically more impulsive than light drinkers on BIS-11 total scores, motor or non-planning subscales. This is inconsistent with the present hypothesis and previous research (Bjork et al., 2004; Dom et al., 2006). Such findings may reflect the issues concerning self-report impulsivity measures; criticised for being unable to decipher objective understandings of behaviour, unlike operationally defined laboratory paradigms which are uninfluenced by subjective bias (de Wit, 2009). Although the BIS-11 is considered the most commonly used trait impulsivity measure (Spinella, 2007) not all findings have consistently supported its 3-factor structure in different clinical populations (Ireland & Archer, 2008). Such limitations of the BIS-11 need further clarification; as it may go some way to explain the lack of difference found between drinking status and overall impulsivity scores.

However, heavy drinkers did demonstrate significantly increased BIS-11 cognitive attentional impulsivity, compared to light drinkers. de Wit (2009) suggests that when attempting to avoid a substance, inattention or the inability to sustain attention/focus, increases the chance of drug consumption; as even momentary attentional lapses can lead to the initiation of substance use or relapse. This might explain why heavy drinkers engaged in consuming alcohol on a continued weekly basis; highlighted in the TLFB procedure. Collectively, BIS-11 findings support de Wit’s (2009) proposal that inattention might be an entirely separate, underlying facet of impulsive behaviour (as is inhibitory control); hence why differences were found along this dimension and not BIS-11 subscales combined. As impulsivity is still not clearly defined within the literature (de Wit, 2009) research findings across studies have been inconsistent and contradictory (Leshem & Glicksohn, 2007). Therefore it is evident that the nature of this effect needs further exploration; particularly whether attentional impulsivity is a dispositional predictor of increased alcohol consumption or whether increased consumption impacts on attentional systems of impulsivity (de Wit, 2009).

No significant difference in impulsive choice was established between heavy or light drinkers; discounting delayed rewards at a comparable rate, contrary to previous research demonstrating greater discounting in heavy (Vuchinich & Simpson, 1998) and dependent drinkers (Bjork et al., 2004; Petry, 2001). As differences were not established in delayed discounting or BIS-11 non-planning, this indicates that impulsive choice, characterised by investment in future outcomes, is not a prominent
construct in the present sample and may not influence decisions regarding alcohol consumption. Research suggests that young individuals have lower risk perception of the short and long-term implications of alcohol consumption (Boluarte et al., 2011) and tend to engage in ‘risky drinking’ (Ayers & Myers, 2012). This raises the question as to whether young individuals are generally more impulsive, regardless of drinking status. The present study sampled individuals aged 18-30 ($M = 20$); therefore if elevated impulsivity features more generally within a young population, this may have hindered the finding of group differences. This emphasises the need to explore impulsivity and drinking status across different ages; particularly calling for longitudinal studies, as those which exist have tended to focus on drug use alongside trait and not state measures of impulsivity (de Wit, 2009).

Kirby and Petry (2004) also reported a lack of difference in delay discounting between controls, abstinent and active alcoholics. The leniency of the criteria by which alcoholics were classified was considered at fault, by not producing significant group differences. The present study may have also encountered such problems, as Field et al. (2011) highlights the limitations of categorising alcohol consumption, a continuous variable, into the grouping variables of ‘light’ and ‘heavy’. Furthermore, grouping both males and females into the same light drinking category (<10 units, Field et al., 2004) may not have generated accurate group differences. Gill (2002) suggests that males drink in excess of females; highlighting the importance of controlling for gender as this could potentially confound results. The accuracy of reported alcohol consumption is also questionable, due to issues surrounding social desirability bias in revealing daily units and the inherent difficulties of retrospective recall. Therefore implementing weekly diary entries, rather than the 4-week TLFB, may produce more accurate, unbiased recall (Hoeppner et al., 2010).

In the present study, the exponential model was applied in order to determine $k$ and $R^2$ values in the delay discounting paradigm. Mean $R^2$ values demonstrated a particularly poor fit for light drinkers, and a statistically significant difference in $R^2$ values between light and heavy drinkers. These findings suggest that the model did not accurately describe the effect of the data, with unexpected variance in the goodness of fit between drinkers. This may contribute to explaining why differences were not found in impulsive choice across groups; exposing a potential flaw in the experimental design. The hyperbolic model has been argued to more accurately establish $k$-values (Vuchinich & Simpson, 1998) and provide better fit to data (Coffey et al., 2003). Therefore the use of this function is suggested for future delay discounting research. Furthermore, impulsive choice is postulated to become less prominent with age, especially within delay discounting paradigms (Reynolds, 2006) and hence age may be an important factor to consider here, as previously discussed.

No significant differences in inhibitory control were established between light and heavy drinkers in the stop task, refuting the experimental hypothesis. These findings suggest that impaired inhibitory control is not a relevant construct in heavy or light drinkers and appears inconsistent with previous research (Verbruggen & Logan, 2008; Murphy & Garavan, 2011). However these findings may reflect the limitations of the stop task, being sensitive only to behavioural inhibition in alcohol-dependency. Therefore it might not be plausible to compare the present design to Verbruggen and Logan’s (2008) research, which revealed differences between alcohol-dependent
individuals and controls. Similarly, it is also difficult to relate to findings which used the go-no-go task (e.g. Murphy and Garavan, 2011). Eagle, Bari and Robbins (2008) suggest that different neurochemical systems are involved in the stop and go-no-go task, and hence tap into different forms of inhibition, despite both being perceived as generally measuring ‘inhibitory control’. Again this may help in explaining the discrepancy between the current results and that of previous research.

Furthermore, it was hypothesised that heavy drinkers would demonstrate greater attentional bias to alcohol-related stimuli in the visual dot-probe, commanding both initial orientation and maintenance of attention. However results did not reveal such differences, as no significant main effects were found for drinking status or image type at either SOA. This is inconsistent with Field et al., (2004) and Townshend and Duka (2001) who demonstrated increased maintenance of attention to alcohol-related stimuli in heavy drinkers. Interestingly, the effect of drinking status at 2000ms was approaching significance in the present study. Therefore, a larger sample size may have established this difference by increasing the power of the effect. Certain limitations may further account for such discrepancies, for example, in the attentional bias task, participants responded to the dot-probes using both index fingers. This arrangement may have produced a dominant hand bias, despite counterbalancing efforts, and ultimately may not sensitively measure reaction times. Additionally, heavy drinkers were consistently faster to respond to both image types. This suggests that floor effects may have occurred; whereby heavy drinkers’ speed of responses could not increase any further to demonstrate significant alcohol attentional bias. Therefore the use of a response box (Field et al., 2008), replacing the standard keyboard, is a necessary future direction in attempts to improve accuracy in reaction time measurement. Furthermore, Schmukle (2005) sparked debate when testing the reliability of the visual dot-probe; arguing that this task was neither an internally consistent or stable measure of attentional bias in non-clinical samples. Therefore, utilising an alternative method such as the eye-tracker, deemed an ‘unambiguous measure’ of attentional bias (Field et al., 2011), may improve the accuracy of results.

As hypothesised, results demonstrated increased craving following exposure to alcohol-related stimuli in both light and heavy drinkers. Additionally, heavy drinkers consistently reported higher subjective craving compared to light drinkers; consistent with MacKillop (2006) and Field et al. (2008). However no interaction effect was established, as both light and heavy drinkers’ craving levels increased proportionally, following cue-exposure. Robinson and Berridge (1993) postulate that the conditioned, reinforcing properties of substance-related stimuli produce intense craving in their presence; consistent with the significant results established. However this effect is primarily assumed in experienced, long-term substance users. Myrick et al. (2004) demonstrated that alcoholics but not social drinkers experienced increased craving and brain activation in the central reward-circuitry, when presented with alcohol-related images. Consequently, the present findings have important implications; highlighting that cue-induced craving occurs even in non-dependent individuals. This illustrates the potential for alcohol use to escalate with continued exposure (Field et al., 2010) and could denote a trend towards future problematic drinking; which positions craving as a crucial construct of exploration in attempts to improve clinical rehabilitation programmes.
Correlational analysis indicated that BIS-11 total scores and subscales of non-planning, attention and motor impulsiveness were all correlated with attentional bias scores at 2000ms. However none of the BIS-11 subscales correlated with bias scores at 200ms. This suggests that all dimensions of trait impulsivity are associated with maintenance of attention to alcohol-related stimuli; but not initial orientation. Consequently, it can be argued that substance-related cues hold the attention of those who are innately more impulsive; inferring that these individuals would be more susceptible to relapse because they attend to such stimuli (Franken, 2003). Alcohol-related stimuli might also maintain attention by evoking anticipatory reward and even alcohol expectancy (Field et al., 2011) in innately impulsive individuals. However, as little research has explored the relationship between attentional bias and trait impulsivity, it is difficult to make thorough comparisons with other findings.

Attentional bias scores were significantly correlated with all stop task measures at 200ms. This association between impaired inhibitory control and biases in initial orientation implies that alcohol-related cues elicit stronger incentive salience (i.e. the ‘grabbing of attention’) in motor impulsive individuals. Bias scores at 2000ms were correlated with SSRT, which suggests that maintenance of attention towards alcohol-related images was strongly associated with prolonged reaction times; again indicative of behavioural disinhibition. Individuals who demonstrate impaired inhibitory control may be unable to inhibit motivations of alcohol seeking, especially as craving increased post-cue exposure; hence why attention was not only ‘grabbed’ but maintained in their presence. Findings thus far support the present hypothesis that highly impulsive individuals, both in trait and state levels, would demonstrate greater attentional bias to alcohol-related stimuli; inferring that impulsive individuals may ‘seek out and attend to’ alcohol images due to their incentive salience (Robinson & Berridge, 1993).

Attentional bias scores were not significantly correlated with impulsive choice (k-values) at either 200ms or 2000ms. This is inconsistent with Field et al. (2007) and Murphy and Garavan (2011) and may relate to the issue of age, as discussed. However, as these studies utilised the stroop task to measure attentional bias it is difficult to draw comparisons between findings. Interestingly, alcohol attentional bias is related to impaired inhibitory control (stop task) but not impulsive choice (delay discounting). These concepts are considered independent of one another (de Wit, 2009) with neurobiological evidence suggesting that dysfunction of the frontal striatal system is primarily associated with impaired inhibitory control; whereas deficits in the orbitofrontal and prelimbic cortex are related to impulsive choice (Jentsch & Taylor, 1999). However as the present study investigated light and heavy drinkers that have not transitioned into dependency, it would appear that they have not been exposed to heavy, long-term alcohol use in order to bring about the neuroanatomical changes implicated in impulsive behaviours (Jentsch & Taylor, 1999). Nevertheless, both light and heavy drinkers experienced significantly increased craving, despite not experiencing an allostatic state due to the dysregulation of reward systems, as theorised by Koob and Le Moal (2008). Therefore increased information is needed regarding the neural mechanisms at play in non-clinical samples (Jentsch & Taylor, 1999). Establishing how long participants have been drinking for would determine whether impaired inhibitory control, and its association with attentional bias, hints at a potential trend towards problem drinking. Consequently, future research may
benefit from replicating the present study in dependent individuals; broadening the understandings of attentional biases and their link to impulsivity throughout the cycle of addiction. Additionally, due to research constraints, the present study could only control for smoking. Ideally, future research should also control for illicit substances to ensure that this is not affecting the results obtained.

Interestingly, the present findings demonstrate that trait impulsivity relates only to biases in maintenance of attention; suggesting that innate impulsiveness relates to the preoccupation with alcohol-related stimuli. Whereas impaired inhibitory control, a situational facet of state impulsivity, is associated with alcohol-related stimuli’s ability to grab initial attention and maintain this attention. This complex relationship between different biases and dimensions of impulsivity requires further exploration. Improved knowledge could potentially highlight the factors involved in the transition from voluntary to involuntary drinking (Robinson & Berridge, 2003) especially as both trait and state impulsivity are associated with biased attention. However, due to the comparative relationship determined between impulsivity measures, attentional bias and alcohol use, causation cannot be established in the present study. Therefore directionality cannot be assumed as to whether impulsive individuals are more susceptible to alcohol attentional bias or whether attentional biases influence an individual’s state impulsivity; issues similarly raised by Doran, Spring and McChargue (2007) in relation to smoking. Consequently, further research into the nature of this relationship, alongside subjective craving, is needed; building on the recent findings by Papachristou et al. (2012) whom argue that the association between response inhibition and increased cue-induced alcohol craving can be considered a risk factor motivating consumption.

Fundamentally, the present findings support the hypothesis that interplay exists between attentional bias and certain facets of state and trait impulsivity. Such findings may explain why highly impulsive individuals relapse (Jentsch & Taylor, 1999); as they more readily attend to alcohol-related cues in their environment, which elicits increased craving; motivating alcohol-seeking behaviours. Identifying those most at risk, i.e. highly impulsive individuals, would help to inform pharmacological and behavioural intervention strategies, such as: reducing exposure to alcohol-related stimuli during treatment, teaching alternative ways to respond to this stimuli and attention diversion techniques (Fadardi & Cox, 2009). Interlinking both facets implicated in alcohol-drinking behaviour may prove a more effective strategy. Ultimately, insight into the complex relationship between these concepts will help develop comprehensive preventative and rehabilitation schemes; needed to combat society’s escalating alcohol-related problems.
References


Gill, J. (2002). Reported levels of alcohol consumption and binge drinking within the UK undergraduate student population over the last 25 years. *Alcohol and Alcoholism, 37*, 109-120.


