THE ROLE OF AUTOMATIC COGNITION AND IMPULSIVITY IN HAZARDOUS DRINKING

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Paul Christiansen

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THE ROLE OF AUTOMATIC COGNITION AND IMPULSIVITY IN HAZARDOUS DRINKING

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Abstract

The current thesis aimed to explore the associations between hazardous drinking, automatic processing of alcohol-related cues and behavioural impulsivity. In addition to assessing the impact of these processes on hazardous drinking in isolation, the specific prediction of dual process models of addiction, that the association between automatic cognitive processes and hazardous drinking would be moderated by behavioural impulsivity, was also tested. These general research questions were investigated cross sectionally in young adult student populations (chapter 3), adolescents (chapter 4), and older adults (chapter 5). The aim of the study described in chapter 6 was to investigate the effect of a priming dose of alcohol on these processes and their association with alcohol-seeking behaviour. Finally, chapter 7 was an investigation into the mediators and moderators of the effects of ego depletion on drinking behaviour from the perspective of dual process models of addiction.

Automatic processing of alcohol-related cues predicted drinking behaviour in all cross sectional studies, although the specific aspects of automatic processes that predicted drinking behaviour differed. The effects of the alcohol prime and anticipated effects of alcohol on different measures of cognitive bias were inconsistent, and automatic cognitive processes only predicted ad-lib drinking in non-intoxicated individuals. Impulsivity only had a direct association with drinking within the sample of older adults, and neither the alcohol prime or ego depletion manipulation increased either measure of behavioural impulsivity. There was evidence that the alcohol priming effect was the result of general impairments in executive cognitive functioning. In the cross sectional studies support for dual process models of addiction was only found in the adolescent sample, in which

impulsive decision making moderated the association between attentional bias and problem drinking in adolescents. There was evidence that increases in automatic approach responses towards alcohol-related cues following an alcohol prime were the result of impairments in executive cognitive function. Although ego depletion resulted in increased alcohol consumption this was not due to increased behavioural control by automatic cognitive processes.

The results from the current thesis offer considerable support for incentive-motivational models of addiction, and highlight the importance of the roles of specific aspects of automatic cognitive processing in different samples. As behavioural impulsivity was only associated with hazardous drinking in older adults this suggests that cumulative experience with alcohol is necessary before these processes impact behaviour. Although support for dual process models of addiction was only found in adolescents, previous research would suggest that the predictions of these models are most likely to be found in such samples.

Declaration

No portion of this work has been submitted in support of any other application for degree or qualification at this or any other University or institute of learning.

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Chapter One

General Introduction

1.1 Alcohol use prevalence and costs

The World Health Organisation (WHO) estimates that approximately one billion people worldwide consume alcohol making it the second most commonly consumed psychoactive substance after caffeine. The Global status report on alcohol (WHO 2004) revealed that alcohol use has significant worldwide health impacts, e.g. alcohol is the eighth most prevalent cause of worldwide deaths and third in terms of causing increases in the percentage of disability adjusted life years (DALY's). In high income countries such as the UK, alcohol ranks second in DALY's, and is accountable for 6.7% of DALY's behind only tobacco (10.7%). Alcohol also contributes to numerous diseases and injuries, e.g. 50% of cirrhosis deaths, 30% of oesophageal and liver cancers and up 50% of traffic accidents worldwide. Significantly the worldwide impact of alcohol is more severe in males than in females (7.4% and 1.4% of DALY's respectively).

With regard to the UK, alcohol use has significant health, social, and economic impacts. The Adult Psychiatric Morbidity Survey (APMS 2007) reported that alcohol is the most commonly consumed drug in the UK with 73% of men and 57% of women consuming at least one alcoholic beverage each week. The British Household Panel Survey (BHPS 2006) reported that the average weekly alcohol consumption was 19.9 UK units for males and 9.2 UK units for females. With regard to specifically 'dangerous' levels of drinking the APMS (2007) found that 24% of the population aged 16 or over were classified as hazardous drinkers (as defined by the 8+ cut-off of the Alcohol Use Disorders Identification Test, AUDIT; Saunders et al., 1993). Again, males were found to be more hazardous drinkers than females (33% compared to 16%). This survey also assessed alcohol dependence using the

Severity of Alcohol Dependence Questionnaire - Community version (SADQ-C), reporting that 5.9% of the population was classed as alcohol dependent (8.7% males, 3.3% females); although in the vast majority of cases this dependence was classified as mild, rather than severe.

Alcohol use and dependence has a significant economic impact in the UK. Treatment services for alcohol dependence cost the government approximately £57.4 million per annum (£55.3 million on services, £2.1 million on prescribed drugs). In addition to these costs specifically associated with alcohol dependence, hospital admissions due to alcohol consumption are estimated to cost the NHS £168 million per annum and admissions partially attributable to alcohol use (e.g. due to falling while intoxicated) cost just over one billion pounds per annum. In addition to the direct cost to the NHS to treat alcohol-related illness/injury it is estimated that alcohol-related crime costs the government £7.3 billion per annum. Finally it is estimated that alcohol misuse has a significant effect on work place productivity in the public and private sector, costing approximately £6.4 billion per annum.

With regards to adolescents in the UK a similar pattern of drinking is observed as in adults. A report by the Institute of Alcohol Studies (IAS 2007) found that 84% of adolescents aged 12-17 reported that they had consumed alcohol at least once. The Information Centre for Health and Social Care (ICHSC 2007) estimated that in 2006 adolescents (aged from 11-15) consumed an average of 11.4 units per week, with males consuming more (12.3) than females (10.5), this represents a large increase from the average of 6.0 units consumed by adolescents in 1990 (7.0 and 4.3 units for males and females respectively; ICHSC 2007). Hospital admissions in adolescents due to alcohol use remain relatively low with 8894 in 2005/2006, although this does represent an increase from the 6667 admissions reported in 1997/1998. With regard to alcohol-related crime Matthews et al. (2006) found that although only 14% of adolescents drank regularly, those who did committed 37% of crimes in this age group. Significantly, delinquency problems in the UK resulting from adolescent alcohol use are three times that of the European average (Hibell et al., 2007).

These statistics highlight the prevalence of alcohol-related problems in the UK. It is important to note that it is not just alcohol dependence that has a significant socio-

economic impact, hazardous drinking behaviours and alcohol abuse also contribute to what is a significant problem. Indeed, if alcohol-related problems are viewed as developmental in nature, the understanding of hazardous drinking behaviour as a precursor to alcohol abuse and dependence may offer the best solutions to addressing the issue of alcohol consumption in the UK.

1.2. Substance dependence and substance abuse

The Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) defines substance dependence and substance abuse as separate disorders with different physical and behavioural symptoms (Table 1.1). There is therefore a clear distinction between abuse and dependence. Firstly, there is no physical dependence on drugs in what is defined as substance abuse. Drug abuse is damaging to health (as well as having a negative social impact) but is not necessarily out of control. Substance abuse still contributes to significant social and economic costs to both society and the individual; indeed, substance abuse can be seen as a precursor to substance addiction, which is essentially an escalation of the problems associated with abuse. Therefore the escalation from social drinking, to abuse, onto dependence can be seen as developmental in nature.

An associated concept is non-dependent hazardous drinking. This represents drinking behaviours that are increasing the risk of alcohol-related harm to the users (as well as others) even though these behaviours may not yet meet the DSM-IV criteria for abuse or dependence (Saunders et al., 1993). So again, like abuse is a precursor to dependence, hazardous drinking is a precursor of abuse. The current thesis aims to investigate the specific psychological factors that are associated with hazardous drinking. To investigate this, the AUDIT will be used to assess drinking patterns, this widely used self report measure gives a score from zero to forty with scores of eight and above indicating hazardous or harmful alcohol use. By using this measure an index of hazardous drinking behaviour can be ascertained.

Table 1.1: DSM-IV criteria for substance abuse and substance dependence

Substance Abuse	Substance Dependence
Substance use results in a failure to fulfil major obligations	Withdrawal
Use of substances when it is physically hazardous	Escalating use
Have substance-related legal problems	Desire and/or failure to control use
Continued social and interpersonal problems directly due to substance use	A large amount of time spent obtaining drugs
Tolerance	Reduction in time spent on other social and/or occupational activates in order to use and obtain drugs
	Knowledge that current physical, social and psychological problems are exacerbated by substance use.

1.3 Explaining drug use and drug dependence: Dual process models of drug use.

As well as having a significant socio-economic impact on society as a whole (as outlined above) alcohol consumption has an impact on individual health and well being. Indeed it is the continued use of drugs such as alcohol in the face of such negative consequences that lies at the heart of drug abuse and eventually addiction. As described earlier drug abuse involves prioritising drug use despite having explicit knowledge of its negative consequences. This indicates that in drug abuse there is a breakdown of the rational decision making processes, leaving behaviour at least partially governed by factors outside the control of the individual. However, it is important to note that substance abuse is characterised by an abnormally strong motivation to take psychoactive substances, even when the negative consequences become apparent. The strong motivational drive to consume drugs indicates that drug

abuse and dependence cannot be seen as simply a series of poor decisions made in isolation. The powerful motivational component is fundamental to the disorders, indicating that two interacting processes are central to the aetiology of drug abuse and addiction. Recent theoretical models of addiction have framed the disorder as a breakdown in the ability of controlled processes to regulate behaviour and increased control of behaviour by automatic motivational processes.

Specific dual process models of drug addiction (e.g. Goldstein & Volkow 2002; Jentsch & Taylor 1999; Wiers et al., 2007) make detailed predictions about how these two systems interact during the development and maintenance of drug dependence. For example, Goldstein and Volkow (2002), describes drug addiction as the result of 'impaired response inhibition and salience attribution (I-RISA)' (pp1643). They hypothesise that repeated drug administration results in abnormally high levels of salience being attached to drug-related rewards compared to other environmentally available rewards; this results in a powerful automatic drive to consume drugs. This automatic drive motivates an individual to continue to use a drug even in the presence of negative effects and a lack of a subjective 'high'. In addition to increasing the salience of drug-related stimuli this theory hypothesises that drug use also results in a breakdown of behavioural control (specifically response inhibition), with this greatly contributing to binges and relapse. Therefore, according to Goldstein and Volkow (2002), addiction can be conceived of as an imbalance between the attribution of salience to drug-related cues and the ability to inhibit appetitive or impulsive responding towards these cues, with a dominance of the attribution of salience over inhibitory processes. Jentsch and Taylor (1999) make similar predictions inasmuch that continued drug use results in drugs developing conditioned incentive-motivational properties which subsequently gives rise to a strong desire to consume them. They also argue that chronic drug use directly affects the frontal cortex, impairing inhibitory mechanisms that are used to regulate behaviour. Significantly, although these models are based upon neuroimaging data and animal studies (respectively) they both make the same predictions regarding the role of automatic conditioned processes and inhibitory control in the aetiology of drug abuse and addiction.

Similarly, Wiers et al. (2007) propose that after repeated alcohol use an individual will become sensitised to alcohol and alcohol-related stimuli, resulting in the automatic appraisal of alcohol-related stimuli as appetitive. The impact of the automatic processing of alcohol-related stimuli on drinking is hypothesised to be governed by controlled processes, which can be viewed as two separate constructs; an individual's motivation and ability to control behaviour. When drinking first starts it is unlikely that an individual will be motivated to attempt to control their drinking as they have not built up negative expectancies from drinking episodes or experienced physical or socio-economic problems as a result of alcohol use. Instead individuals will be motivated to drink due to expectation that alcohol will have a positive effect such as having a good time, fitting in with a social group, relieving tension or increasing sexual encounters (e.g. Goldman et al., 1997). At this point controlled processes will be driving alcohol consumption, and the conditioned effects of alcohol-related cues will be strengthened resulting in an increased automatic motivation to consume alcohol. If an individual eventually becomes motivated to change their behaviour then their ability to do so becomes important. At this stage they will have two competing motivational tendencies: automatic tendencies to drink (resulting from strengthened automatic appetitive tendencies) and motivation to limit drinking or abstain (resulting from controlled processes). Similar to the I-RISA theory, which motivational orientation drives drinking behaviour will be determined by the ability of controlled processes to inhibit the automatic drive to consume alcohol. Therefore, the relative strength of the two competing systems will then dictate whether alcohol is consumed or the individual successfully abstains.

This hypothesised imbalance between automatic and controlled cognitive processes is the result of gradual changes which slowly contribute towards increased behavioural control by automatic processes. Indeed, in severely addicted individuals we would expect the imbalance between these two systems to be much greater than in a very heavy social drinker who regularly abuses alcohol, who in turn, would show a greater imbalance than a heavy 'weekend' drinker. Therefore dual process models of addiction can be seen as developmental, with a gradually escalating imbalance between the two systems contributing to transitions from social drinking, to alcohol abuse and onto alcohol dependence. This indicates that individual

differences in the magnitude of the imbalance between these two systems should have a direct relationship with alcohol use, particularly hazardous drinking behaviour.

The following sections of this chapter will break down dual process models of addiction and describe their specific subcomponents, how they are assessed, and their relationship to individual differences in the use of alcohol and other drugs. First, theories describing the development of strong automatic motivations to take drugs will be discussed, focusing on why an abnormal amount of incentive salience is attached to drug-related cues as opposed to other naturally occurring rewards. Then, experimental evidence for the association between automatic processing of drug-related cues and indices of substance use will be described (focusing on alcohol use). Secondly, the different aspects of controlled processes will be described, with a specific focus on the related concept of behavioural impulsivity. Following which the evidence base for the relationship between impulsivity and drug use (again, specifically focusing on alcohol) will be assessed. Finally, experiments which have tested the specific predictions of dual process models of addiction, that individual differences in behavioural impulsivity will moderate the impact of automatic responses to alcohol-related cues on drinking behaviour, will be discussed.

1.4 Drug use and automatic cognitive processes

1.4.1 Neurobiological models of cue reactivity

As discussed earlier the processing of environmental cues can lead to the automatic activation of behaviour. Goldstein and Volkow (2002) hypothesised drugs and drug-related cues acquire abnormally high levels of salience compared to other cues in the immediate environment resulting in a strong automatic drive to consume drugs. The Incentive Sensitisation (IS) hypothesis (Robinson & Berridge 1993; 2001) gives a neurobiological explanation as to how drug-related stimuli come to effectively

dominate automatic cognitive processes. This theory hypothesises that strong automatic motivations to consume drugs in response to drug-related cues are due to the effects of drugs on the mesolimbic dopamine system, the central reward processing system of the brain. Obtaining any innately rewarding stimuli, e.g. food and sexual stimuli (Bassareo & Di Chiara 1997; Robinson et al., 2002; Robinson et al., 2001), results in the activation of mesolimbic dopamine system. This causes dopamine release which signals incentive-salience of the perceived stimuli (and associated environmental cues) thereby increasing attentional allocation and approach responses towards them (Franken 2003). The central tenet of the IS theory is that psychoactive drugs have long lasting effects on the organisation of the reward system of the brain. Specifically, psychoactive drugs (e.g. alcohol) stimulate the release of dopamine, with repeated use resulting in the reorganisation of the mesolimbic dopamine system leaving it hypersensitised to drugs and drug-related stimuli. Indeed, whereas the effects of naturally occurring rewards on mesolimbic dopamine functioning are rapidly habituated to, the effect of psychoactive drugs on dopamine release mean they continue to work as novel stimuli (Di Chiara & Bassareo 2007). This results in abnormally strong associations between the drug (and associated environmental stimuli) and reward being formed. This process means that drug-cue associations are more powerful than those between natural rewards and environmental cues. Psychoactive drugs therefore effectively 'hijack' the reward system of the brain with drug use continuing to strengthen drug-reward associations due to their ability to act as novel stimuli even after repeated exposure. It is important to note that the mesolimbic dopamine system does not dictate whether a given stimuli is 'liked', only that it is salient and therefore 'wanted', and so can result in a motivational drive to consume drugs regardless of explicit knowledge of the damage they will cause.

In addition it is notable that sensitisation of the mesolimbic dopamine system may not be substance specific. Studies investigating the phenomena of cross-sensitisation have revealed that the administration of one psychoactive substance results in a heightened sensitisation for a different drug and its associated cues. For example rats that have been administered cocaine show sensitisation to the condition rewarding effects of morphine (Kim et al., 2004). This indicates that using one drug (e.g.

cocaine) could result in a hypersensitised response to cues associated with other drugs (e.g. alcohol). Therefore the conditioning effects of a drug on the mesolimbic dopamine system may result in incentive motivational properties being placed upon a variety of drug-related stimuli.

1.4.2 Cognitive models of cue reactivity

In addition to neurobiological models of addiction, other models from mainstream cognitive psychology have also been used to explain the effects of drug-related cues on behaviour. Indeed, these groups of models converge to give a clear theoretical framework for the study of automatic cognitive processes in addiction. A number of memory network models, such as spreading activation models (e.g. Anderson & Pirolli 1984; Collins & Loftus 1975; Freedman & Loftus 1971) and multiple trace theory (Hintzman 1986), have been adopted from mainstream cognitive psychology to explain the role of implicit cognition in addictive behaviours. Generally, these models describe automatic cue reactivity in the context of the automatic activation of drug-related memories in the long term associative store without the influence of short term memory or attentional systems. Instead, memories are automatically activated by preceding memory activation or the current affective/environmental situation. The ability of stimuli to have an excitatory effect on drug-related memories depends upon their mutual compatibility- which is dictated by the frequency and salience of previous pairings. Therefore, non drug-related stimuli can develop the ability to activate a drug-related memory it has been frequently paired with. This activation of drug-related memories can, in turn, initiate goal directed motor responses outside conscious awareness, thereby driving drug use.

Cognitive-motivational models of drug use such as Baker et al. (1987) and Cox and Klinger (1988) propose a fundamental role for the automatic processing of alcohol-related cues but also hypothesise that current motivation will have a role in their behavioural expression. In circumstances when an individual is motivated to drink due to a current incentive, based upon expectancies of a specific beneficial outcome,

then they will more readily process alcohol-related stimuli. In addition, Baker et al. (1987) argue that drug-related cues activate motivational systems, so in an ongoing drug user who has no explicit desire to quit use, the presence of drug-related cues can activate a positive motivational desire to consume drugs. Tiffany (1990) described an automatic cue reactivity theory which states that after repeated drug use in the presence of drug-related cues (e.g. a certain bar), the drug-related cues start to elicit drug-seeking behaviour automatically, even in the absence of an explicit motivation to use the drug. In other words, while drug-related cues might initially activate drug-related memories, which motivate drug-seeking behaviour (stimulus-outcome-response, S-O-R learning), over time those stimuli are able to elicit drug-seeking behaviour automatically, in the absence of retrieval of memories of drug effects (stimulus response, S-R learning).

Although incentive-sensitization theory, cognitive-motivational models and cognitive models of addiction such as Tiffany (1990) are similar, there is an important distinction. Cognitive-motivational (and neurobiological) models are based upon pre-conscious processes, i.e. those that influence behaviour automatically. These processes influence behaviour by activating a motivational state, which in turn, drives behaviour. Cognitive models of addiction (such as Tiffany 1990) are based upon post-conscious processes which are behaviours that have become automatic, such as complex motor behaviours that have become motor programs after repeated performance (e.g. driving a car, tying shoelaces, rolling a cigarette), but are originally volitionally initiated. In these models the motivational component is not required for cues to elicit behaviour automatically, i.e. behaviour is initiated without any strong motivation.

Despite the aforementioned theories of addiction offering a different explanation as to how cue reactivity specifically drives drug use, similar predictions about the contribution of automatic cognition to drug use can be made from both. (1) On a basic level drug using individuals respond to drug-related cues by showing attentional bias towards them as well as activating automatic approach associations; (2) automatic cognitive processes should explain variance in drug use beyond that explained by explicit cognition; (3) manipulating implicit cognition will affect drug consumption.

1.5. Evidence base for automatic cognitive processes in human drug use

1.5.1. Measuring automatic cognitive processes in humans

Tests of automatic cognitive processes have been defined as measures that do not involve effortful introspective cognition. This is not to say that they measure unconscious processes which an individual has no awareness of. The tying of a shoelace, for example, is an automatic process involving the conscious initiation of behaviour in order to achieve a conscious goal (the act is not unconsciously initiated), but the actual process of tying the shoelace occurs unconsciously and can be completed with no introspection or conscious effort. Tests of automatic cognition therefore measure an attribute which effects behaviour; the individual may be aware of both the attribute and the behaviour, what is critical is the specific processes through which the attribute effects behaviour must not be known to the individual (Bargh & Morsella 2008; De Houwer et al., 2009). So in relation to alcohol use, a heavy drinking individual may be aware that alcohol-related pictures in advertisements capture their attention, and will certainly be aware that they drink, but they might be unaware of the process that links these two things i.e. the specific process is automatic.

The automatic processing of drug-related cues has been measured in a variety of ways. Firstly, there are tasks that assess selective attention, the degree to which drug related cues grab and/or hold attention, these are usually referred to as attentional bias tasks. Attentional bias towards pictorial cues has been assessed using visual probe, attentional cueing, emotional Stroop, and flicker-induced change blindness (flicker ICB) tasks, while attentional bias towards words has been measured with the attentional blink task and the emotional Stroop. The second broad category is association tests. In word association tasks participants are given a word or sentence (usually ambiguous e.g. draft, high) and have to state the first word that comes to mind after reading it, with more drug-related responses to ambiguous words/sentences being indicative of stronger memory associations. Recently,

reaction time based association tasks have been used to investigate automatic cognitive processes in addiction. The most common of these tasks is the implicit association test (IAT; Greenwald et al., 1998), a reaction time based measure in which participants rapidly categorise drug-related and control words alongside words belonging to two different categories e.g. positive and negative, approach and avoid. Other implicit categorisation tasks involve making symbolic approach responses towards pictorial stimuli e.g. stimulus response compatibility task (SRC), and actual approach and avoid responses with the approach and avoid task (AAT). It is beyond the scope of the current thesis to review in detail all of these tasks and their association with drug use (see Field & Cox 2008; Stacy & Wiers 2010 for detailed reviews). The subsequent sections will summarise the most pertinent findings focusing on attentional bias and automatic approach responses towards drug-related stimuli (with alcohol-related research being described in the most detail).

1.5.2 Attentional bias

1.5.2.1. Addiction Stroop

There is a large evidence base demonstrating the automatic processing of drug-related words in drug users compared to controls. In the emotional Stroop task participants are presented with drug-related words (e.g. Beer, Whisky, Lager) and control words. Control words are usually matched on length, syllables, frequency as well as being from a single semantic category (e.g. music-related words; Field et al., 2007a). Words are presented in different colours (often red, yellow, green and blue) and participants are instructed to read out the colour which the words are presented in as fast as possible. Theoretically, salient words are hypothesised to grab the attention of the participants, increasing the latency of the colour-naming response and increasing the likelihood of errors. There is a considerable amount of evidence suggesting that the emotional Stroop is efficacious in discriminating drug users from controls (Copersino et al., 2004; Franken et al., 2000b; Munafo et al., 2003; Field

2005) as well as heavy drinkers from light drinkers (Field et al., 2007a; Cox et al., 2000; Sharma et al., 2001). Furthermore, Stroop interference has also been shown to predict treatment outcomes in smokers, alcoholics, heroin and cocaine addicts (Waters et al., 2003b; Carpenter et al., 2006; Marissen et al., 2006; Cox et al., 2002).

Despite this evidence base there has been a significant amount of criticism of the Stroop task as a measure of attentional bias. A recent meta-analysis (Cox et al., 2006) found that the blocked format (words from a single semantic category presented together) gave larger effect sizes than when words were presented in a randomised format. A series of studies found that this was due to slowed colour naming of addiction-related words being carried over to subsequent neutral words, suggesting the greater effect sizes seen in blocked format are the result of carry over effects inflating attentional bias (Waters et al., 2005; Waters et al., 2003a; Sharma & Money 2010). Recent studies have also demonstrated that the Stroop is not a pure measure of attentional bias as it is confounded by other cognitive processes. For example, Algom et al. (2004) reported that the (emotional) Stroop effect is the result of a general cognitive slowing in reaction to threat-related words rather than a specific attentional bias towards them. In addition to measuring attentional bias the Stroop task also measures inhibitory control, the ability to inhibit a pre-potent response (reading a word) in order to produce an atypical response (naming of the colour). Although the one study to date that has controlled for inhibitory control deficits still found attentional bias in an alcohol Stroop (Fadardi and Cox 2006), it is worth considering this as a possible confound especially in samples where one might expect a degree of impairment in executive cognitive functioning. Due to these methodological concerns with the addiction Stroop, recent research has started to focus on tasks that measure the allocation of visuo-spatial attention. These tasks offer a cleaner measure of attentional bias as well as allowing inferences about different aspects of the attentional system to be made.

1.5.2.2. Attentional cueing and visual probe tasks

An index of the salience of drug-related cues has been derived from tasks that directly or indirectly measure the allocation of attention towards pictorial stimuli. The attentional cueing task (ACT; Posner et al., 1982) has been used to measure the ability of pictorial cues to grab and hold attention. This task involves the presentation of two blank squares on a computer screen following which a picture from a salient category, (e.g. a bottle of beer) or a neutral category, (e.g. a bottle of water) is presented in one of the boxes for a short duration. A probe (a small black dot) then appears in either the same box as where the picture appeared (congruent trial), or in the opposite box (incongruent trial). Participants respond to the position of the probe as rapidly as possible. Theoretically, salient stimuli should result in attention being allocated towards the box where it appeared, reducing reaction times to congruent probes (as attention is already allocated to that box) and increasing reaction times to incongruent probes (as attention is allocated to the other box). In addition, the attentional cueing task has been used in an attempt to discriminate different aspects of attention (initial orienting from maintenance of attention) by presenting pictorial cues for different stimulus onset asynchronies (SOA's). Theoretically, short presentations of cues (SOA's $\leq$ 100 ms) assess initial orientation of attention, and longer presentation of cues (SOA's $\geq$ 250 ms) assesses the holding of attention (Cisler & Koster 2010; Posner & Petersen 1990). Although a significant amount of research using the ACT has been conducted in the anxiety literature (e.g. Fox et al., 2001; Stormark et al., 1995) little has been conducted in assessing cue reactivity in addictive behaviours. Franken et al. (2000a) used the ACT to investigate attentional bias in cocaine dependent inpatients, and although there was no overall differences in reaction times to cocaine or neutral cues at either the long (500 ms) or short SOA's (100 ms), inpatients with higher levels of drug craving did show difficultly disengaging from cue locations in the short cue presentation condition. Likewise, Stormark et al. (1997) used the ACT to assess the allocation of attention in a sample of abstinent alcoholics. Alcohol-related cues presented for the short duration produced initial orientation of attention towards them whereas there was

disengagement of attention from cues presented for the longer duration (i.e. slower reaction times to congruent probes, faster reaction times to incongruent probes).

Recently, a significant number of studies have investigated attentional bias towards drug-related cues using the visual probe task. This task involves the simultaneous presentation of two visual cues (usually pictures) on a computer screen; one of the cues depicts the proposed salient cue (e.g. an alcohol-related picture) and the other a neutral cue that has been matched upon perceptual properties. Both the cues then disappear at the same time followed by a probe (usually an arrow pointing up or down) appearing in one of the picture locations. Faster reaction times to congruent probes (appearing in the same location as the salient stimuli) compared to incongruent probes (appearing in the same location as the neutral stimuli) are indicative of greater attentional bias towards the critical stimuli. In addition to using reaction times as an index of attentional bias towards drug-related stimuli, some studies have also used concurrent eye tracking to assess duration of gaze dwell times on pictorial cues as an additional measure of attentional bias. The visual probe task has consistently demonstrated significantly slower response latencies to incongruent probes compared to congruent probes in smokers (Hogarth et al., 2003; Chanon et al., 2010; Mogg et al., 2003; Mogg et al., 2005; Vollstädt-Klein et al., 2011), heroin addicts (Lubman et al., 2000), ketamine users (Morgan et al., 2008) and cannabis users (Field et al., 2006) compared to non-user controls. Studies that have utilised concurrent eye tracking have demonstrated similar effects for smokers (Mogg et al., 2003) and cannabis users (Field et al., 2006). Heavy drinkers and alcoholics have also been shown to exhibit greater attentional bias towards alcohol-related stimuli than light drinkers in visual probe tasks using the reaction time index (Field et al., 2004b; Miller & Fillmore 2010; Townshend & Duka 2001; Noel et al., 2006; Vollstädt-Klein et al., 2009) and gaze dwell times (Miller & Fillmore 2010).

Researchers have also varied SOA's in the visual probe task to try and look at different aspects of attention (i.e. initial orientation vs. maintenance). Field et al., (2004b) investigated attentional bias in heavy and light social drinkers using a visual probe task that presented cues for 200 ms, 500 ms and 2000 ms. Interestingly, no differences were found between heavy and light drinkers at 200 ms but heavy drinkers showed significantly greater attentional bias at both 500 and 2000 ms

SOA's, with the greatest difference being in the latter. This has been replicated in non-dependent drinkers (Miller & Fillmore 2010; Townshend & Duka 2001). However, in alcohol dependent patients a different pattern of attentional bias is exhibited with dependent indivduals showing attentional bias when cues are presented for short (< 200 ms) SOA's, but avoidance of cues presented for longer SOA's (> 500 ms; Noel et al., 2006; Vollstädt-Klein et al., 2009; Townshend & Duka 2007). These findings (and those from the ACT) raise the possibility alcoholics and heavy social drinkers demonstrate a different time course in their allocation of attention to alcohol-related stimuli.

To test whether there is a causal relationship between attentional bias and drug use, recent studies have attempted to manipulate attentional bias and investigated subsequent changes in craving and drug-seeking behaviour. For example, Field and Eastwood (2005) trained heavy social drinkers to attend to alcohol-related cues in a modified visual probe task. Attentional retraining led to an increase in craving and alcohol consumption in a bogus taste test. However, there have been failures to replicate the effects of attentional retraining on beer consumption in the laboratory (Field et al., 2007b; Schoenmakers et al., 2007). Similarly, studies that have manipulated attentional bias in smokers (Attwood et al., 2008; Field et al., 2009; McHugh et al., 2010) have not consistently demonstrated a causal relationship between attentional bias and craving or smoking behaviour. In addition, Schoenmakers et al. (2010) investigated the efficacy of attentional bias modification as a treatment in a sample of abstinent alcoholics. Alcoholics were trained not to attend alcohol-related pictures in a visual probe task (a control group completed an irrelevant reaction time task). Those who were trained to direct their attention away from alcohol-related cues took longer to relapse than a control group, although groups did not differ in overall relapse rates at three month follow-up. These mixed results suggest that more research is required before a causal relationship between attentional bias and craving and substance use can be confirmed.

In summary, cross sectional studies assessing attentional bias have consistently demonstrated that alcohol-related cues will hold the attention of heavy drinkers compared to light drinkers. What specific aspects of attention are associated with drinking in different populations is less clear. There is some evidence that heavy

drinkers do not show initial orientation of attention towards alcohol-related cues but do maintain their gaze on alcohol-related cues; while alcoholics show the opposite pattern of results. This indicates that hazardous drinkers/alcohol abusers show a different time course of attentional biases than alcoholics. The evidence for a causal relationship between attentional bias and alcohol consumption is not clear, with relatively few studies manipulating attentional bias and investigating subsequent changes in alcohol consumption.

1.5.3. Automatic approach tendencies and implicit associations

Automatic responses to drug-related cues have been assessed in a number of different ways other than attentional bias. Much of the research into automatic cognitive processes in addiction has been dominated by the implicit association test (IAT). This task is used to measure the strength of implicit associations between any two categories of stimuli, for example 'alcohol' and 'positive'. During the IAT participants categorise four different sets of words or pictures (e.g. alcohol, soft drinks, positive and negative) using one of two computer keys to respond. In one block of the task alcohol/positive share one response key and soft drink/negative share the other response key, and in another block this is reversed (alcohol/negative and soft drink/positive share response keys). The assumption of the IAT is that if automatic associations between two of the categories are stronger (e.g. alcohol-positive) than the reverse association (e.g. alcohol-negative) then response times will be faster when compatible categories share a key. The IAT has repeatedly revealed that substance-related concepts are automatically associated with concepts such as positive valence and arousal (as reviewed by Roefs et al., 2011; Stacy & Wiers 2010).

Neurobiological theories of addiction suggest that repeated drug use should also increase the associations between drug-related cues and automatic approach responses. Animal studies have reliably shown that cues paired with drug-related stimuli will initiate approach responses in rats (e.g. Krank et al., 2008). Recent

human research has also demonstrated stronger drug-approach associations in drug users compared to controls. For example, individual differences on the approach-avoid IAT are associated with drug use; individuals who have relatively strong alcohol-approach associations (compared to alcohol-avoidance associations) tend to drink more than those with relatively strong drinking-avoidance and weak drink-approach associations, with similar findings being found in smokers (e.g. De Houwer et al., 2006; Lindgren et al., 2009; Palfai & Ostafin 2003). In addition to the IAT other conceptually similar tasks have been developed that investigate the speed at which drug users make symbolic approach responses towards alcohol-related and neutral pictorial stimuli e.g. stimulus response compatability task (SRC; De Houwer et al., 2001). In the SRC task, drug-related or drug-unrelated (control) pictures are presented on a computer screen with a manikin which is either above or below the picture. In one phase of the task (the 'approach drug' block) participants are required to rapidly move the manikin towards drug-related pictures and away from control pictures (by pressing up or down on a response box); in another phase of the task (the 'avoid drug' block) participants are required to move the manikin towards the control pictures and away from the drug-related pictures. Correct responses are met with the image of the manikin walking towards or away from the picture (depending on picture type and block). If participants are faster on the approach-alcohol block compared to the avoid-alcohol block this suggests that automatic associations between drug-related cues and the approach response facilitate the encoding of the stimuli, whereas those in the avoid-alcohol block are not consistent with automatic associations, slowing responses. The SRC task has demonstrated that heavy, but not light, social drinkers are quicker to direct symbolic approach responses towards alcohol-related cues than they are to avoid them (Field et al., 2008; Field et al., 2011). Furthermore, this task has also been shown to discriminate cannabis users (Field et al., 2006) and smokers (Bradley et al., 2004; Bradley et al., 2008) from controls. Other studies (e.g. Field et al., 2011) have utilised an irrelevant feature version of the SRC. In these tasks participants do not characterise alcohol and control pictures based upon their content but on an irrelevant feature such as orientation (e.g. approach landscape, avoid portrait). Field et al., (2011) found that although there was evidence for approach bias towards alcohol pictures in the relevant feature version of the SRC task there, was no evidence of faster approach

responses to alcohol-related pictures in the irrelevant feature version of the task. This suggests that stimuli must be appraised as being alcohol-related for them to initiate a (symbolic) automatic approach response.

An alternative version of the irrelevant feature SRC task utilises physical approach (arm flexion) and avoid (arm extension) responses. This task (approach avoid task; AAT) has been shown to successfully discriminate heavy drinkers from light drinkers (Wiers et al., 2009), particularly in individuals with the OPRM1-G allele (which is associated with the rewarding effects of alcohol). However, van Hemel-Ruiter et al. (2011) did not find that approach responses towards alcohol-related cues were facilitated in heavy drinking adolescents, who were actually faster to avoid alcohol-related pictures than approach them.

In an attempt to investigate the causal relationship between automatic approach responses and alcohol consumption Wiers et al. (2010) used an AAT task to train hazardous drinking participants to approach or avoid alcohol-related cues. Those who were trained to approach alcohol-related cues consumed more beer in a laboratory taste test than those trained to avoid them. In addition, the effects of the training generalised to a new set of pictures in a subsequent AAT, as well as words in an IAT (i.e. the training was not cue specific). As with attentional retraining, the retraining of automatic approach tendencies has also been investigated as a possible treatment for alcohol dependent individuals. These manipulations have also shown a degree of efficacy in altering the drinking behaviour in a clinical sample. Wiers et al. (2011) retrained alcoholics to avoid alcohol in an AAT task and found improved outcomes at a one year follow up, after controlling for demographics, problem duration and markers of stress and depression. These findings suggest that, rather than being epiphenomena of drug use, there is a causal relationship between automatic alcohol-approach associations and alcohol consumption.

1.5.4. The acute effect of alcohol on attentional bias and automatic approach behaviours

As reviewed above measures of automatic cognition have been consistently shown to predict individual differences in drinking behaviour, (see Roefs et al., 2011; Rooke et al., 2008 for meta analyses). However it is also important to consider the context in which these measures are taken. It is likely that there are contexts which exaggerate automatic responses to alcohol-related cues and increase their association with drinking behaviour. Recently, Waters et al. (2011) used ecological momentary assessment (EMA) to continually assess attentional bias in recently abstinent heroin and cocaine addicts and found that increased attentional bias was associated with 'temptation episodes'. This suggests that attentional bias fluctuates and may be elevated in certain contexts which, in turn, can increase motivation to consume drugs (i.e. behavioural control by automatic processes can be greater in some situations than others). Clinically, this suggests that the identification of risk situations is important and that targeting the modification of attentional bias as a treatment (e.g. Schoenmakers et al., 2010) may not be sufficient to promote abstinence if attentional bias can be significantly increased by contextual factors. Indeed, the identification of high-risk situations may have as much clinical significance as directly manipulating attentional bias. In non-dependent samples this suggests that in certain contexts the automatic processing of alcohol-related cues can be elevated and have a greater impact on subsequent alcohol seeking behaviour.

It has been hypothesised that priming doses of alcohol will strengthen automatic alcohol associations and therefore increase the automatic drive to consume alcohol (Field et al., 2010). Intoxication may therefore represent one high-risk situation in which automatic cognitive processes exert more control over behaviour. In such circumstances an individual may have an intention to limit drinking to one or two drinks but fail to do so due to the priming effect on automatic processes. However, studies investigating the effects of priming doses of alcohol on both attentional and approach bias have shown mixed results. Schoenmakers et al. (2008) investigated the effect of an alcohol prime on both attentional bias (visual probe), and automatic

approach responses (SRC task). They reported that a 0.3 g/kg dose of alcohol increased attentional bias compared to placebo, but had no effect on SRC performance compared to a placebo, notably this is the only study to date that has investigated the acute effects of alcohol on SRC task performance. Similarly a 0.4 g/kg priming dose has also been shown to increase attentional bias (Adams et al., 2011). In their investigation into the effect of different priming doses on attentional bias Duka and Townshend (2004) also demonstrated that a 0.3 g/kg dose of alcohol led to an increased in attentional bias for alcohol-related pictures, compared to placebo, although at a higher dose (0.6 g/kg) attentional bias did not significantly differ from that in the placebo condition. Other studies such as Miller and Fillmore (2011) found no effect (compared to a placebo) of either a 0.64 g/kg or 0.32 g/kg dose of alcohol on attentional bias. In regard to automatic approach responses one other study (Farris & Ostafin 2008) has investigated the impact of alcohol consumption on this construct and did find some evidence that alcohol administration strengthened automatic alcohol-approach associations (assessed with an IAT). It is important to note that in this study participants consumed as much alcohol as they wanted before testing and baseline performance was assessed pre-drink and not following placebo administration, so any conclusions made about the pharmacological effects of alcohol on alcohol–approach associations from this study are speculative. Significantly, none of the above studies investigated whether alcohol induced increases in the automatic processing of alcohol-related cues mediated the increase in alcohol consumption that is commonly seen following an alcohol prime (the alcohol priming effect). In addition to the effects of priming doses of alcohol there are demonstrations that other contextual factors such as exposure to alcohol-related cues (Lindgren et al., 2009; Schulze & Jones 1999), induction of positive mood (Birch et al., 2004) and stress (Field & Powell 2007; Field & Quigley 2009) can influence automatic alcohol-related cognitions.

1.5.5. Summary: Automatic cognitive processes in addiction

A significant literature exists demonstrating that drug-related cues grab the attention and elicit automatic approach responses in drug users. Significantly, these measures not only discriminate drug users from controls, but also discriminate individuals based on frequency and quantity of drug use (e.g. heavy vs. light drinkers). Prospective studies have also shown that measures in automatic cognition predict variance in alcohol use after controlling for explicit outcome expectancies (Houben & Wiers 2007; 2008). These findings along with manipulation studies and retraining in clinical samples indicate that automatic cognitive processes play a central role in the aetiology of drug use. Indeed, the automatic cognitive processes discussed in the previous sections may be one of the fundamental driving forces behind the increased automaticity of drug taking behaviour that is hypothesised to dominate behavioural responses according to dual process models of addiction.

Although this evidence base is significant it is worth noting an important caveat. A recent meta-analysis (Reich et al., 2010) investigated the contributions of automatic alcohol associations (measured with variations of the IAT), as well as questionnaire based measures of explicit alcohol-outcome expectancies, to indices of alcohol use. This analysis found that although measures of automatic cognitive processing of alcohol-related cues explain some unique variance in alcohol use, there was a large degree of overlap in variance explained by both explicit and automatic measures. In addition, the explicit measures predicted more variance in alcohol use indices than the automatic measures (weighted β of .29 and .23 respectively).

1.6 Impulsivity and executive cognitive functioning

As well as proposing an association between automatic processing of drug-related cues and drug consumption, dual process models of addiction also propose a fundamental role for controlled processes in the aetiology of drug use. These

processes are hypothesised to exert top-down control over behaviour through slow, deliberated processing. Decisions made by the controlled system are based upon the perception of environmental stimuli, inputs from the associative system and long and short term behavioural goals. This system therefore makes reasoned judgements through syllogistic reasoning allowing the formation of complicated strategic plans to pursue goals.

Although the different drives in the associative system can be conceptualised in a similar way (automatic behaviours as the result of conditioning) there is a much greater heterogeneity in what can be seen as controlled processes. Firstly, controlled processes can be viewed as a logical decision made according to the expected outcome of behaviour. So in the current context the expectation that alcohol consumption will have beneficial effects would inform a controlled decision to drink. Wiers et al. (2007) argue that early on in an individual's drinking career it is these expectancy-based decision making processes that will dominate drinking behaviour. Alcohol-outcome expectancies have been shown to predict a significant amount of variance in drinking, especially in younger drinkers (aged < 35; Leigh & Stacy 2004). However, controlled processes can also be conceptualised as impulsive or non-impulsive behaviours. Indeed both Goldstein and Volkow (2002) and Jentsch and Taylor (1999) argue that it is impulsive behaviours that are central to a lack of control over alcohol use and Wiers et al., (2007) cites impulsivity as an aspect of executive cognitive functioning which is implicated in the aetiology of hazardous drinking and addiction. Impulsivity is generally defined as 'maladaptive or inappropriate behaviours' (de Wit 2009 p 23), which also describes many of the symptoms that are associated with substance abuse (see Table 1) as well as hazardous drinking. Therefore, from the perspective of dual process models, impulsive behaviours can be seen as a failure of the controlled system to successfully exert control over behaviour and make a decision that will benefit the individual long term and instead allowing behaviour to be dictated by the associative system. It is important to note however that conceptually 'impulsivity' shares a great deal of overlap with the concept of executive cognitive (dys)function. Executive cognitive function refers to a set of inter-connected cognitive abilities which subserve the ability to inhibit pre-potent responses (which is also specifically described as one of

the components of impulsivity), updating information working memory, and switch between different mental sets (see Miyake et al., 2000). Although much of the recent research into executive cognitive functioning has been based on this argument that executive cognitive functioning is made up of three core components it is notable that Miyake and colleagues (e.g. Miyake & Friedman 2012; Friedman et al., 2006; Friedman et al., 2008) suggest that updating information in working memory may be the core executive cognitive function. Indeed these more recent models suggest that inhibitory control contributes no unique variance to executive cognitive functioning beyond that accounted for by shifting and updating working memory. It is possible that the aspect of executive cognitive function hypothesised to be the key controlled behaviour in dual process models of addiction such as Goldstein and Volkow (2002) and Jentsch and Taylor (1999) is actually a construct that shares all its variance with updating working memory. Despite these current ambiguities in the executive cognitive functioning literature, it can be expected that if executive cognitive functioning is impaired then the ability to regulate and change behaviour will be impaired leading to 'impulsive' behaviours, and all these abilities can be seen as being part of the controlled system, and are all vital to making conscious behavioural decisions.

Although a significant amount of research has investigated the role of impulsivity in drug use there is no single operational definition for impulsivity. For example, questionnaire measures of impulsivity assume it to be a relatively stable personality trait that can be accessed through introspection (Eysenck et al., 1985; Patton et al., 1995; Whiteside et al., 2005). These questionnaires are often broken down into further subscales of impulsivity, for example the Barratt Impulsivity Scales (BIS-11; Patton et al., 1995) describes Attentional, Motor and Non-Planning impulsivity, which although inter-correlated, represent distinct forms of self-report impulsivity. These measures have been shown to consistently predict indices of alcohol consumption (e.g. Fernie et al., 2010; Gunnarsson et al., 2008; McAdams & Donnellan 2009; Von Diemen et al., 2008; Von Knorring et al., 1987).

Impulsivity can also be inferred from performance on behavioural tasks. Both de Wit and Richards (2004) and Olmstead (2006) argue for two distinct components of 'behavioural' impulsivity, which can be directly measured with behavioural tasks

rather than relying on self-report questionnaires. The first of these components is impulsive decision making- the degree to which individuals discount the value of future rewards as the delay to the receipt of the reward increases. So, for example, an impulsive decision would be preference for a small immediate reward (e.g. drinking tonight, taking cocaine), over a long term large reward, (e.g. exam success, improved health). Such decisions can be seen as a breakdown of the controlled decision making process as the immediate reward (often dictated by automatic behavioural motivation) is less beneficial to the individual than the delayed reward but the deliberative processes has still resulted in drug taking as the preferred behavioural option. It has been suggested that delay discounting is strongly associated with internal clock speed (ICS; Wittman & Paulus 2008) inasmuch as individuals who perceive time to pass more slowly assign a higher cost to delays and are therefore more likely to accept a small immediate reward than wait for a larger delayed reward. Recent research has found support for this assertion with participants with slower internal clock speeds showing steeper discounting of future rewards (Corvi et al., 2012). The second, independent, measure of behavioural impulsivity is inhibitory control. This refers to the ability of an individual to suppress and control pre-potent responses and therefore ties in very closely with dual process models of addiction (i.e. Goldstein & Volkow 2002; Jentsch & Taylor 1999). So for example, if an individual has a strong conditioned approach response towards alcohol then they will need to use inhibitory control to suppress this response, if their inhibitory control is not strong enough then their behaviour will be driven by their conditioned reaction towards alcohol cues resulting in alcohol consumption.

There has been considerable debate into the independence of self-report measures and behavioural measures of impulsivity. Studies investigating this have generally used principal component analysis on multiple measures of inhibitory control and impulsive decision making. For example, Reynolds et al. (2006a) tested the independence of the Stop-Signal, Go/No-Go, delay discounting and the balloon analogue risk task (BART; Lejuez et al., 2003). This analysis revealed that the BART and the delay discounting task loaded on to a separate factor (impulsive decision making) from the Stop-Signal and Go/No-Go tasks (impulsive disinhibition). Swann et al. (2002) also assessed the independence of components of

impulsivity using a different task to assess inhibitory control (Continuous Performance task) as well as two real time discounting tasks, (Two Choice Delay task and Single Key Impulsivity Paradigm). Again, despite different tasks being used, two distinct components of behavioural impulsivity where identified ('rapid response' and 'reward delay' impulsivity). Despite the relatively strong evidence for the independence of impulsive decision making and inhibitory control the relationship between trait measures and behavioural measures is unclear. White et al. (1994) conducted a factor analysis on multiple measures of behavioural and self report impulsivity and found them to represent independent constructs. Reynolds et al.(2006a) also assessed multiple measures of self report impulsivity, and although they did not add them to their principal component analysis, they did find that there were no correlations between self-report impulsivity and behavioural impulsivity. However, both Swann et al. (2002) and de Wit et al. (2007) found correlations between behavioural impulsivity and self report impulsivity. Taken together these results suggest that the relationship between self report and behavioural measures of impulsivity is unclear.

As previously stated impulsivity has much in common with executive cognitive functioning. In their seminal paper, Miyake et al. (2000), found that inhibitory control (as assessed by the Stop-Signal, Antisaccade and Colour-Conflict Stroop tasks) was one of the three core components of executive function (with the other two components being working memory and switching). Similar findings regarding the breakdown of executive function were also found by Verdejo-Garcia and Perez-Garcia (2007). It is possible that a significant amount of variance is shared among behavioural impulsivity and measures of other aspects of executive cognitive functioning. There are measures such as phonemic fluency tasks which assess multiple aspects of executive cognitive functioning (inhibitory control, working memory and switching; Abwender et al., 2001; Troyer et al., 1997). This task may therefore be a useful tool if we wish to investigate whether the inability of controlled processes to regulate behaviour is specifically the result of increases in behavioural impulsivity, or a general impairment in executive cognitive functioning.

1.7. Evidence base for impulsivity in drug use

1.7.1. Measuring impulsive decision making

Impulsive decision-making is commonly measured using the delay discounting procedure (e.g. Madden et al., 1997). This task assesses an individual's preference for large rewards that are available after a delay or smaller rewards that are available immediately. The key assumption of the delay discounting procedure is that as the temporal distance to a reward increases its subjective value will decrease; the steeper the perceived decrease in the value of the delayed reward the more impulsive an individual is. Therefore, an impulsive individual would prefer the small immediate reward over a larger delayed reward.

In a delay discounting task participants are given a choice between two different amounts of money e.g. "would you rather have £150 now or £250 in two weeks?" participants then state which of the two rewards they would prefer. The delayed reward remains fixed while the immediate reward and the delay period adjust. Participants simply state their preference between the two rewards until they select the delayed reward (this is the indifference point- when both delayed and immediate rewards have similar values to the participant). Participants complete the process for several different delay periods (e.g. one week, one month, six months, and one year). Measures of delay discounting differ in format of presentation, with multi-choice tasks utilising blocked questionnaire formats and computerised measures adjusting according to responses made (e.g. Du et al., 2002). In addition, to multi-choice measures there is the monetary choice questionnaire (MCQ; Kirby et al., 1999) and single question measures of discounting (e.g. Reimers et al., 2009). A recent meta-analysis (MacKillop et al., 2011) assessed the efficacy of these different delay discounting methodologies and found that the MCQ and multi-choice delay discounting tasks showed similar effect sizes, with those for single items being smaller. It is also worth noting that although studies that utilise delay discounting

procedures often use hypothetical monetary rewards and delays, discounting rates for hypothetical rewards are similar to those obtained using real rewards (Madden et al., 2003; Madden et al., 2004).

A diverse set of methodologies have also been used to calculate discounting rates. The three main methods by which discounting rates have been calculated are based on different theoretical assumptions (or no theoretical assumptions). Firstly, exponential discounting models makes the assumptions that immediate rewards are guaranteed but there is a risk involved in receiving delayed rewards, and that this gives rise to a simple linear relationship between delay and preference. The hyperbolic discounting model (Kirby 1997) characterises delay discounting as a choice between a delayed and immediate reward with increases in the delay decreasing the ratio of reward to delay thereby decreasing its value, this model therefore assumes that reward value decreases rapidly with short increases in delay, but then levels off (i.e. a hyperbolic relationship). These two methodologies for assessing discounting try to fit data to these curves. Finally, the area under the curve methodology (AUC; Myerson et al., 2001) is a theoretically neutral measure that calculates a discounting rate by calculating the area under the discounting curve that is produced as the value of delayed rewards decreases as a function of time. Therefore no attempt is made to fit data to theoretical models of discounting. The diversity in the methodologies employed to study delay discounting may be one of the factors that contributes to the inconsistencies which are apparent in the alcohol and drug use literature.

1.7.1.1. Evidence for delay discounting in drug and alcohol use

There is some evidence to suggest that drug users discount the value of future rewards at much steeper rates than non users. For example, heroin addicts show steeper rates of discounting than non-using controls (Kirby et al., 1999; Madden et al., 1997) as do current cocaine addicts (Coffey et al., 2003). Kirby and Petry (2004) analysed differences in discounting between cocaine addicts, heroin addicts,

alcoholics and non-dependent controls; they found that both cocaine and heroin addicts had significantly steeper rates of discounting compared to alcoholics and controls (who did not differ). This study suggests that delay discounting may be more closely associated with dependence on some drugs compared to others. Indeed, there is a less consistent relationship between delay discounting and drugs of abuse other than heroin and cocaine. For example, Johnson et al. (2010) found no significant difference in delay discounting rates between cannabis addicts, former addicts and controls. Studies investigating delay discounting in nicotine dependent populations show inconsistent results in both adolescent and adult samples (Baker et al., 2003; Reynolds et al., 2004; Fields et al., 2009b; Reynolds et al., 2003; Melanko et al., 2009; Johnson et al., 2007; Rezvanfard et al., 2010).

The evidence base for the relationship between delay discounting and alcohol use is also inconsistent. There is evidence for increased discounting in heavy drinking adolescents (Field et al., 2007a), undergraduate students (Vuchinich & Simpson 1998) and alcoholics (Bobova et al., 2009; Mitchell et al., 2005) in comparison to light drinking controls. In addition, Murphy and Garavan (2011) found that delay discounting was the best predictor of problem drinking (as assessed by AUDIT scores) in undergraduate students. There have, however, been numerous failures to replicate these findings. For example, Fernie et al. (2010) and MacKillop et al. (2007) failed to show an association between alcohol consumption and discounting in undergraduate drinkers. Similarly, both Kirby and Petry (2004) and Bjork et al. (2004) failed to demonstrate increased discounting rates in alcoholics. A recent meta-analysis (MacKillop et al., 2011) found that effect sizes in delay discounting studies tend to be larger in clinical samples than they do for non-clinical samples. For example, although both alcoholics and non-dependent heavy drinkers showed steeper discounting of delayed rewards than controls, effect sizes are larger in clinical samples. This suggests that inconsistent findings may be due to the samples examined not having high enough levels of dependence. So, for example, we would expect inconsistent results when investigating the relationship between delay discounting and alcohol use in a sample of undergraduates, whereas the association between delay discounting and alcohol use in alcoholics should be much stronger.

1.7.2. Measuring Inhibitory control

The second, independent, measure of impulsivity described by de Wit and Richards (2004) and Olmstead (2006) is deficient inhibitory control. Several different tasks have been developed to measure ability to control or suppress pre-potent responses. The most commonly used methods are the colour conflict Stroop, Go/No-Go, Stop-Signal and antisaccade tasks. The colour conflict Stroop (Stroop 1935) involves participants suppressing a pre-potent response to read a word rather than state the colour the word is written in. For example, if a participant was presented with the word 'blue' written in red ink the correct response would be to state 'red'. Greater latencies in colour-word mismatch trials are indicative of poor inhibitory control, as they are hypothesised to be the result of increased difficulty in suppressing the pre-potent word reading response which is required to state the colour. The other three measures of inhibitory control all involve the suppression of a pre-potent motor response. There are several different versions of the Go/No-Go task in all of which participants are required to respond as rapidly as possible to visually presented 'Go' targets, these can be coloured shapes, pictures, words or numbers, however some targets will be 'No-Go' targets which participants must not respond to. For example in the passive avoidance version of the Go/No-Go task (Colder & O'Connor 2002; Newman & Kosson 1986) participants are presented with eight, two-digit, numbers four of which are 'correct' and four of which are 'incorrect', participants have to learn through trial and error which are the four correct numbers and only respond to them while inhibiting responses to incorrect numbers. An index of inhibitory control is derived from counting the number of commission errors made (responding to No-Go stimuli). The cued Go/No-Go task (Marczinski et al., 2005) is a variation on traditional Go/No-Go tasks in which a cue symbolising the increased probability of the Go stimuli or No-Go stimuli subsequently appearing is presented to participants. The aim of the cue is to initiate the Go response before the No-Go cue is presented so it is increasingly difficult for participants to inhibit their response, this version of the Go/No-Go task therefore has considerable overlap with the Stop-Signal Task. The Stop-Signal task also involves responding to visual cues as rapidly as possible. For example, in one version of the task (Bitsakou et al., 2008) participants are

presented with a large 'X' or 'O' in the centre of the screen and have to respond by pressing the appropriate key as rapidly as possible, on a minority of trials (usually about 25%) a stop-signal is presented (an auditory tone). When participants hear the tone they are instructed to inhibit their response. Usually, the Stop-Signal delay adjusts according to the participant's performance, successful inhibition will add 50 ms to the stop-signal latency (making inhibition more difficult), with failures resulting in a 50 ms decrease in a stop-signal latency (making inhibition easier). Performance is often measured using stop latencies, which is the stop-signal latency at which participants can inhibit 50% of their responses, with higher stop-signal latencies being indicative of better inhibitory control. In addition, stop-signal reaction time (SSRT) can also be calculated, which is an estimate of time taken to inhibit a response while taking 'Go' reaction times into account, with higher scores meaning impaired response inhibition (see Band et al., 2003). Finally, the antisaccade task has also been used as measure of inhibitory control (Hallett 1978). Unlike the Go/No-Go and Stop-Signal tasks this task relies on participants inhibiting a reflex (orientating attention towards cues appearing in the periphery of the visual field), rather than a pre-potent motor response that has been learnt during the task itself. The antisaccade task involves participants inhibiting ocular responses to visual probes appearing in a visual display. These tasks generally consist of two forms of trial; prosaccade trials require participants to look at cues presented, while antisaccade trials require participants to direct attention away from cues. Errors on antisaccade trials, looking toward peripheral cues instead of away, are indicative of a failure in inhibitory mechanisms to halt the reflex response that orientates attention towards peripherally appearing stimuli.

1.7.2.1. Evidence for impaired inhibitory control in drug and alcohol use

As with delay discounting the findings regarding the contribution of impaired inhibitory control to drug use are mixed, with stronger associations being demonstrated for some drugs compared to others. In their assessment of the contribution of inhibitory control to adolescent drug and alcohol use (and related

problems), Nigg et al. (2006) found poor inhibitory control to be associated with both drug and alcohol use but not with drug and alcohol-related problems. Regarding specific drugs, deficits in inhibitory control have been most strongly associated with stimulant-type drugs. Cocaine addicts have been consistently found to have impaired inhibitory control using both Stop-Signal (Fillmore & Rush 2002) and Go/No-Go tasks (Kaufman et al., 2003; Verdejo-García & Pérez-García 2007; Verdejo-García et al., 2007). Similar findings have also been reported in ecstasy users (Halpern et al., 2004) and methamphetamine addicts (Monterosso et al., 2005). Findings with other drugs have been less consistent, neither heroin or cannabis users have been found to have impaired inhibitory control (Verdejo-García et al., 2007; Constantinou et al., 2010; Hester et al., 2009). Similarly, findings with smokers are inconsistent with some studies showing that deficits in inhibitory control predict severity of smoking dependence (Luijten et al., 2011; Billieux et al., 2010), and relapse (Powell et al., 2010). There have, however, been numerous failures to replicate these results in both adults (Dinn et al., 2004) and adolescents (Galván et al., 2011; Reynolds et al., 2007).

as with research into delay discounting there have been mixed findings in regards to the association between inhibitory control and alcohol consumption. Alcohol consumption in non-dependent samples has been shown to be predicted by performance in both Go/No-Go tasks (Colder & O'Connor 2002) and Stop-Signal tasks (Weafer et al., 2011). Murphy and Garavan (2011) also found that performance on a Go/No-Go task predicted alcohol-related problems, although it was more strongly associated with variance in drinking behaviours in non-hazardous drinkers. Furthermore, studies using alcohol dependent samples have also demonstrated poorer inhibitory control as assessed by Stop-Signal tasks (Goudriaan et al., 2006; Lawrence et al., 2009). As with the evidence base in smokers there have also been numerous failures to replicate these findings in heavy vs. light social drinkers (Fernie et al., 2010; Yan & Li, 2009) and alcoholics vs. controls (Duka et al., 2003; Kamarajan et al., 2005). In addition, duration of alcohol dependence did not affect performance on a Go/No-Go task in a study which compared participants with long and short duration of alcohol dependence (Loeber et al., 2009).

There are several explanations for the inconsistent findings regarding the association between inhibitory control and alcohol use. As with delay discounting the diverse methodologies utilised to measure this single construct may have created some ambiguity in the literature. Furthermore, impairments in inhibitory control may be greater in some samples compared to others (alcoholics vs. undergraduate drinkers). An additional possibility is that state factors, such as acute alcohol administration, may mediate the impact that inhibitory control has on alcohol consumption.

1.7.3. Acute alcohol affects on impulsivity and executive cognitive functioning

As with the automatic processing of alcohol-related cues there is also evidence that measures of behavioural impulsivity and executive cognitive function can fluctuate according to situational factors. Indeed, dual process models of cognition such as Strack and Deutsch (2004) explicitly state that controlled processes operate best under certain optimal conditions. The relative instability of these processes may be one of the contributing factors to the mixed findings in studies that assess the contribution of delay discounting and inhibitory control to alcohol use. This suggests that the specific predictions made by dual process models of drug use may be clearly observable in some states yet not in others. For example, if alcohol intoxication increases impulsivity then this may represent a condition in which automatic processes assume greater control over behaviour. Clinically, this indicates that certain circumstances may represent high risk situations increasing the likelihood of relapse.

Interestingly, alcohol primes seem to have different effects on impulsive decision making compared to inhibitory control. The effects of acute priming doses of alcohol on delay discounting are inconsistent. For example, Reynolds et al. (2006b) found no effects of 0.4, 0.5 or 0.8 g/kg priming doses on questionnaire based measures of delay discounting. In addition, Ortner et al. (2003) found 0.7 g/kg of alcohol reduced delay discounting. Significantly, priming doses of alcohol have been shown to have

an impact on inhibitory control as well as other aspects of executive cognitive functioning. For example, priming doses of alcohol in excess of 0.4 g/kg, have been found to reliably impair inhibitory control when assessed with Cued- Go/No-Go tasks (Marczinski et al., 2005; Marczinski et al., 2007) and Stop-Signal tasks (de Wit et al., 2000; Fillmore & Vogel-Sprott 1999; Reynolds et al., 2006b) although there are some null findings (e.g. Ortner et al., 2003 found no effect of 0.7 g/kg prime on a Go/No-Go task, for a review see Field et al., 2010). Significantly, the degree of impairment in inhibitory control caused by an acute alcohol prime has been found to predict ad-lib alcohol consumption (Weafer & Fillmore 2008). Although much research has concentrated on the disinhibiting effects of alcohol on inhibitory control, other aspects of executive functioning have been investigated. Balodis et al. (2007) and Grattan-Miscio and Vogel-Sprott (2005) found that working memory was significantly impaired following 0.7 g/kg and 0.62 g/kg of alcohol respectively. Guillot et al. (2010) found that mental set shifting, assessed by the Trial making test and the Wisconsin card sorting task, was impaired following a priming dose of alcohol. Other measures that assess multiple aspects of executive cognitive functioning, such as phonemic fluency tasks (e.g. controlled oral word association test, COWAT; Benton 1968) are also impaired following high doses (1.0 g/kg) of alcohol (e.g. Peterson et al., 1990).

Aside from acute alcohol primes other state factors have also been shown to impair inhibitory control. In a cue exposure study Gauggel et al. (2010) found that alcohol-related cues (smelling alcoholic drinks) impaired inhibitory control in detoxified alcoholics. Recently a series of studies (Jones et al., 2011a; Jones et al., 2011b) has demonstrated that inhibitory control can be manipulated by priming disinhibited mindsets in social drinkers, which leads to increased alcohol consumption in bogus taste tests suggesting a direct causal relationship between inhibitory control and alcohol consumption. Furthermore, the sensitivity of inhibitory control to state manipulations could explain the inconsistent findings in previous research, if inhibitory control is highly sensitive to state factors then inconsistent results can be explained by individual differences in populations tested and testing conditions.

In addition to directly assessing controlled processes such as inhibitory control other studies have investigated the role of controlled processes in alcohol consumption

from a different perspective. Recently, a considerable amount of research into the ability to control behaviour has focussed on the 'strength' model of self-control (Baumeister et al., 1998). This model proposes that self-control is a limited resource; if demands on self-control are maintained this resource becomes depleted resulting in a reduced ability to control subsequent behaviour, this state of self-control depletion is usually referred to as 'ego depletion' (Baumeister et al., 1998; Muraven, Tice, & Baumeister, 1997). There is a significant evidence base demonstrating that if individuals exert self-control then they find it harder to exert self-control in later tasks even when the domains of the tasks are different (see Hagger et al., 2009 for a review). Regarding alcohol consumption, Muraven et al. (2002) found that if participants had their self-control resources depleted they consumed more beer in a laboratory taste test even when given a financial incentive to control their alcohol consumption. In addition, Muraven et al. (2005) used EMA to investigate fluctuations in self-control demands in real-world settings and found participants who had elevated self-control demands consumed more than their self imposed drinking limits. It is possible that ego-depletion may be mediated by impairments in inhibitory control and decision making processes, indeed ego depletion may represent a state variable which increases the association between these measures and alcohol consumption.

1.8. Evidence base for the moderation of the association between automatic processes and alcohol use by impulsivity

One of the specific predictions of dual process models of addiction is that the automatic processing of alcohol-related cues will have a greater impact on drinking behaviour in more impulsive individuals (those with poor executive control). Theoretically, more impulsive individuals will be more likely to respond in a way that is consistent with their automatic motivation as they lack the necessary resources to control their behaviour and offset the reward of drinking. The direct association between both automatic reactions to alcohol (and drug-related) stimuli as well as impulsivity have been investigated in great detail (as reviewed above). Despite this

the specific predictions involving the moderation of the association between automatic processing of alcohol-related cues and alcohol use by measures of impulsivity and executive cognitive functioning have received surprisingly little attention.

Recently, some research has investigated the specific predictions of dual-process models that the impact of automatic cognitive processes on alcohol use will be moderated by impulsivity/executive cognitive functioning. This research has generally focused on working memory as the controlled process that acts as the moderator in this relationship. In a prospective study Thush et al. (2008) tested automatic alcohol cognition, explicit alcohol outcome expectancies and working memory in a sample of adolescent drinkers. They found that automatic alcohol–active associations (assessed with an IAT) predicted variance in drinking behaviour at one month follow-up in participants with poor working memory but not in those with good working memory. In addition, alcohol consumption in participants with a higher working memory capacity was associated with explicit alcohol outcome expectancies but not in those with poor working memory. Grenard et al. (2008) assessed automatic alcohol and cigarette memory associations (using word association tests) and working memory in a sample of high school students with problem behaviours. Both automatic alcohol-related memory associations and working memory capacity predicted alcohol use and, as predicted by dual process models of addiction, automatic memory associations predicted more variance in alcohol consumption in individuals with poor working memory. Likewise, automatic cigarette-related memory associations also predicted variance in smoking behaviour in those participants with poorer working memory function. However, a similar study tested automatic alcohol-approach associations (using both symbolic and physical approach/avoid responses) in adolescents and did not find any evidence that the association between automatic alcohol-approach responses and alcohol use was moderated by working memory capacity (van Hemel-Ruiter et al., 2011). Finally, in a novel investigation, Houben et al. (2011) found that a working memory training program resulted in reduced alcohol consumption in hazardous drinkers. In support of dual process models of addiction, working memory training only reduced alcohol consumption in individuals with strong automatic alcohol-pleasant associations,

suggesting that the effect of working memory training was mediated by the subsequent reduced impact of automatic cognitive processes, rather than working memory improvements directly affecting alcohol consumption.

The use of working memory as the key moderator of automatic cognition is derived from Kane and Engle (2002), who describe working memory as the aspect of executive cognitive function which subsumes all other components. The evidence for this assertion is mixed, for example both Miyake et al. (2000) and Verdejo-García and Pérez-García (2007) found working memory to be only a sub-component of executive cognitive function. Indeed, some dual process models of addiction (Goldstein & Volkow 2002; Jentsch & Taylor 1999) state that impulsivity (specifically inhibitory control), rather than working memory, is the aspect of executive functioning most likely to be involved in the aetiology of drug abuse and addiction. Despite this assertion there is surprisingly little evidence investigating the role of impulsivity as a moderator of the impact of automatic cognitive processes on drinking behaviour. There is one study to date that has specifically investigated the role of inhibitory control in the moderation of the impact of automatic responses to alcohol-related cues on alcohol consumption (Houben & Wiers 2009b). This study used an IAT to assess automatic associations between alcohol and positive affect and a colour conflict Stroop to assess inhibitory control in a sample of undergraduates. Consistent with the predictions made by dual process models of addiction the impact of automatic processing on alcohol consumption was moderated by individual differences in inhibitory control. Specifically, in participants with poor inhibitory control there was a stronger association between automatic alcohol-positive associations and alcohol use.

As previously discussed ego depletion studies (e.g. Muraven et al., 2002; Muraven et al., 2005) have demonstrated that when self control resources are depleted there is a subsequent increase in alcohol consumption. This raises the possibility that ego-depletion impairs the ability to successfully control behaviour resulting in increased behavioural control by automatic processes. Indeed, Ostafin et al. (2008) found that automatic alcohol-approach associations (measured with an IAT) predicted beer consumption in a taste test, but only following the depletion of self-control resources. From the perspective of dual process models it could therefore be

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hypothesised that the increased behavioural control by automatic alcohol-approach associations found by Ostafin et al. (2008) is the result of ego depletion impairing the ability of controlled processes (e.g. inhibitory control) to moderate their impact on behaviour, although this assertion is yet to be formally tested.

1.9. Hypotheses and Aims

The overall aim of the current thesis is to investigate the contribution of the automatic-processing of alcohol-related cues and behavioural impulsivity to alcohol consumption in different samples of drinkers, with a particular focus on hazardous drinking (as measured by the AUDIT). In addition to investigating the contributions of these two components of cognition in isolation, the specific predictions of dual process models, that the association between automatic processing of alcohol-related cues and hazardous drinking will be moderated by individual differences in behavioural impulsivity will be investigated. The core prediction is that in individuals with higher levels of behavioural impulsivity the impact of automatic alcohol-related cognitions on drinking behaviour will be greater than in individuals with lower levels of behavioural impulsivity. Although there is some evidence that working memory moderates the impact of automatic alcohol-related cognition, only one study to date has examined the role of inhibitory control (Houben & Wiers 2009b). Both Goldstein and Volkow (2002) and Jentsch and Taylor (1999) hypothesise that inhibitory control will moderate the impact of automatic cognitive processes on drinking behaviour. As inhibitory control is hypothesised to be an aspect of behavioural impulsivity (de Wit & Richards 2004; Olmstead 2006) the current thesis will also investigate whether the second aspect of behavioural impulsivity, impulsive decision making, also has a similar role in moderating the impact of automatic cognitive processes on alcohol use. Furthermore, previous investigations into dual process models of addiction have only focused on automatic memory associations and automatic alcohol-arousal and alcohol-positive associations (measured with the IAT), the current thesis also aims to expand upon this. Therefore all of the subsequent studies will assess automatic processing of

alcohol-related stimuli using attentional bias tasks as well as the SRC task to assess automatic approach responses to alcohol-related cues, as these two measures of automatic cognition have been demonstrated to have a causal influence on drinking behaviour (Field & Eastwood 2005; Wiers et al., 2010). By investigating these aspects of automatic and controlled processes the experiments conducted should expand the relatively small evidence base for dual process models of addiction and give a clearer indication of which specific processes are associated with hazardous drinking in non-dependent populations.

These hypotheses were investigated with a series of cross sectional studies. The first two studies used multiple measures of impulsive decision making (study one) and inhibitory control (study two) together with measures of attentional bias and automatic cue-approach in student samples. These two studies tested the direct association between these measures and hazardous drinking as well as the prediction that behavioural impulsivity moderates the impact of automatic processing of alcohol-related cues on hazardous drinking. In the third study these hypotheses were tested in a sample of adolescents. In the final cross sectional study older adults are investigated, as this population will have more cumulative experience with alcohol and have reasons to be motivated to control their drinking. The final two studies attempted to manipulate aspects of impulsivity in order to examine if increasing impulsivity will strengthen the association between automatic processing of alcohol-related cues and ad-lib alcohol consumption. Study five investigated the effects of a 0.65 g/kg alcohol prime (and a placebo) on attentional bias, automatic approach responses, behavioural impulsivity, executive cognitive function and alcohol seeking. In addition to looking at the main effects of the alcohol prime on these measures their contribution to increased alcohol seeking was also investigated. Finally the hypothesis that increases in attentional and approach bias would be mediated by increases in impulsivity/impairments in executive cognitive functioning was also tested. Study six aimed to explore the effects of ego depletion on alcohol seeking and the association between automatic cognitive processing and alcohol seeking. It was hypothesised that ego depletion reduces the ability to control behaviour through its effects on impulsivity and executive cognitive functioning.

Chapter 2

General Methods

The current thesis used multiple methods to assess aspects of automatic responding to alcohol-related stimuli and behavioural impulsivity; therefore these methods are explained in detail in the appropriate chapters. Measures that are used multiple times e.g. the computerised delay discounting task (studies three, four, five and six) are explained in the first instance only. Although the visual probe task was used in all studies except study one, this task was refined throughout the thesis, so is described in the appropriate chapters also. The only behavioural task utilised in all chapters is the Stimulus Response Compatibility (SRC) task which is explained below. In addition to this the AUDIT, Time Line followback (TLFB) and the Barratt impulsivity scales (BIS-11) are used in all chapters (except for the study three in which adolescents are tested), and are therefore also explained in detail along with their psychometric properties below. The majority of this chapter consists of a detailed description of the statistical methods used for data reduction and analysis.

2.1 Materials

Stimulus Response Compatibility (SRC) task (Field et al., 2008). The SRC task was programmed in Inquisit version 1.33 (Millisecond software, 2002). Each trial of the task commenced with the presentation of either an alcohol-related picture or an alcohol-unrelated (control) picture in the centre of the screen along with a small manikin above or below the picture. Participants were instructed to move the manikin either toward or away from the picture by pressing up or down on a two button response box according to the task instructions (see below). If participants made the appropriate response, the manikin moved towards or away from the

picture. If they made an inappropriate response (e.g. pushing the 'up' button when a 'down' response was required) a large red cross was presented in the centre of the screen for 1000 ms.

There were 128 trials of the task in total, split into two blocks of 64 trials. In the 'approach alcohol' block, participants were instructed to move the manikin towards alcohol-related pictures, and away from alcohol-unrelated pictures. These instructions were reversed in the 'avoid alcohol' block. Each block began with eight practice trials in which four alcohol-related and four alcohol-unrelated pictures were presented. After the practice trials, the instructions were then reiterated before participants completed 56 experimental trials. During these trials, the 14 alcohol-related and 14 alcohol-unrelated pictures were each presented twice, once with the manikin above the picture and once with the manikin below the picture. Trials were presented in a random order for each participant. The order of completion of 'approach alcohol' and 'avoid alcohol' blocks was counter-balanced across participants. Reaction time (the time taken to initiate movement of the manikin) was measured on each trial and the dependent variables were mean reaction time during experimental trials of the 'approach alcohol' and 'avoid alcohol' blocks. An overall SRC bias score was derived by subtracting approach alcohol block reaction times from avoid alcohol block reaction times, with higher bias scores being indicative of increased automatic approach responses towards alcohol-related stimuli.

Questionnaires

Time Line Follow Back (TLFB; Sobell & Sobell 1990). The TLFB self report questionnaire was used to assess alcohol consumption in UK units (one UK unit = 8g alcohol). In studies one, two and five participants had to estimate the number of alcohol units consumed over the preceding seven days and in the remaining studies participants reported alcohol consumption over the previous fortnight. Although some previous studies used TLFB procedures that assess alcohol consumption over a thirty day or even a six month period (e.g. Miller & Fillmore 2010) these have shown to be considerably less accurate than for shorter periods (Hoeppner et al., 2010).

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993). The AUDIT was used to assess hazardous drinking. The AUDIT consists of ten fixed response questions regarding alcohol consumption and consequences of drinking. Scores on the AUDIT range between 0 and 40 with scores of 8 or above indicating hazardous or harmful alcohol use. Recently, Reinert and Allen (2007) conducted an extensive reanalysis of the reliability of the AUDIT and found it to be extremely reliable (the median reliability coefficient taken from 18 studies was .83). With regard to internal consistency Shields et al. (2004) found that, in a sample of college students, both consumption and adverse consequences of drinking subscales had good internal validity (α= .81, α= .72 respectively). Furthermore, when viewed as a single factor scale the internal consistency is also good (α= .82), suggesting that the AUDIT is a useful tool when used as a single factor measure of hazardous drinking.

Barratt Impulsivity Scale (BIS-11; Patton et al., 1995). This scale is a multidimensional measure of impulsivity with three subscales – Attentional (eight items), Motor (ten items), and Non-planning impulsiveness (twelve items). The BIS-11 consists of 30 fixed response questions scored from 0-4 with high scores being indicative of increased impulsivity. Total scores on the BIS-11 have been demonstrated to have good internal consistency in student samples (Patton et al., 1995; Cronbach's α = .82, Stanford et al., 2009; Cronbach's α = .83). Although it is worth noting that Stanford et al. (2009) found that the internal consistency of the subscales varied (attentional, α = .74; Motor, α = .59; Non-planning, α = .72). The BIS-11 has been shown to have good criterion validity, as it discriminates students from substance abuse patients and psychiatric patients ($p < .001$). Finally, Stanford et al. (2009) found that total BIS-11 scores had good test re-test reliability ($r_s = .83$, $p < .01$).

2.2. Data Preparation and Analysis

Data Reduction

Before analysis of the reaction times from the tasks which measure automatic responses towards alcohol-related cues, outliers were removed according to criteria used in previous research (e.g. Field et al., 2008). This data reduction involved excluding reaction times less than 200 ms, greater than 2000 ms, as these reaction times represent pre-emptive responding and a loss of concentration respectively. Furthermore individual trial reaction times that were more than three standard deviations above the individual mean were discarded, as these trials are also likely to be the result of the participant losing concentration. Finally, reaction times from error trials were also discarded. Regarding the other cognitive tasks box plots were used to assess outlying participants, which were excluded from the analysis. Finally, distribution was assessed using Kolomogov-Smirov tests, borderline significant Kolomogov-Smirov tests were further assessed using histograms and comparing the standard error of the mean (SEM) to the skewness statistic; skewness statistics over double the SEM were deemed to have violated parametric assumptions. Dependent variables (e.g. AUDIT or beer consumed in taste tests) were log-transformed so that the distribution met parametric assumptions if necessary.

Reporting statistical significance

Throughout this thesis exact p values will be given if they are less than .1. Those p values less than .1 but.05 or greater are referred to as trends. Values less than .05 are deemed statistically significant.

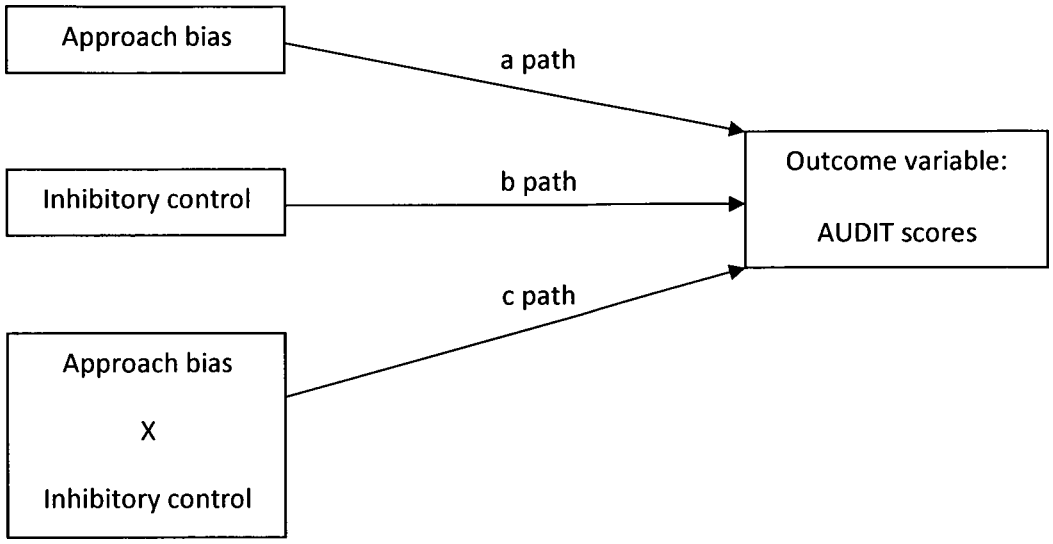
General Analysis Strategy

Integrative models of addiction hypothesise that the association between cognitive bias towards alcohol-related cues and alcohol use is moderated by impulsivity/ECF. To investigate this, studies one to four, and study six used hierarchical regression

analyses to assess the relationship between these variables and hazardous drinking (AUDIT scores/other alcohol use variables). Separate regression analyses were conducted on the different measures of cognitive bias as recent evidence indicates that they are quantitatively distinct (for reviews see Field & Cox 2008; Stacy & Wiers 2010). The exception to this was the adolescent sample (study three) in which the very large sample size allowed for simultaneous analysis of attentional bias and automatic approach responses.

The hierarchical regression analyses for moderation were set up as follows; in the first step demographic variables (if associated with the DV) were added. In the second step self report impulsivity (BIS-11 total scores) are added (although this is not used in study three as it is not validated for use in adolescents). In the third step an automatic cognitive processing measure is added (e.g. SRC bias scores), in the forth step the measures of behavioural impulsivity (and/or ECF) are added (e.g. measures of inhibitory control). Finally, the product of the normalised automatic cognition and moderator variables are added as a final step in the regression equation. This method enables a good approximation of the amount of variance in the dependent variable that is explained by these clusters of variables. The identification of evidence for the moderation of the impact of automatic cognitive processes on alcohol consumption by behavioural impulsivity (and/or ECF) in these regression analyses involves investigating the contribution of the predictor variable (a path), hypothesised moderator (b path) and interaction between predictor and moderator (c path), see Figure 2.1.

Figure 2.1: Schematic overview of moderation analysis.



For the moderator effect to be statistically significant then the c path must significantly predict the DV. Neither the 'a' or the 'b' path needs to be statistically significant for there to be evidence of moderation. However if there is a significant relationship between either the 'a' and 'b' path and the DV then the 'c' path must predict variance beyond that which has been explained by the 'a' and 'b' paths independently (Baron and Kenny 1986).

Recent studies demonstrating the moderation of the relationship between automatic cognition and alcohol use by executive cognitive functioning have had relatively low statistical power. For example Thush et al. (2008) recruited approximately eight participants per predictor variable. Therefore the regression analyses in the current thesis aim to have approximately 10 participants per predictor variable which previous research has shown is sufficient to find evidence of the moderation of the association between automatic cognition and drinking behaviour by impulsivity/ ECF, and is generally recommended as a minimum number of participants per predictor for regression analyses (Field 2009). Demographic variables will only be considered in the regression analysis (as a first step) if they are associated with the

dependent variables. For example, age will be added if it correlates with measures of hazardous drinking, implicit cognition or behavioural impulsivity and multivariate analysis of variance (MANOVA) and subsequent comparisons will be used to investigate gender differences in these measures.

Mediation analysis

Mediation analyses are only utilised in studies five and six. Due to differing sample sizes and number of conditions in these two studies, different techniques to assess mediation were utilised. These are explained in the respective chapters.

Principal component analysis (PCA)

This thesis also investigates the independence of different measures of impulsive decision making (study one), inhibitory control (study two) and both inhibitory control and impulsive decision making (study four) as separable constructs of impulsivity. Furthermore, each one of these studies looks at the independence of behavioural measures of impulsivity from self report impulsivity as measured by the BIS-11 subscales. In order to identify independent dimensions of impulsivity a series of PCA's were conducted.

Based upon the recommendations of Jolliffe (1972; 1986) components in the PCA which had eigenvalues ≥ 0.7 were maintained. The ≥ 0.7 cut-off was selected as Kaiser's rule of maintaining eigenvalues ≥ 1.0 is deemed too conservative in this circumstance as at least some independence of components, i.e. in all PCA's it would be expected that subscales of the BIS-11 would be independent from behavioural measures (see Reynolds et al., 2006a; White et al., 1994). Furthermore, Kaiser's rule is more useful when applied to factor analysis rather than PCA.

In addition, a series of tests were conducted to ensure the validity of the PCA. First, sampling adequacy was established using the Kaiser-Meyer-Olkin measure (KMO). This measure is essentially an index of variance among variables that might be shared variance. KMO values below .5 are considered poor, between .5 and .7 are

considered adequate, .7+ are considered good to excellent (Hutcheson & Sofoniou 1999). In order to ensure sufficient correlations between items to conduct the PCA Bartlett's test of sphericity was also performed; this test approximates a χ^2 distribution for the sample. Significant values ($p < .05$) indicate that there is a high probability that there are significant inter-item correlations to conduct the PCA, values $p > 0.1$ indicate the data is not suitable for PCA.

A orthogonal (varimax) rotation is used in studies one and two as it was expected that there would be a degree of an association between the measure of decision making (study one) and inhibitory control (study two), although it would be expected that these measures are fully independent of the self report measures of impulsivity. In study four it was expected that all the components of impulsivity measured (inhibitory control, decision making and self report impulsivity) would be independent, therefore an oblique rotation (oblimim) was utilised.

Finally the criteria suggested by Stevens (2002) was used for the interpretation of factor loadings. Factor loadings greater than .3 are traditionally considered statistically significant; this method does not take into account sample size. As factor loadings are essentially a Pearson's correlation between the variable being considered and the underlying factor then specific critical values at $\alpha = .01$ can be derived for different sample sizes. For studies one and two in which the sample size is approximately 80, factor loadings greater than .572 are considered significant ($\alpha = .01$, one-tailed), in study four which had a sample size of 95, factor loadings greater than .512 are deemed statistically significant ($\alpha = .01$, one-tailed).

Chapter Three: Experiments 1 & 2

The role of impulsive decision making, inhibitory control and automatic responses to alcohol-related cues in hazardous drinking among students.

3.1. Abstract

According to current theoretical models, automatic responses to alcohol-related-stimuli should drive drinking behaviour, but this relationship should be moderated by individual differences in different components of impulsivity, such as impulsive decision-making and deficient inhibitory control. The current chapter consists of two studies investigating whether impulsive decision-making (study one) and inhibitory control (study two) would moderate the association between approach tendencies and attentional bias elicited by alcohol-related cues on hazardous drinking (AUDIT scores). Participants completed a battery of impulsive decision-making tasks (delay discounting, Two Choice Decision, and Time Estimation tasks in study one) and inhibitory control tasks (Go/No-Go and antisaccade tasks) as well as general measure of executive cognitive functioning (phonemic fluency) in study two. Automatic approach responses were measured with the stimulus response compatibility (SRC) task, and attentional bias was measured with the attentional cueing task (ACT; study one) and visual probe task (study two). Study one found no direct association between attentional bias and AUDIT scores, although maintenance of attention on alcohol-related cues did correlate with AUDIT scores in study two. Approach bias correlated with AUDIT scores in both studies. It is notable that none of these measures predicted AUDIT scores after controlling for age and self report impulsivity. In addition, no measure of impulsive decision making or inhibitory control was directly associated with, or moderated the impact of approach or attentional bias on, AUDIT scores. These results suggest that in non-dependent student drinkers, automatic responses to alcohol-related cues and attentional bias are associated with hazardous drinking, although these associations are relatively weak.

3.2. Introduction

The current chapter aims to investigate the prediction of dual process models of addiction that both heightened salience of alcohol-related cues and increased 'impulsivity' play a central role in the loss of control over alcohol use. For example, it has been suggested that alcohol-related cues acquire conditioned incentive-motivational properties ('incentive salience'), causing those cues to both capture attention and initiate approach behaviours, ultimately leading to alcohol consumption (Robinson & Berridge 1993). More recent models (Goldstein & Volkow 2002; Jentsch & Taylor 1999; Wiers et al., 2007) make similar predictions, although these models also suggest that the ability of alcohol-related cues to initiate drinking behaviour is moderated by the extent to which executive cognitive function has been impaired by chronic alcohol use. For example, Goldstein and Volkow (2002) put forward a model which suggests increased salience attribution to alcohol-related cues should result in increased alcohol consumption. However, they also argue that the relationship between the incentive salience of alcohol cues and alcohol consumption should be moderated by individual differences in executive cognitive functioning which are related to 'impulsive' behaviours and associated with the prefrontal cortex. So, the relationship between the incentive salience of alcohol cues and actual drinking behaviour should be most pronounced in impulsive individuals who have the greatest impairments in executive cognitive function. In contrast, the association between drinking behaviour and the incentive salience of alcohol cues should be relatively weak in non-impulsive individuals with relatively intact executive cognitive function, as the latter individuals should be well equipped to resist the motivational 'pull' of alcohol cues.

The incentive salience of alcohol-related cues has been measured with a variety of cognitive tasks which have been adapted from those used in mainstream experimental psychology. The first two studies will investigate two aspects of this. Firstly, tasks that assess selective attention for alcohol-related cues ('attentional bias') suggest that such cues tend to 'grab the attention' among alcoholics and heavy social drinkers (e.g. Stetter et al., 1995; Townshend & Duka 2001; for a recent

review, see Field & Cox 2008). Individual differences in attentional bias for alcohol-related cues prospectively predict alcohol use among heavy drinking University students (Fadardi & Cox 2008), and the degree of attentional bias predicts relapse or treatment dropout among treatment-seeking alcoholics (Cox et al., 2002). Significantly, laboratory studies have revealed that by training participants to attend to alcohol-related pictures rather than control pictures increases beer consumption in a taste test, suggesting that there is a causal relationship between attentional bias and alcohol consumption (Field & Eastwood 2005). The current chapter aims to investigate attention processing using variations of attentional cueing tasks (ACT, study one; visual probe task, study two). In the standard attentional cueing task, participants are shown a screen with a fixation point and a grey box on either side. An image is then briefly presented in one of the boxes, followed by a probe which appears where the picture was presented (congruent trial) or in the opposite box (incongruent trial). Participants simply have to respond to the position of the probe as rapidly as possible. Theoretically, if attention is oriented towards alcohol-related cues then there should be faster responding on congruent trials and slower responding on incongruent trials when alcohol cues are presented compared to when neutral cues are presented. The visual probe task is a variation of this in which two pictures (an alcohol and control picture matched on perceptual properties) are presented simultaneously. The probe either appears where the alcohol picture was located (congruent trial) or where the neutral picture was presented (incongruent trial). Faster reaction times on congruent trials compared to incongruent trials are indicative of increased attentional bias towards alcohol-related cues. The current chapter will also utilise attentional tasks to investigate which aspects of attention are most strongly associated with hazardous drinking in a student sample. To achieve this variations in the duration of cue presentation, which should allow the determination of which aspects of attentional processes are most strongly associated with hazardous drinking, will be utilised. It is argued that cues presented for SOAs of $\leq$ 100ms assess initial orientation of attention, while cues presented for SOAs $\geq$ 250ms assess the holding/maintenance of attention (Cisler & Koster 2010; Posner & Petersen 1990). Recent research using the visual probe task suggests that within non-dependent samples frequency and volume of alcohol consumption is associated with attentional bias towards cues presented for longer ($\geq$500 ms) durations but not for

shorter durations (Field et al., 2004b; Miller & Fillmore 2010; Townshend & Duka 2001). Alcohol dependent samples, however, tend to orient attention away from cues presented for longer SOAs, but show attentional bias to alcohol-related cues at shorter SOAs ($\leq$ 200 ms; e.g. Vollstädt-Klein et al., 2009; Townshend & Duka 2007).

The second aspect of automatic cognitive processes these two studies will investigate is approach responses elicited by alcohol cues. It has been demonstrated that animals will direct approach behaviours towards cues that have been paired with the availability of alcohol (Krank et al., 2008), and several investigators have developed experimental paradigms for studying cue-elicited approach in humans. Although multiple tasks have been developed to test the strength of associations between alcohol and approach the current thesis will utilise the stimulus response compatibility (SRC) task (De Houwer et al., 2001). This investigates the speed at which heavy and light social drinkers direct symbolic approach and avoidance responses towards alcohol-related pictorial cues. In the SRC task, alcohol-related or alcohol-unrelated (control) pictures are presented on a computer screen alongside a manikin. In one phase of the task (the 'approach alcohol' block) participants are required to rapidly move the manikin towards alcohol-related pictures and away from control pictures; in another phase of the task (the 'avoid alcohol' block) participants are required to move the manikin towards the control pictures and away from the alcohol-related pictures. Heavy, but not light drinkers, respond more rapidly during the 'approach alcohol' block compared to the 'avoid alcohol' block, which suggests that alcohol-related cues elicit an automatic approach tendency among such heavy drinkers (Field et al., 2008). Automatic alcohol-approach associations in heavy drinkers have also been demonstrated using the IAT (Palfai & Ostafin 2003) and AAT (Wiers et al., 2009). In addition, Wiers et al. (2010) found that training hazardous drinkers to approach alcohol-related cues increased beer consumption in a taste test compared to those who were trained to avoid alcohol-related cues which suggests that, like attentional bias, there is a causal relationship between approach responses towards alcohol-related cues and drinking.

The second aspect of cognition proposed by dual process models of addiction to affect alcohol use is controlled processes or executive cognitive functioning.

Executive cognitive function refers to a set of inter-connected cognitive abilities which subserve the ability to inhibit pre-potent responses, hold information in working memory, and switch between different mental sets (Miyake et al., 2000). Impairments in executive cognitive function are associated with chronic alcoholism and heavy social drinking, and this has been suggested as a core factor in loss of control over drinking (Lyvers 2000; Parker et al., 1991). More recently, researchers have studied the role of 'impulsivity' in alcohol abuse and other addictions (see Verdejo-Garcia et al,. 2008, for a recent review). Impulsivity is commonly defined as rash or risky behaviours which do not take into account future consequences and are exemplified by a lack of planning (e.g. Dawe & Loxton 2004). As such the concept of impulsivity has considerable overlap with the notion of executive cognitive (dys)function.

Impulsive cognition and behaviour has been measured directly using experimental procedures, which have the advantage that they do not rely on subjective awareness of impulsivity or the ability or willingness to report it. Both de Wit and Richards (2004) and Olmstead (2006) identified two distinct components of impulsivity, both of which are associated with heavy drinking and alcohol problems. The first component is impulsive decision-making, in which individuals are over-sensitive to immediate rewards but insensitive to delayed rewards or adverse consequences. Impulsive decision making is commonly measured using the delay discounting procedure (e.g. Madden et al., 1997), which assesses the desire for immediate gratification at the expense of longer-term gain. Participants are given a series of choices between small sums of money which are available immediately, versus larger sums of money which are available after a delay. Monetary rewards and delays are often presented hypothetically, and participants are asked to make their choices as if the rewards were real. Such simulations tend to yield similar results to those obtained when real monetary rewards and real delays are used (Madden et al., 2003; Madden et al., 2004). There is some evidence that heavy social drinkers and alcoholics show an increased rate of delay discounting (i.e. preference for smaller immediate rewards) compared to light-drinker controls (Field et al., 2007a; Petry 2001; Vuchinich & Simpson 1998). However, several studies have revealed no association between delay discounting rate and individual differences in alcohol

consumption or alcohol problems (Kirby & Petry 2004; MacKillop et al., 2007; Fernie et al., 2010). In addition to delay discounting methodologies, impulsive decision making has also been assessed with other behavioural tasks. For example, Cherek et al. (1997) developed the Two Choice Delay task to assesses tolerance to experienced rather than hypothetical delays. This task has been shown to distinguish 'impulsive' populations from controls e.g. individuals with a diagnosis of ADHD (Paloyelis et al., 2009) and violent criminals (Cherek & Lane 1999; Cherek et al., 1997). The ability to estimate the passage of time has also been utilised to investigate impulsivity (Wingrove & Bond 1997); theoretically, impulsive individuals should underestimate the passage of time compared to controls. It has been suggested that time estimation is a fundamental component of delay discounting, with fMRI studies showing both tasks use the same brain regions (the posterior insular cortex and striatum;Wittmann et al., 2007; Wittmann & Paulus 2008). Furthermore, Wittmann and Paulus (2008) suggest that individuals with slower internal clock speeds will be more likely to accept a smaller immediate reward as the cost of a delay is percieved as being higher than it is for an individual with a faster internal clock speed, who do not assign as a high a cost to a delay. Recently, Corvi et al (2012) confirmed that internal clock speed predicted a signifcant amount of variance in delay discounting rates in a sample of undergraduate students. Significantly, internal clock speed is associated with dopaminergic functioning, with dopamine agonists decreasing internal clock speed as well as facilitating conditioned motivational responses to drug-related cues (see Meck 1996 for a review). Taken together, this indicates that chronic drug use not only increases the incentive motivational properties of drug-related cues, but also decreases internal clock speed and increases delay discounting rates. There is some experimental evidence that time estimation is an efficacious measure of impulsive decision making. For example, individuals with impulse control problems such as conduct disorder (Dougherty et al., 2007) and cocaine addicts with antisocial personality disorder (Bauer 2001), tend to underestimate the passage of time.

The second, independent, component of impulsivity defined by de Wit and Richards (2004) and Olmstead (2006) is deficient inhibitory control. Inhibitory control refers to the ability to control or suppress pre-potent responses. This construct has been

assessed with behavioural tasks including Go/No-Go (Newman & Kosson 1986), Stop-Signal (Logan et al., 1984), and antisaccade (Hallett 1978) tasks. Although these tasks use differing methodologies, they all involve the participant having to withhold some form of dominant motor response. In the Go/No-Go and Stop-Signal tasks, participants learn to make a rapid motor response to visually presented targets; however, on some trials they are required to withhold their response, such as when a different visual target is presented (Go/No-Go task), or when an auditory stimulus is presented (Stop-Signal task). A failure to inhibit responding when presented with No-Go cues or stop signals reflects a failure of inhibitory control. Similarly, in the antisaccade task, participants' eye movements are recorded while visual stimuli are presented in the periphery of a visual display. On 'antisaccade' trials, participants are required to make antisaccades' - inhibit the tendency to look towards the visual stimulus, and instead shift their gaze in the opposite direction. A failure to initiate an eye movement away from the visual target on antisaccade trials is indicative of a failure of inhibitory control. Some recent studies suggest that heavy drinking and alcoholism are associated with failures of inhibitory control on these tasks (Go/No-Go task: Colder & O'Connor 2002; Stop-Signal task: Goudriaan et al., 2006). However, as with the delay discounting data, the findings are not completely consistent across studies. For example, Fernie et al. (2010) and Kamarajan et al., (2005) did not detect a selective impairment in response inhibition among alcoholics or heavy drinkers using a Stop-Signal and a Go/No-Go task respectively.

In addition to using 'pure' measures of inhibitory control a more general measure of executive cognitive functioning was used in study two. A phonemic fluency task, the controlled oral word association test (COWAT; Benton 1968) was used to investigate whether any association between hazardous drinking and inhibitory control was specific to that aspect of executive cognitive functioning or due to a general weakness in executive cognitive functioning. The COWAT was used for this as it is particularly sensitive to impairment in prefrontal cortex functioning, and it has been used extensively to assess impaired executive cognitive functioning in clinical samples (e.g. Troyer et al., 1998a; Troyer et al., 1998b). Furthermore, it has been suggested that phonemic fluency tasks assess a cluster of executive cognitive functions; working memory, inhibition and mental set switching (Abwender et al.,

2001; Troyer et al., 1997) which indicates that it could be a useful assessment tool for investigating general deficits in executive cognitive function. There are, however, some studies that suggest that the COWAT is a more specifically a measure of access to semantic memory (e.g. Joyce et al., 1996; Lee et al 1999). Likewise, the principal component analysis of Verdejo-Garcia and Perez-Garcia (2007) found that a phonemic fluency task loaded onto an updating factor. Significantly, it has been suggested that updating is the key aspect of executive cognitive functioning, accounting for more unique variance than either mental set switching or inhibitory control (e.g. Miyake & Friedman 2012), and that is also strongly associated with related measures such as intelligence (Joyce et al., 1996).

In addition to these behavioural measures, both studies utilise a measure of self report impulsivity, specifically the BIS-11. Self report impulsivity has been consistently shown to predict indices of alcohol use (e.g. Fernie et al., 2010; MacKillop et al., 2007), indeed the results for self report impulsivity are far more consistent than for behavioural measures. The current studies will therefore control for self report impulsivity to investigate if behavioural measures of impulsivity predict variance in hazardous drinking beyond that explained by the BIS-11.

Previous researchers have generally studied in separate investigations, either the relationship between heavy drinking and the salience of alcohol-related cues or that between heavy drinking and aspects of impulsivity. However, the dual process models discussed above suggest that individual differences in aspects of behavioural impulsivity may moderate the relationship between the salience of alcohol-related cues and individual differences in alcohol consumption. Only three studies of note have found evidence for this. Both Grenard et al. (2008) and Thush et al. (2008) found the impact of automatic alcohol-related cognition (measured by word association tests and an alcohol-active IAT respectively) on drinking behaviour was moderated by working memory capacity in adolescents. In addition, Houben and Wiers (2009b) found that the relationship between heavy drinking and automatic alcohol-positive associations (as measured with an IAT) was moderated by inhibitory control in undergraduate students.

The primary aim of the two studies reported in the current chapter was to test this hypothesis, namely that the association between hazardous drinking and measures of incentive salience of alcohol-related cues (automatic approach responses and attentional bias) would be moderated by individual differences in impulsivity, with negligible or non-existent associations in individuals with low impulsivity, and larger and more robust associations in individuals with high impulsivity. It is important to be mindful that 'impulsivity' does not appear to be a unitary construct, as exemplified by the previously discussed theoretical distinction between impulsive decision-making and deficient inhibitory control (de Wit & Richards 2004; Olmstead 2006), which has been supported by evidence which shows that these are two independent components of behavioural 'impulsivity' (Reynolds et al., 2006a). Therefore two studies were conducted to assess the role of impulsive decision-making (study one) and inhibitory control (study two). Firstly, the current studies assessed the contribution of the automatic processing of alcohol-related cues and impulsivity to hazardous drinking in isolation (as both groups of measures should independently explain variance in hazardous drinking, e.g. de Wit 2009; Robinson & Berridge 2001). Following this, the hypothesis that the association between hazardous drinking and incentive salience of alcohol-related cues would be moderated by impulsive decision-making (study one) or inhibitory control (study two) was examined. In addition to these primary analyses a principal component analysis on the behavioural and self report measures of impulsivity was conducted. This was to investigate whether tasks that ostensibly assess the same aspect of behavioural impulsivity do actually assess the same underlying construct, and to see whether any behavioural measures of impulsivity overlap with self report measures. The hypothesis for the principal component analysis is that measures that assess impulsive decision making (study one) and inhibitory control/executive cognitive functioning (study two) will load on the same factor, whereas self report impulsivity will load onto a separate factor.

3.3 Method

Materials used in both experiments

Cognitive bias tasks

Stimulus Response Compatibility (SRC) task.

Questionnaires

Time Line Follow Back (TLFB).

The Alcohol Use Disorders Identification Test (AUDIT).

Barratt Impulsivity Scale (BIS-11).

Pictorial stimuli

The SRC task and the attentional bias tasks used a picture set containing 14 alcohol-related pictures and 14 (matched) alcohol-unrelated pictures. Alcohol pictures consisted of alcohol-related scenes (such as a bottle and a glass of wine presented on a table). The alcohol-unrelated pictures were matched to the alcohol pictures on perceptual characteristics but did not contain any alcohol-related cues (e.g. a bottle and a glass of water presented on a table). All the pictures were 100mm high X 125mm wide. The picture set was identical to that used by Field and Eastwood (2005) and Field et al. (2008).

Experiment 1

Participants

79 participants (50 female) aged between 18 and 38 years (mean 20.68 ±3.26) participated in the experiment. All participants had to be social drinkers who

consumed at least one alcoholic drink in an average week. Participants were recruited from the student population of the University of Liverpool via intranet advertisement and word of mouth. Potential participants were excluded if they self-reported a current or past alcohol use disorder, or if their vision was not normal or corrected-to-normal. All participants provided informed consent, and the study was approved by the University of Liverpool Ethics committee.

Cognitive bias tasks

The attentional cueing task (ACT; Fox et al., 2001). The ACT was used to investigate differences in engagement and disengagement of attention from alcohol and control visual cues. Each trial of the task consisted of a white fixation cross presented on a black background for 500 ms. Two grey boxes were also presented on either side of the fixation point (60 mm apart) which remained on screen throughout the entire duration of each trial. Following this an alcohol or control picture was presented within one of the grey boxes. Pictorial stimuli appeared for 100 ms (short condition) or 250 ms (long condition). The pictorial stimulus was then blanked out followed by the appearance of a probe (a 2 cm diameter black dot) 200 ms (following the short picture presentation) or 50 ms (following the long picture presentation) after the disappearance of the pictorial stimuli. This resulted in a 300 ms cue onset asynchrony in both the 100 ms and 250 ms conditions. The probe appeared in either the opposite grey box (incongruent trial) or the same box (congruent trial) as to where the pictorial stimulus was presented. Participants had to respond to the position of the probe using a two button response box as quickly as possible.

The ACT consisted of 456 trials in total. Participants first completed eight practice trials in which neutral practice pictures were presented. Participants were then informed that they were to move onto the main task and the instructions were re-iterated. The main task consisted of 448 experimental trials split into two blocks of 224 experimental trials using alcohol or control pictures. Participants were given a one minute break between blocks. The 14 alcohol pictures and 14 neutral pictures were presented in both the left field and the right field four times (three times as

congruent and once as incongruent trials). Participants completed 224 trials at 100 ms delay and 224 at 250 ms delay, 75% of which were valid trials and 25% invalid trials. Parameters used were based on Fox et al. (2001) - experiments 2 and 3. The average reaction time to the probe was measured as the dependent variable. Bias scores for each presentation time were calculated by subtracting congruent reaction times from incongruent reaction times. Higher scores are indicative of greater attentional bias

Impulsive decision making tasks

Delay Discounting task (Field et al., 2007a). This is a paper and pencil task which measures the degree of preference for small immediate monetary rewards over larger monetary rewards that are available after a variable delay. This version was identical to one used in a previous study from our group (Field et al., 2007a), which was itself based on a measure developed by Giordano et al. (2002). The task consisted of 7 blocks, each of which contained 27 choices (e.g. '£250 now or £500 after one week'). For each choice, participants were required to indicate their preference for either the immediate or the delayed sum of money. As in previous studies that used this task, all choices were hypothetical – participants were informed that they would not receive any money, but they were instructed to respond as if the choices were real.

Each block had a different delay period; in the first block the choice was between a variable amount of money available immediately versus a large amount of money (fixed at £500) which was available after a delay of one week. In subsequent blocks, the delay was increased to two weeks, one month, six months, one year, five years and twenty-five years. The values of the immediate rewards were 100% (£500), 99%, 96%, 92%, 85%, 80%, 75%, 70%, 65%, 60%, 55%, 50%, 45%, 40%, 35%, 30%, 25%, 20%, 15%, 10%, 8%, 6%,4%, 2%, 1%, 0.5% and 0.2% (£1) of the value of the delayed reward. Participants completed the choices in one of two sequences, which were counterbalanced across participants (see Heil et al., 2006). In the descending sequence, the value of the immediate reward on the first trial was 100% of the value of the delayed reward, and the value of the immediate reward decreased

on successive trials. In the ascending sequence, the value of the immediate reward on the first trial was 0.2% of the value of the delayed reward, and it increased on successive trials. As in previous studies that used this measure (e.g. Field et al., 2007a), an 'indifference point' was obtained for each of the 7 delays, which was defined as the value of the immediate reward at which participants switched their preference from the delayed reward to the immediate reward (ascending condition) or from the immediate reward to the delayed reward (descending condition). Indifference points for each of the 7 delays were analysed by computing area under the curve (AUC) values (Myerson et al., 2001). Lower values of AUC indicate steeper delay discounting, or increased impulsive decision-making, therefore AUC values were reversed for subsequent analyses so that high scores were indicative of increased impulsivity.

The Two Choice Delay task (Dougherty et al., 2003). This is an additional measure of delay discounting in which participants actually experience delays before delivery of delayed rewards (unlike the paper and pencil measure described above). Participants were instructed to earn as many 'points' as possible by selecting one of two symbols on the computer screen with a computer mouse. Selection of one of the symbols, a black square (Delayed Large; DL) produced a large reward (15 points) after a relatively long variable delay, whereas selection of the other symbol, a black circle (Immediate Small; IS) produced a small reward (5 points) after a fixed short delay (5 sec). Selection of one of the symbols caused both symbols to disappear for the delay period before the selected symbol was presented again, this time flashing. Participants could earn points at this stage by clicking on the flashing symbol. A running total of the points earned was displayed on the computer screen for the entire duration of the task.

The delay to delivery of DL was initially set at 15 sec but it was adjusted in response to the selections made by the participant. Each selection of IS resulted in reduction of the delay for DL by 2 sec, while each selection of DL resulted in an increase in the delay for DL by 2 sec. Participants initially completed 6 practice trials, which were not analysed, before completing 50 critical trials. Each trial terminated with

delivery of points after a variable delay, as detailed above. Participants were not informed of the relationships between the different stimuli and the number of points available or the delay to delivery of points. The dependent variable was the mean delay until delivery of points after clicking DL; shorter delays indicate more impulsive responding. Again, like with AUC values, these mean delay scores were reversed so impulsive responding was represented by higher scores.

Time Estimation Task (Dougherty et al., 2003b). This task was used to assess participant time perception. Participants were given verbal instructions to estimate the passage of one minute, five times. Participants were presented with a grey screen with 'click to start timer' in black lettering at the top of the screen. The participant was required to press the left key on a computer mouse to start the estimation period, during which the lettering changed to say 'click to stop timer', and then press the left mouse key again when they estimated one minute has passed. On the second mouse click feedback (the actual time they estimated as being a minute) was given on the screen. This was completed five times for each participant and their mean estimation was recorded as the dependent variable in this task, with underestimation of the time period being indicative or increased impulsivity. Like with the previous tasks these scores were reversed.

Experiment 2

Participants

80 participants (48 female) aged between 18 and 30 years (mean 19.23 ±2.54) participated in the experiment. All participants had to be social drinkers who consumed at least one alcoholic drink in an average week. Participants were recruited from the student population of the University of Liverpool via intranet advertisement and word of mouth. Potential participants were excluded if they self-reported a current or past alcohol use disorder, or if their vision was not normal or

corrected-to-normal. All participants provided informed consent, and the study was approved by the University of Liverpool Ethics committee.

Eye Tracking

Eye movements were recorded during the visual probe task using the Eyetrace 300x system (Applied Science Laboratories, Bedford, MA, USA). This is a head mounted system that uses infra-red to track horizontal eye movements. To avoid participants moving their heads instead of their eyes during this task an adjustable table mounted chin rest was utilised.

Cognitive bias tasks

The visual probe task. The visual probe task was programmed in Inquisit version 1.33 (Millisecond software, 2002). Each trial of the visual probe task commences with a central white fixation cross presented on a black background for 500 ms. Immediately after this a pair of pictures was presented for 200 ms or 2000 ms, one picture to the left of the fixation the other to the right, 60mm apart. Immediately after picture offset a probe (a white arrow on a black background, pointing up or down) appeared in one of the picture locations. Participants had to respond to the orientation of the probe by pressing up or down on a two button response box. There was an intertrial interval of 500 ms.

The visual probe task consisted of 130 trials in total. Participants first completed 16 practice trials in which neutral picture pairs were presented, following which instructions were re-iterated before they completed the main task. The main task consisted of 2 buffer trials (of neutral picture pairs) followed by 112 critical trials. Each of the 14 picture pairs appeared eight times with the alcohol and control pictures appearing twice on the right side of the screen and twice on the left with probes replacing alcohol and control pictures an equal number of times in both the 200 ms and 2000 ms trial types. Trials were presented in a new random order for each participant. Reaction time (the time taken to react to the orientation of the

probe) was measured on each trial and the dependent variables were mean reaction time to congruent probes (those that appeared in the same position of the alcohol picture) compared to incongruent probes (those that appeared in the same position of the neutral picture). An attention bias score (for each trial type) was derived by subtracting congruent probe reaction times from incongruent probe reaction times, with higher scores being indicative of increased attentional bias.

Measures of inhibitory control and executive cognitive function

Antisaccade task (Pettiford et al., 2007). This task was also programmed using Inquisit 1.33 (Millisecond Software, 2002). This task measures the ability of participants to direct eye movements towards or away from peripherally presented targets. During the task participants were required to wear the head mounted Eyetrace 300x goggles in order to monitor their horizontal eye movements. Participants rested their head on a chin rest 1m from the computer screen in order to minimise head movements during testing. In this task there were two trial types, antisaccade and prosaccade. Participants had to withhold eye movements towards illuminated peripheral targets on antisaccade trials and to direct eye movements towards illuminated peripheral targets on prosaccade trials.

At the start of the task the equipment was calibrated while participants fixated on a central fixation cross for 10 seconds. On each trial a central fixation symbol appeared for 1500 ms. Participants were instructed that a circle (o) fixation symbol indicated that an antisaccade trial was to follow and a cross (+) fixation symbol indicated that a prosaccade trial was to follow. After the fixation point disappeared there was a 250 ms blackout followed by an illumination of one of two peripheral targets (2cm diameter white dots) 10cm away from the fixation point on the left or right of the screen for 1750 ms. On prosaccade trials participants were instructed to look at the target when it appeared; on antisaccade trials participants were instructed to direct their gaze in the opposite direction of the target at approximately the same distance from the central point. After a 200 ms blackout a red cross appeared in the correct location; i.e. where the participants should have directed their gaze. This

feedback appeared regardless of whether the participants responded correctly or incorrectly.

The task consisted of 8 practice trials (two antisaccade trials and two pro-saccade trials in each probe position), following which instructions were reiterated before the 110 experimental trials. The 110 experimental trials consisted of 30 prosaccade trials and 80 antisaccade trials presented in a random order with an even number of left and right targets across both trial types.

An incorrect response on antisaccade trials was defined as trials in which participants directed their gaze towards, rather than away from, targets at least 150 ms after target onset. Prosaccade errors (looking away from the target stimuli in prosaccade trials) were defined in the same way as for antisaccade errors though gazes would have to be directed away from, rather than towards, targets. Task parameters and definition of errors are taken from Pettiford et al. (2007). The primary dependent variable extracted from the task was the total number of antisaccade errors; more errors indicate impaired inhibitory control.

Go/No-Go task (Newman & Kosson 1986). This 'passive avoidance' version of the Go/No-Go task was programmed in Inquisit version 1.33 (Millisecond software, 2002). The task requires participants to learn through trial and error which numerical stimuli are 'correct' (Go cues) and which are 'incorrect' (No-Go cues). Participants were instructed to withhold responses to the incorrect stimuli (No-Go cues), but respond quickly to correct stimuli (Go cues) by pressing the spacebar on the keyboard. On each trial of the task, one of eight two-digit numbers was presented. Four numbers (34, 42, 51, 93) were Go cues and four (18, 29, 63, 85) were No-Go cues. Participants initially completed 8 practice trials, in which each number was presented once, followed by three blocks of experimental trials. Each experimental block consisted of 24 trials in which each of the eight numbers was presented 3 times each. After completion of each block, participants received feedback on the percentage of correct responses to both go and no-go cues.

Each trial began with the presentation of a white fixation cross in the centre of the screen for 1000 ms, before a Go or No-Go cue was presented. Cues remained on the screen until a response or a three second timeout period had elapsed. Correct responses to Go cues resulted in the text 'Correct!' appearing on the screen in green font for 300 ms. Commission errors (inappropriate responses to No-Go cues) resulted in the text 'Wrong!' appearing on the screen in red font for 300 ms. If no response was made no feedback was given. The primary dependent measure from this task was the number of commission errors, with a high rate of these being indicative of impaired inhibitory control.

Phonemic Fluency: The Controlled Oral Word Association Test (COWAT; Benton 1968). This task was used to assess phonemic fluency as a measure of executive functioning. In this task participants were given a letter and instructed that they had one minute to verbally state as many words beginning with that letter as possible, excluding proper nouns and identical words with a different suffix, (e.g. talk*ing*, talk*ative*). Participants produced words for the letters F, A and S. A voice recorder was used to record responses for future analysis. The dependent measure from the COWAT was the total number of switches between word clusters (with a greater number of switches reflecting increased executive cognitive functioning). Word clusters were defined as consecutive words which begin with the same two letters, which differed only by a vowel, or were homonyms or rhyming words (Troyer et al., 1997). This method for assessing switches was found to best reflect frontal functioning in phonemic fluency as well as having high test-retest reliability (Ross et al., 2007). Scores on the COWAT were reversed before analysis.

Design (experiments 1 and 2)

Both experiments used a correlational design to investigate whether measures of automatic cognitive processing and behavioural impulsivity predicted variance in hazardous drinking (AUDIT scores). In addition to looking at these factors in isolation the interaction between measures of automatic cognition and behavioural

impulsivity in the prediction of hazardous drinking was also investigated. As measures of automatic cognition have been shown to be quantitatively distinct from each other the impact of each measure of automatic cognition (as well as its interaction with multiple measures of behavioural impulsivity) were assessed separately.

Procedure (experiments 1 and 2)

Upon arrival in the laboratory participants were breathalysed using a Lion Alcometer 500 (Lion laboratories, Barry U.K.). All participants had a breath alcohol level of zero. Participants then provided informed consent before completing the questionnaire measures described above. Participants then completed a battery of cognitive tasks which included the following: in experiment 1, participants completed the SRC task, the ACT, the Delay Discounting task, Two Choice task and the Time Estimation task, this testing session lasted approximately 105 minutes. In experiment 2, participants completed the SRC task, the visual probe task, the Go/No-Go task, Antisaccade task and the COWAT, with the testing session lasting approximately 50 minutes. At the end of the experiment participants were thoroughly debriefed before receiving course credit or financial compensation (£10) for their travel expenses and time.

3.4. Results

3.4.1. Experiment 1

Sample Characteristics

Participants consumed a mean of 20.50 (±20.39 range 1-84) UK units of alcohol per week (1 UK unit = 8g alcohol), and the sample mean AUDIT score was

11.47(±6.80). For descriptive statistics and correlations between all questionnaire measures and cognitive tasks see Table 3.1.

Gender differences in alcohol use, self report impulsivity and all cognitive tasks (Table 3.2)

Between subjects ANOVAs revealed that males did not significantly differ from females on any measures of alcohol use or cognition, for descriptive statistics see Table 3.2.

Principle component analysis for dimensions of impulsive decision making

The PCA was conducted on the behavioural measures impulsive decision making and BIS-11 subscales with orthogonal rotation (varimax). The sampling adequacy was deemed to be good (KMO = .712) and Bartlett's test of sphericity demonstrated that correlations between items were large enough for PCA ($\chi^2(10) = 58.077$, $p < .001$). The PCA revealed four distinct components that explained 82.44% of variance. Table 3.3 shows the factor loadings following rotation, which suggests that cluster one represents self report impulsivity, cluster two represents delay discounting and cluster three represents Two Choice mean delay and cluster four represents time estimation. This indicates that the questionnaire measure of impulsivity is distinct from all the behavioural tasks and that, contrary to expectations, the measures of impulsive decision making do not load onto the same underlying factor.

Predicting hazardous drinking: Initial orientation of attention and impulsive decision making (Table 3.4)

The full regression model predicted 21% of the variance in AUDIT scores (R^2 adjusted = 0.21, $F(9, 61) = 3.06$, $p = .004$). Age was a significant predictor of AUDIT scores (β=-.30, $p = .018$), with older drinkers having lower scores, age accounted for approximately 5% of variance in AUDIT scores explained by the

regression model (see Step 1 table 3.4). The only other significant predictor of AUDIT score was BIS-11 total scores (β=.41, $p < .001$), with high BIS-11 scores being associated with high AUDIT scores. Attentional bias in the 100 ms condition of the ACT was a not significant predictor of AUDIT scores ($p > .1$); this suggests that initial orientation of attention is not associated with hazardous drinking. Furthermore there was no association between any of the measures of impulsive decision making and AUDIT scores (all ps > .1). Finally, there was no evidence that any of the measures of impulsive decision making moderated the association between attentional bias and AUDIT scores as none of the interaction variables were found to be significant predictors of alcohol AUDIT scores (all ps > .1).

Predicting hazardous drinking: Maintenance of attention and impulsive decision making (Table 3.5)

The full regression model predicted 20% of the variance in AUDIT scores (R^2 adjusted = 0.20, $F(9,61) = 2.91$, $p = .006$). Again, age was a significant predictor of AUDIT scores in the full regression model (β=-.24, $p = .018$), with older drinkers having lower scores. The only other significant predictor of AUDIT score was BIS-11 total scores (β=.41, $p < .001$), with high BIS-11 scores being associated with high AUDIT scores. As before Attentional bias in the 250 ms condition of the ACT was not a significant predictor of AUDIT scores ($p > .1$) this suggests that maintenance of attention on alcohol-related cues is not associated with hazardous drinking. As before, there was no association between any of the measures of impulsive decision making and AUDIT scores (all ps > .1). Finally, there was no evidence that any measure of impulsive decision making moderated the association between attentional bias and AUDIT scores as none of the interaction variables were found to be significant predictors of AUDIT scores (all ps > .1).

Predicting hazardous drinking: Automatic approach responses and impulsive decision making (Table 3.6)

The full regression model predicted 23% of the variance in AUDIT scores (R^2 *adjusted* = 0.23, $F(9,64) = 3.43$, $p = .002$). As before age (β=-.29, $p = .021$), and BIS-11 total scores (β=-.43, $p < .001$) were significant predictors of AUDIT scores. SRC bias scores were not a significant predictor of AUDIT scores ($p > .1$). Again, there was no significant association between any of the measures of impulsive decision making and AUDIT scores (all ps > .1). Finally, there was no evidence that any measure of impulsive decision making moderated the association between approach bias and AUDIT scores as none of the interaction variables were found to be significant predictors of alcohol AUDIT scores (all ps > .1).

Table 3.1: Descriptive statistics and correlations between age, alcohol consumption measures, self report impulsivity and cognitive tasks, experiment 1

	Mean (±SD)	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	20.68(3.26)	-0.29**	-0.31**	0.04	-0.09	-0.11	-0.09	-0.19*	-0.08	-0.26*	0.13	0.09	-0.45**
2. Past 7 day alcohol consumption	20.11(20.72)	-	0.72**	0.21*	0.26**	0.39**	0.38**	0.06	-0.02	0.21*	-0.00	-0.21*	0.24*
3. AUDIT	11.47(6.84)		-	0.30**	0.27**	0.47**	0.46**	-0.02	-0.18*	0.21*	0.10	-0.22*	0.16
4. BIS-11 (Attentional)	17.95(2.63)			-	0.42**	0.53**	0.76**	0.06	0.13	-0.01	0.22*	0.25*	0.01
5. BIS-11 (Motor)	23.01(3.60)				-	0.39**	0.74**	0.11	0.10	-0.07	0.12	0.21*	0.02
6. BIS-11(Non-planning)	26.57(5.09)					-	0.87**	0.07	0.10	0.11	0.24*	0.23*	0.12
7. BIS-11 (Total)	67.53(9.07)						-	0.10	0.13	0.03	0.25*	0.28**	0.08
8. ACT RT bias (100ms)	1.38(18.82)							-	-0.01	0.08	-0.01	-0.10	0.17
9. ACT RT bias (250ms)	-0.33(19.99)								-	-0.09	0.13	0.00	0.01
10. SRC Bias	33.57(99.30)									-	-0.06	0.01	0.06
11. Delay Discounting (AUC)	0.60(0.23)										-	-0.16	-0.10
12. Two choice mean delay (sec)	16.29(8.00)											-	0.00
13. Mean time estimate (sec)	59.93(7.30)												-

Weekly alcohol consumption (UK units), 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol use = combined Z scored weekly alcohol consumption and AUDIT. Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. ACT RT = Mean reaction time to probes incongruent to alcohol cues minus mean reaction times to congruent cues, higher values indicate increased attentional bias; ms represent duration of cue presentation. SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimate = Mean duration of 5 estimates of the passage of one minute. **$p < .01$ *$p < .05$.

Table 3.2: Comparison of gender differences, experiment 1. Values are Mean (±SD).

	Male	**Female**
	(N=29)	(N=50)
Age (years)	20.93 (2.90)	20.53 (3.48)
Past 7 day alcohol consumption	23.11 (24.86)	18.99 (17.39)
AUDIT	12.66 (7.37)	10.78 (6.49)
BIS-11 Non-Planning	27.45 (5.25)	26.06 (4.98)
BIS-11 Motor	22.00 (3.14)	23.60 (3.75)
BIS-11 Attentional	17.97 (2.64)	17.74 (2.65)
BIS-11 Total	67.41 (9.02)	67.60 (9.19)
ACT RT bias (100ms)	-0.39 (20.64)	2.25 (21.54)
ACT RT bias (250ms)	4.40 (15.74)	-2.65 (18.03)
SRC bias	45.46 (114.55)	27.03 (90.43)
Delay Discounting (AUC)	0.60 (0.20)	0.60 (0.24)
Two Choice Delay mean (sec)	17.80 (8.40)	15.45 (7.73)
Mean Time estimate (sec)	60.34 (6.19)	59.69 (7.90)

Weekly alcohol consumption (UK units), 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol use = combined Z scored weekly alcohol consumption and AUDIT. Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. ACT RT = Mean reaction time to probes incongruent to alcohol cues minus mean reaction times to congruent cues, higher values indicate increased attentional bias; ms represent duration of cue presentation. SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimate = Mean duration of 5 estimates of the passage of one minute. **$p < .01$ *$p < .05$.

Table 3.3: Principle component analysis for behavioural measures of impulsive decision making and BIS-11 subscales (N=78)

	Rotated components			
	1	2	3	4
Eigenvalues	2.18	1.05	0.88	0.84
Variance (%)	36.31	17.54	14.59	14.00
BIS-11 Attentional	**0.82**	0.00	0.28	-0.25
BIS-11 Motor	**0.78**	-0.05	-0.01	-0.21
BIS-11 Non-planning	**0.78**	0.26	0.32	-0.22
Mean Time estimation	0.04	**0.98**	-0.02	0.00
Two Choice Mean delay	-0.27	-0.01	-0.14	**0.99**
Delay discounting(AUC)	0.22	-0.02	**0.98**	-0.16

Note: Factors highlighted load above 0.572 and are considered significant (Stevens, 2002).
Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimate = Mean duration of 5 estimates of the passage of one minute.

Table 3.4: Regression analysis showing trait impulsivity, impulsive decision making measures, and initial orienting of attention (attentional cueing task reaction time, ACT RT), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.06	.05	$F(1,69) = 4.67$*	-0.87	0.39	-.29*
Step 2						
BIS-11 total score	.26	.24	$F(1,68) = 18.16$**	0.32	0.09	.41**
Step 3						
ACT RT bias (100 ms)	.27	.25	$F(1,67) = 1.45$	-0.07	0.04	-.19
Step 4						
Delay discounting (AUC)	.30	.23	$F(3,64) = 0.64$	.45	3.74	.01
Two Choice Mean delay				0.19	0.13	.18
Mean Time estimation				-0.02	0.14	-.02
Step 5						
ACT RT (100ms)XDelay discounting (AUC)	.31	.21	$F(3,61) = 0.38$	0.04	0.20	-.02
ACT RT (100ms)XTwo Choice Mean delay				0.00	0.01	.02
ACT RT (100ms)XMean Time estimation				-0.01	0.01	-.13

BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum). ACT RT = Mean reaction time to probes incongruent to alcohol cues minus mean reaction times to congruent cues, higher values indicate increased attentional bias; ms represent duration of cue presentation. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimation= Mean duration of 5 estimates of the passage of one minute. Cognitive bias measureXImpulsive decision making measure = product of normalized variables. *$p < .05$, **$p < .01$.

Table 3.5: Regression analysis showing trait impulsivity, impulsive decision making measures, and maintenance of attention (attentional cueing task reaction time, ACT RT), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.06	.05	$F(1,69) = 4.67$*	-0.72	0.36	-.24*
Step 2						
BIS-11 total score	.26	.24	$F(1,68) = 18.16$**	0.32	0.09	.41**
Step 3						
ACT RT bias (250 ms)	.27	.24	$F(1,67) = 1.03$	0.03	0.04	.10
Step 4						
Delay discounting (AUC)	.29	.22	$F(3,64) = 0.55$	1.44	3.77	.04
Two Choice Mean delay				0.13	0.12	.12
Mean Time estimation				-0.04	0.14	-.04
Step 5						
ACT RT (250ms)XDelay discounting (AUC)	.30	.20	$F(3,61) = 0.29$	-0.10	0.20	-.05
ACT RT (250ms)XTwo Choice Mean delay				-0.00	0.01	-.01
ACT RT (250ms)XMean Time estimation				0.01	0.01	.09

BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum). ACT RT = Mean reaction time to probes incongruent to alcohol cues minus mean reaction times to congruent cues, higher values indicate increased attentional bias; ms represent duration of cue presentation. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimation= Mean duration of 5 estimates of the passage of one minute. Cognitive bias measureXImpulsive decision making measure = product of normalized variables. *p < .05, **p < .0

Table 3.6: Regression analysis showing trait impulsivity, impulsive decision making measures, and automatic approach responses (SRC bias), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.07	.05	$F(1,72) = 5.11$*	-0.74	0.31	-.29*
Step 2						
BIS-11 total score	.26	.24	$F(1,71) = 18.46$**	0.33	0.09	.43**
Step 3						
SRC Bias (ms)	.28	.25	$F(1,70) = 1.70$	0.06	0.01	.21
Step 4						
Delay discounting (AUC)	.29	.23	$F(3,67) = 0.40$	2.30	3.55	.07
Two Choice Mean delay				0.07	0.12	.08
Mean Time estimation				-0.01	0.12	-.01
Step 5						
SRC Bias (ms)XDelay discounting (AUC)	.33	.23	$F(3,64) = 1.11$	-0.02	0.03	-.06
SRC Bias (ms)XTwo Choice Mean delay				0.00	0.00	.05
SRC Bias (ms)XMean Time estimation				0.00	0.00	.21

BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum). SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Two Choice task mean delay = mean delay to delivery of the delayed large reinforcer, higher delays indicate steeper delay discounting. Mean time estimation= Mean duration of 5 estimates of the passage of one minute. Cognitive bias measureXImpulsive decision making measure = product of normalized variables. *$p < .05$, **$p < .01$

3.4.2. Experiment 2

Sample Characteristics

Participants consumed an average of 24.44 (±21.17 range 1-134) UK units of alcohol (1 unit = 8g alcohol) in the last week, and mean AUDIT scores were above the cut off for hazardous drinking 12.18 (±5.58). For descriptive statistics and correlations between all questionnaire measures and cognitive tasks see Table 3.7.

Gender differences in alcohol use, self report impulsivity and all cognitive tasks

Between subjects ANOVA's revealed that males did not significantly differ from females on any measures of alcohol use or cognition, for descriptive statistics see Table 3.8.

Principle component analysis for dimensions of inhibitory control

The PCA was conducted on the behavioural measures inhibitory control, phonemic fluency, and BIS-11 subscales with orthogonal rotation (varimax). The sampling adequacy was deemed to be acceptable (KMO = .723) and Bartlett's test of sphericity demonstrated that correlations between items were large enough for PCA ($\chi^2(10) = 85.98$, $p < .001$). The PCA revealed four distinct components that explained 85.61% of variance. Table 3.9 shows the factor loadings following rotation, which suggests that cluster one represents trait impulsivity, cluster two represents Go/No-Go errors, cluster three represents COWAT switches and four represented antisaccade errors. Like in experiment one the questionnaire measures of impulsivity were distinct from the behavioural tasks and, contrary to expectations, none of the measures of inhibitory control, and the more general measure of executive cognitive functioning loaded onto the same factor.

Predicting hazardous drinking: Initial orientation of attention and inhibitory control (Table 3.10)

The full regression model predicted approximately 13% of variance in AUDIT scores (R^2 adjusted = 0.13, $F(8,66) = 2.23$, p = .031). The only significant predictor of AUDIT scores was BIS-11 total scores (β=.40, p = .001), with higher scores being associated with increased AUDIT scores. Attentional bias in the 200 ms SOA condition of the visual probe task did not significantly predict AUDIT scores. Furthermore, No-Go errors, antisaccade errors or COWAT switches did not predict significant variance in AUDIT scores. Finally, the interactions between attentional bias in the 200 ms SOA condition and measures of inhibitory control / ECF did not explain additional variance in AUDIT scores (ps > .1).

Predicting hazardous drinking: Maintenance of attention and inhibitory control (Table 3.11)

The full regression model predicted 13% of the variance in AUDIT scores (R^2 = 0.23, R^2 adjusted = 0.13, $F(9,65) = 2.21$, p = .033). Again, the only significant predictor of AUDIT scores in the simultaneous model was BIS-11 total scores (β=.40, p = .001), with higher scores being associated with increased AUDIT scores. Attentional bias in the 2000 ms SOA condition did not predict AUDIT scores in the simultaneous model. There was no evidence of a direct association between No-Go errors, antisaccade errors or COWAT switches and AUDIT scores. The interactions between attentional bias in the 2000 ms SOA condition and Go/No-Go errors, antisaccade errors or COWAT scores did not explain additional variance in AUDIT scores (ps > .1).

Predicting hazardous drinking: Automatic approach responses and inhibitory control (Table 3.12)

The full regression model predicted 13% of variance in AUDIT scores (R^2 adjusted = 0.13, $F(9,64) = 2.16$, p= .037). Again, the only significant predictor of AUDIT scores was in the simultaneous model BIS-11 total scores (β=.32, p = .009), with higher scores being associated with increased AUDIT scores. Although SRC bias were not a significant predictor in the simultaneous model it is notable that in the cumulative model SRC bias scores predicted a significant amount of variance (5%) when controlling for age and BIS-11 scores (R^2 change = 0.04, $F(1,70) = 3.31$, p = .048). No-Go errors, antisaccade errors or COWAT switches (all ps > .1) did not predict a significant amount of variance in AUDIT scores. Finally, the interactions between SRC bias and measures of inhibitory control / ECF did not explain additional variance in AUDIT scores (ps > .1).

Table 3.7: Descriptive statistics and correlations between age, measures of alcohol consumption, self report impulsivity and all cognitive tasks in experiment 2.

	Mean (±SD)	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	19.23 (2.54)	-0.14	-0.18	-0.04	-0.09	-0.10	-0.05	0.10	0.04	0.22*	-0.02	-0.01	0.35**
2. Past 7 day alcohol consumption	24.44 (21.18)	-	0.64**	0.14	0.23*	0.35**	0.30**	0.14	0.25*	0.13	-0.02	0.01	-0.06
3. AUDIT	12.18 (5.58)		-	0.27**	0.30**	0.34**	0.37**	0.05	0.23*	0.20*	-0.03	0.04	-0.18
4. BIS-11 (Attentional)	18.78 (2.85)			-	0.58**	0.52**	0.77**	0.01	0.22*	0.09	0.21*	019*	-0.10
5. BIS-11 (Motor)	24.68 (3.92)				-	0.58**	0.85**	-0.06	0.07	0.05	0.18	0.21*	-0.06
6. BIS-11(Non-planning)	26.35 (5.11)					-	0.88**	0.01	0.32**	0.15	0.19*	0.10	-0.07
7. BIS-11 (Total)	69.80 (10.04)						-	-0.02	0.25*	0.12	0.22*	0.19*	0.09
8. VP Bias (200ms)	-9.14 (42.89)							-	0.16	0.09	-0.22*	-0.28**	-0.08
9. VP Bias (2000ms)	4.46 (53.36)								-	0.19*	0.08	-0.06	-0.10
10. SRC Bias	29.39 (112.24)									-	-0.14	-0.11	-0.08
11. Antisaccade errors	31.85 (17.65)										-	0.21*	-0.14
12. Go\No-Go (No-Go errors)	10.66 (6.77)											-	-0.15
13. COWAT switches	24.26 (8.86)												-

Weekly alcohol consumption (UK units), 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol use = combined Z scored weekly alcohol consumption and AUDIT. Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. **p < .01 *p < .05.

Table 3.8: Comparison of gender differences, experiment 2. Values are Mean (±SD).

	Male	**Female**
	(N=32)	(N=48)
Age (years)	18.81 (1.77)	19.50 (2.93)
Past 7 day alcohol consumption	27.66 (25.88)	22.29 (17.32)
AUDIT	11.31 (4.93)	12.75 (5.96)
BIS-11 Non-Planning	25.22 (5.07)	27.10 (5.04)
BIS-11 Motor	23.84 (3.59)	25.23 (4.07)
BIS-11 Attentional	17.97 (2.64)	19.31 (2.85)
BIS-11 Total	67.03 (9.40)	71.65 (10.13)
VP Bias (200ms)	-5.38(47.71)	-11.69 (39.61)
VP Bias (2000ms)	1.44 (66.53)	6.51 (42.83)
SRC bias	28.26 (57.54)	29.94 (134.57)
Antisaccade errors	7.94 (9.19)	9.38 (12.79)
Go\No-Go (No-Go errors)	10.41 (7.47)	10.96 (6.49)
COWAT switches	24.87 (8.04)	24.19 (9.42)

Weekly alcohol consumption (UK units), 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol use = combined Z scored weekly alcohol consumption and AUDIT. Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. **p < .01 *p < .05.

Table 3.9: Principle component analysis for measures of inhibitory control and BIS-11 subscales (N=80)

	Rotated components			
	1	2	3	4
Eigenvalues	2.32	1.15	0.85	0.81
Variance (%)	38.70	19.23	14.22	13.47
BIS-11 Attentional	**0.82**	0.21	0.13	0.12
BIS-11 Motor	**0.86**	0.24	-0.10	0.05
BIS-11 Non-planning	**0.84**	0.00	0.07	006
Go/No-Go	0.18	**0.99**	0.05	0.16
Antisaccade	0.00	0.05	**0.99**	0.13
COWAT Switches	0.09	0.15	0.12	**0.99**

Note: Factors highlighted load above 0.572 and are considered significant (Stevens, 2002)
Range of BIS-11 subscale scores are (minimum to maximum); Attentional 8 to 32, Motor 10 to 40, Non-planning 12 to 48. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function

Table 3.10: Regression analysis showing trait impulsivity, inhibitory control measures, and initial orienting of attention (visual probe task reaction time, VP bias) as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.04	.02	$F(1,73) = 2.74$	-0.26	0.26	-.12
Step 2	.17	.14	$F(1,72) = 11.33$	0.26	0.06	.40**
BIS-11 total score						
Step 3						
VP Bias (200 ms)	.18	.14	$F(1,71) = 0.66$	0.01	0.02	.06
Step 4						
Antisaccade errors	.20	.13	$F(3,68) = 0.77$	-0.03	0.09	-.04
Go\No-Go (No-Go errors)				0.05	0.11	-.07
COWAT switches				0.12	0.08	.18
Step 5						
VP Bias (200 ms)XAntisaccade errors	.24	.13	$F(3,65) = 0.96$	0.00	0.00	.17
VP Bias (200 ms)XGo\No-Go (No-Go errors)				-0.01	0.00	-.11
VP Bias (200 ms)XCOWAT switches				0.01	0.00	.07

Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. Cognitive bias measureXInhibitory control/ECF measure = product of normalized variables. *p < .05, **p < .01.

Table 3.11: Regression analysis showing trait impulsivity, inhibitory control measures, and maintenance of attention (visual probe task reaction time, VP bias), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.04	.02	$F(1,73) = 2.74$	-0.27	0.25	-.12
Step 2						
BIS-11 total score	.17	.14	$F(1,72) = 11.30$**	0.18	0.07	.32**
Step 3						
VP Bias (2000 ms)	.19	.16	$F(1,71) = 2.08$	0.01	0.01	.13
Step 4						
Antisaccade errors	.21	.14	$F(3,68) = 0.63$	0.05	0.09	.07
Go\No-Go (No-Go errors)				0.04	0.11	.08
COWAT switches				0.09	0.08	.12
Step 5						
VP Bias (2000 ms)XAntisaccade errors	.23	.13	$F(3,65) = 0.60$	-0.00	0.01	-.06
VP Bias (2000 ms)XGo\No-Go (No-Go errors)				0.01	0.00	.17
VP Bias (2000 ms)XCOWAT switches				0.01	0.00	.02

Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. Cognitive bias measureXInhibitory control/ECF measure = product of normalized variables. *p < .05, **p < .01.

Table 3.12: Regression analysis showing trait impulsivity, inhibitory control measures, and automatic approach responses (SRC bias), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.04	.02	$F(1,72) = 2.80$	-0.42	0.27	-.20
Step 2						
BIS-11 total score	.15	.13	$F(1,71) = 9.87$**	0.18	0.07	.32**
Step 3						
SRC Bias (ms)	.19	.16	$F(1,70) = 3.13$*	0.01	0.01	.14
Step 4						
Antisaccade errors	.20	.13	$F(3,67) = 0.17$	0.04	0.09	.06
Go\No-Go (No-Go errors)				-0.02	0.11	-.02
COWAT switches				0.04	0.08	.06
Step 5						
SRC Bias (ms)XAntisaccade errors	.23	.13	$F(3,64) = 0.93$	0.00	0.00	-.04
SRC Bias (ms)XGo\No-Go (No-Go errors)				-0.01	0.01	.01
SRC Bias (ms)XCOWAT switches				0.00	0.01	-.18

Possible range of total BIS-11 scores is from 30 to 120, with higher values indicating greater impulsivity. SRC bias = Mean reaction time on the avoid alcohol block minus mean reaction time of approach alcohol block, with higher values indicating increased approach bias. Antisaccade (errors) = Number of failures to inhibit saccadic eye movements towards stimuli on antisaccade trials, higher values indicate more failures of inhibitory control. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. Cognitive bias measureXInhibitory control/ECF measure = product of normalized variables. *p < .05, **p < .01.

3.5. Discussion

The first two studies reported in this thesis investigated the contribution of automatic cognitive processing and different measures of behavioural impulsivity to hazardous drinking in undergraduate drinkers. In addition, the specific predictions of dual process models of addiction that the relationships between hazardous drinking and attentional bias and automatic approach responses elicited by alcohol-related cues would be moderated by individual differences in behavioural impulsivity were tested. Finally, as a secondary analysis, the independence of multiple measures of behavioural and self report impulsivity were also investigated.

Summary of findings: Study 1

With regard to automatic alcohol-approach tendencies there was a significant positive correlation between SRC bias scores and AUDIT scores, although SRC bias was not a significant predictor in the regressions after controlling for age and self report impulsivity, suggesting that this is a weak relationship. Attentional bias assessed by the ACT was also not associated with AUDIT scores (at either SOA). There was also no evidence for any associations between delay discounting or time estimation and AUDIT scores (in the correlation or the regression). There was an association between the Two Choice Decision task mean delay and AUDIT scores, but this was in the opposite direction than predicted, and this relationship was not evident in the regression analysis. In addition, there was no evidence to suggest that any of the measures of impulsive decision making moderated the associations between automatic approach responses or attentional bias towards alcohol cues and AUDIT scores. As expected, elevated scores on the BIS-11 were associated with higher AUDIT scores. Finally, the PCA revealed that the behavioural measures of impulsivity were distinct from the self report measures. Significantly, the three measures of impulsive decision making all loaded onto separate factors, suggesting that they did not measure a single underlying impulsive decision making construct.

Summary of findings: Study 2

Study two found that SRC bias scores did predict variance in AUDIT scores, beyond that explained by age and BIS-11 scores (although this relationship did not come out in the full simultaneous regression model) insofar as those participants with greater automatic approach responses towards alcohol-related cues had higher AUDIT scores. In addition, attentional bias towards alcohol-related cues was correlated with AUDIT scores (although this did not come out in the simultaneous regression model). Significantly, it was only attentional bias in the 2000 ms SOA trials that correlated with AUDIT scores. Neither of the specific measures of inhibitory control, or the phonemic fluency task, predicted AUDIT scores or moderated the association between cognitive bias and AUDIT scores. As in study one, elevated scores on the BIS-11 were associated with higher AUDIT scores. Finally, self report impulsivity loaded onto a separate factor from the behavioural measures of impulsivity. Complementing study one, the measures of inhibitory control and phonemic fluency loaded onto separate factors.

Chapter discussion

With regard to attentional bias there were inconsistent results across the two studies. No evidence for an association between attentional bias (initial orientation or delayed disengagement) and AUDIT scores was found when using the ACT (study one). Although there was evidence that maintenance of attention on alcohol-related pictures in the 2000 ms SOA on the visual probe task was associated with AUDIT scores (study two). The discrepancy in the results concerning attentional bias between these two studies may be because the two different measures of attentional bias task have differing sensitivities to attentional bias in social drinkers. Indeed, the evidence base for the ACT in addiction research is considerably more limited (Franken et al., 2000a; Stormark et al., 1997) than that for the visual probe task (see Field & Cox 2008). One explanation for this may be that the attentional cueing effect tested in the ACT is large regardless of the stimuli type, i.e. there is an orientation of attention towards stimuli appearing in the periphery of the visual field regardless of their content. This cueing effect may result in the specific conditioned attentional

bias effects being essentially 'washed out'. In addition, the ACT has been criticised as it may reflect a general slowing of responses towards emotionally valenced stimuli (Mogg et al., 2008). Due to the null findings and these theoretical and procedural concerns relating to the ACT the second study utilised the visual probe task. Significantly, attentional bias as measured by the visual probe task was associated with AUDIT scores, although this association was only in the 2000 ms SOA condition. This supports previous research (e.g. Field et al., 2004b; Miller & Fillmore 2010; Townshend & Duka 2001) which suggest that alcohol use is associated with maintenance of attention on alcohol-related cues in non-dependent social drinkers, with no association between attentional bias in the short SOA conditions (assumed to represent the initial orientation of attention) and alcohol use. The results regarding automatic alcohol-approach tendencies were also generally consistent with the hypotheses. Although no association between automatic approach responses towards alcohol-related cues and AUDIT scores was revealed in the regression models in study one, SRC bias scores did correlate with AUDIT scores in the predicted direction. In study two SRC bias scores did predict a significant amount of variance in AUDIT scores beyond that explained by age and BIS-11 scores. The lack of any association between SRC bias and AUDIT scores in the regression model in study one may be the result of participants in that study drinking less and having lower AUDIT scores than they did in study two. Overall, the significant correlations between SRC bias scores and drinking behaviour in these studies is a replication of findings from a previous study in which an identical SRC task was used (Field et al., 2008), and it is also consistent with similar findings obtained from related tasks, all of which measure the strength of associations between the concepts of 'alcohol' and 'approach' (Ostafin et al., 2008; Ostafin & Palfai 2006; Ostafin et al., 2003; Wiers et al., 2011; Wiers et al., 2009).

These findings, along with those from the visual probe task, suggest that alcohol-related cues acquire incentive-motivational properties among hazardous drinkers, and as such they provide support for incentive-motivational theories of addiction and alcohol abuse (e.g. Robinson & Berridge 2001). Approach tendencies and attentional biases elicited by alcohol-related cues may become automatised in heavy drinkers, perhaps contributing to loss of control over alcohol consumption. However, only a

relatively small amount of variance in hazardous drinking was predicted by measures of automatic processing of alcohol-related cues (indeed, none of these measures significantly predicted AUDIT scores in the simultaneous regression models). This finding is consistent with a recent meta-analysis into the contribution of automatic alcohol associations to alcohol consumption (Rooke et al., 2008). This analysis found that, although automatic-alcohol associations predicted alcohol use, only a moderate amount of variance was explained by these measures.

Studies one and two also investigated the hypothesis that there is a direct association between alcohol consumption and measures of behavioural impulsivity (e.g. de Wit 2009). As discussed in the introduction, several studies suggest that alcoholics tend to be more impulsive than non-alcoholic controls (Bobova et al., 2009; Goudriaan et al., 2006), and that within non-dependent drinkers, heavier drinkers are more impulsive than lighter drinkers (Colder & O'Connor 2002; Field et al., 2007a; Vuchinich & Simpson 1998). Neither of the experiments was able to replicate these findings. The findings regarding the Two Choice Delay task and Time Estimation task are not surprising as the only previous research to use these tasks in relation to alcohol use found that they did not discriminate binge drinkers from controls (Rose & Grunsell 2008). In addition, the Time Estimation task may have been a weak measure of internal clock speed due to the short time period estimated. The short time period resulted in little variance in accuracy of estimates and also allowed for the utilisation of basic strategies such as simply counting the passage of one minute. It may have been beneficial to utilise a temporal generalisation task (e.g. Wearden et al., 1998) or a temporal estimation task (e.g. Coelho et al., 2004). Likewise, the antisaccade and phonemic fluency tasks have not been utilised in the prediction of alcohol use indices in cross section designs. The null findings regarding delay discounting and the Go/No-Go are more problematic. However, as stated in the introduction there are also several null findings in the literature. For example, both Kirby and Petry (2004) and MacKillop et al. (2007) failed to detect associations between alcohol consumption or alcohol problems and delay discounting, and Kamarajan et al. (2005) did not find any evidence for a selective impairment in response inhibition in alcoholics compared to controls. One explanation for this is suggested by the MacKillop et al. (2011) meta-analysis in which effect sizes for

delay discounting were found to be much larger in drug dependent populations than non-dependent populations. The student samples investigated in the current chapter may not exhibit increased impulsive decision making or impairments in inhibitory control because of a relative lack of experience with alcohol. Alternatively, the null findings may be due to the other factors associated with the samples recruited. Undergraduates have been shown to be motivated to drink by social factors (e.g. Faulkner et al., 2006; Wicki et al., 2010); therefore, undergraduate alcohol consumption may be associated with social drinking and conformity to drinking norms rather than with individual differences in impulsivity. There was, however, the expected association between self-reported impulsivity and hazardous drinking, replicating previous research (e.g Fernie et al., 2010; MacKillop et al., 2007). This suggests that such self report measures may be valuable tools in assessing undergraduate alcohol-risk behaviours. Clearly, further research is required to clarify the nature of any direct relationship between behavioural impulsivity and hazardous alcohol consumption or abuse.

With regard to the secondary analysis of different measures of impulsivity, it was found in both studies that the BIS-11 subscales loaded onto a single factor (self report impulsivity). However measures of impulsive decision making (delay discounting, Two-Choice Delay task, and Time Estimation; study one) and inhibitory control (Go/No-Go, antisaccade, COWAT; study two) all loaded onto separate factors. This finding in regard to the tasks of 'impulsive decision making' is not surprising. The delay discounting task is the only measure of the three that is a well-validated measure of decision making; the other two methods are less widely used (especially within the addiction literature). Indeed, the Two Choice Decision task was widely criticized by the participants for being too boring and making little intuitive sense, insofar as they could see no reason to wait to receive points that are of no intrinsic or extrinsic value. It is therefore possible that this is simply a measure of boredom susceptibility. Likewise, time estimation can be seen as a separate construct from the other two measures and has been suggested to reflect sensation seeking rather than impulsive decision making (Glicksohn et al., 2006). The findings regarding inhibitory control are more unexpected. It would be expected that the Go/No-Go task and the antisaccade task would load onto a single factor. One

explanation for this is that the passive-avoidance format of the Go/No-Go task involves some aspects of working memory (remembering four digits) and the antisaccade is a purer measure of inhibitory control. Alternatively, the difference between these two measures may be due to the fact one of the measures involves inhibiting a learnt response compared to a reflex response. The COWAT, although involving inhibitory control, also involves switching and working memory so the finding that it was distinct from the antisaccade and Go/No-Go tasks is not entirely unexpected. Indeed, the recent principle component analysis by Verdejo-Garcia and Perez-Garcia (2007) revealed this task loads onto an updating factor rather than inhibitory control; this along with the current results suggests that inhibitory control contributes little to performance of this task and it is possible that this task relies most on updating working memory (e.g. Lee et al 1999). Overall, these findings do suggest that although tasks ostensibly are referred to as inhibitory control or impulsive decision making tasks the specific psychological constructs that contribute to performance on them may be subtly different.

The primary hypothesis in the present study was that the contribution of automatic responses elicited by alcohol-related cues to AUDIT scores would be moderated by individual differences in impulsivity. This hypothesis was derived from recent dual process models of addiction (e.g. Goldstein & Volkow 2002; Jentsch & Taylor 1999; Wiers et al., 2007), all of which suggest that the relationship between the incentive-motivational properties of alcohol cues and actual alcohol consumption should be large in highly impulsive individuals, but reduced or non-existent in individuals with low levels of impulsivity. Therefore, it was predicted that the contribution of alcohol cue-elicited approach tendencies and attentional bias to hazardous drinking would be largest in highly impulsive individuals. There was no evidence that any of the measures of impulsive decision making or inhibitory control moderated the relationship between attentional bias or automatic-approach tendencies towards alcohol cues and AUDIT scores. It is important to note that recent studies that have provided some support for dual process models of addiction have focussed on different types of automatic alcohol cognitions and different aspects of controlled processing. Grenard et al. (2008) and Thush et al. (2008) both assessed whether the impact of automatic cognitive processes would be moderated by individual

differences in working memory. This may suggest that the theoretically important aspect of executive cognitive function is working memory, rather than impulsive decision-making or response inhibition, although Houben and Wiers (2009b) did find the relationship between automatic alcohol-related cognition and alcohol consumption was moderated by response inhibition, as assessed with a colour-conflict Stroop task. In addition to the differing methodologies regarding the assessment of controlled processes these studies employed different methodologies to assess automatic alcohol cognitions, an alcohol-arousal IAT (Houben & Wiers 2009b; Thush et al., 2008), and word association tasks (Grenard et al., 2008). It is possible that these tasks may have differing sensitivity to the moderating effect of aspects of executive cognitive functioning than attentional bias and automatic approach response tasks that were utilised in studies one and two. Indeed van Hemel-Ruiter et al. (2011) failed to find evidence that working memory moderated the impact of automatic approach responses on alcohol consumption. A further consideration is the sample that the studies have utilised. Both Grenard et al. (2008) and Thush et al. (2008) sampled 'at risk' adolescents , who may not have fully developed executive cognitive functioning (Rubia et al., 2000). However, it is notable that Houben and Wiers (2009b) did find evidence for dual process models of addiction in a young adult sample.

3.6. Chapter summary

The current chapter offers some support for incentive-motivational models of addiction insofar as attentional bias was correlated with AUDIT scores (study two), and that there were correlations between automatic approach tendencies towards alcohol-related cues and AUDIT scores in both studies. There was, however, no direct association between any measure of behavioural impulsivity and AUDIT scores. Significantly, there was also no evidence for the specific hypotheses of dual process models of addiction, that measures of behavioural impulsivity would moderate the impact of automatic processes on alcohol use. Finally, principle component analyses revealed that there is a considerable amount of heterogeneity

between tasks that are assumed to assess 'impulsive decision making' and 'inhibitory control'.

Chapter 4

The role of impulsive decision making, inhibitory control and automatic responses to alcohol-related cues in drinking among adolescents.

4.1. Abstract

Hazardous drinking in adolescents has been found to be associated with both increased impulsivity and the automatic processing of alcohol-related cues. Furthermore, emerging evidence suggests that the relationships between adolescent alcohol consumption and automatic alcohol cognitions may be moderated by individual differences in impulsivity. The current study aimed to investigate the contribution of impulsivity and automatic cognitive processing to hazardous drinking, and to examine if the relationship between hazardous drinking and automatic cognitive processing would be moderated by individual differences in impulsivity. 256 participants (135 female) aged between 15 and 16 years completed questionnaire measures of alcohol consumption and hazardous drinking. Participants also completed computerised measures of automatic cognition; alcohol approach tendencies (stimulus-response compatibility (SRC) task) and attentional bias (visual probe task), and two behavioural measures of impulsivity (Stop-Signal and delay discounting tasks). The results indicate that attentional bias was directly associated with adolescent alcohol consumption, but automatic approach tendencies and measures of impulsivity were not. As predicted, the association between attentional bias on hazardous drinking was moderated by individual differences in impulsive decision making inasmuch as the association between attentional bias and hazardous drinking was larger in more impulsive adolescents, when impulsivity was assessed with the delay discounting task.

4.2 Introduction

Adolescence is associated with risk-taking behaviour, for example drug and alcohol experimentation, risky sexual behaviour and dangerous driving have all been shown to peak in adolescence (for reviews see; Arnett 1992; Spear 2000). Engaging in risky behaviour such as heavy drinking has also been found to affect aspects of adolescent life and future health. For example, heavy drinking in adolescence is associated with poor school performance (Balsa et al., 2011), crime (Hibell et al., 2007) and alcohol use disorders in adulthood (Grant & Dawson 1997). It has been argued that adolescence represents a critical period for the development of hazardous drinking patterns due to the significant changes in the neurological substrates of appetitive motivational processes, as well as behavioural control (Dayan et al., 2010; Gladwin et al., 2011). Theories of adolescent brain development (e.g. Steinberg 2008) propose that adolescence is associated with rapid maturation of brain areas involved in appetitive motivation such as the mesolimbic dopamine system (e.g. Galvan et al., 2006); while areas such as the prefrontal cortex, which are implicated in behavioural control, develop later in adolescence (e.g. Luna & Sweeney 2004; Crews et al., 2007). It is argued that it is the different developmental time course of these systems that is one of the fundamental factors predisposing adolescents to engage in risky behaviour.

Incentive-motivational theories of addiction (e.g. Robinson & Berridge 1993) state that cognitive bias towards drug-related stimuli develops slowly as the result of repeated drug use. However, the rapid development of brain regions associated with motivational orientation in adolescence has been postulated to increase behavioural approach towards any rewarding stimuli in the environment (Somerville et al., 2011), therefore a sufficient conditioning history would not be required for biased processing of appetitive cues such as alcohol. It is therefore possible that this general increase in appetitive motivation may be reflected in tasks which measure the biased processing of alcohol-related stimuli. Indeed, it has been demonstrated using cross-modal priming tasks that children/adolescents (mean age 11.8 years) have stronger alcohol-positive than alcohol-neutral associations even before

initiation of drinking, with a similar pattern also exhibited for cigarettes (O'Connor et al., 2007). This suggests that cognitive bias towards alcohol-related stimuli is exhibited before any specific sensitisation of motivational systems. Furthermore, cognitive bias in adolescents has been found to be associated with parental alcohol use, with particularly strong cognitive bias in the children of alcoholics (e.g. Zetteler et al., 2006). This indicates that rather than being solely the result of a sensitisation of motivational processes (specific or general sensitisation), cognitive bias in adolescents may be also influenced by social learning (Pieters et al., 2011; Zucker et al., 1995).

Although the vast majority of research into the automatic processing of alcohol-related cues has focussed on adult (usually undergraduate) drinkers, there is evidence that adolescents show cognitive bias towards alcohol-related stimuli. For example, both Ames et al. (2007) and Stacy et al. (1996) found that performance on word association tests (i.e. more drug-related responses given to ambiguous cue words) was associated with alcohol and cannabis use in adolescents. More specifically, in a sample of adolescent drinkers Thush and Wiers (2007) found that automatic alcohol-positive and alcohol-arousal associations were positively associated with alcohol use at one-year follow up, and that alcohol-negative associations were associated with decreased alcohol use at follow up. Automatic alcohol-arousal associations have also been shown to predict alcohol use in children as young as 11 to 13 years old (Pieters et al., 2010). With regard to attentional bias, Field et al. (2007a) found that heavy drinking adolescents (aged 16-18 years old) showed increased interference in comparison to their light drinking counterparts on an alcohol Stroop task. There have however been notable failures to replicate this association between cognitive bias and alcohol use. For example, Zetteler et al. (2006) found a negative correlation between Stroop interferences and alcohol use in adolescents with alcohol-dependent parents (who are defined as 'at risk'), and no association between Stroop interference and alcohol use in control participants. Although there is some evidence for increased attentional bias in adolescents the only study to date that has investigated automatic alcohol-approach associations found that heavy drinking adolescents were actually faster to avoid alcohol-related cues than controls using an AAT (van Hemel-Ruiter et al., 2011). Taken together, these studies indicate that although there is some

evidence for biased processing of alcohol-related cues in adolescents (particularly in 'at risk' participants), studies directly linking this to alcohol use have so far been equivocal.

The rapid development of motivational brain systems in early adolescence is not matched by a development in frontal regions associated with behavioural control, which develop in later adolescence (e.g. Christakou et al., 2011 for a review see Olson & Luciana 2008). Indeed, behavioural impulsivity has been repeatedly demonstrated to be elevated in adolescents, with increased rates of discounting of future rewards as well as poor inhibitory control compared to adults (Olson et al., 2007; Rubia et al., 2006; Whelan & McHugh 2009). There is also evidence these different aspects of behavioural impulsivity follow different developmental trajectories inasmuch as a reduction in impulsive decision making develops later in adolescence compared to improvements in response inhibition (and other measures of executive cognitive functioning such as working memory; Prencipe et al., 2011). Studies have also investigated whether behavioural impulsivity is elevated in samples that are defined as being at risk of developing alcohol related problems. For example, Nigg et al. (2004) investigated which measures of behavioural impulsivity and executive cognitive function were elevated in children and adolescents (aged between 3-14 years old) who were defined as being at risk. Significantly, at risk children and adolescents had poorer response inhibition and delay gratification than control participants, although they did not differ on other measures of executive functioning. This suggests that both aspects of behavioural impulsivity may be specific factors that predispose individuals to the risk of developing hazardous patterns of alcohol use.

As well as predicting at risk status, both these aspects of behavioural impulsivity have been shown to be associated with alcohol (and other drug) use in adolescents. With regard to impulsive decision making, steeper discounting of future rewards has been repeatedly demonstrated in adolescent smokers (Fields et al., 2009a; Fields et al., 2011) and has also been found to be associated with poor treatment outcomes in adolescent smokers (Krishnan-Sarin et al,. 2007). It is however worth noting that, like in the adult literature, there have been notable failures to replicate the association between delay discounting and smoking in adolescent samples (e.g.

Melanko et al., 2009; Reynolds et al., 2003). With regard to alcohol consumption, the only study to date to specifically assess the association between heavy drinking and delay discounting in adolescents found heavy drinkers discounted rewards more steeply than their light drinking counterparts (Field et al., 2007a). Retrospective studies have also shown that elevated delay discounting in undergraduates is associated with early onset of heavy drinking (Kollins 2003). Generally, these studies indicate that drug-related risk behaviours may be associated with elevated delay discounting, although specific evidence for alcohol use is limited, with only Field et al. (2007a) demonstrating a clear association between delay discounting and heavy drinking.

Much of the research into the role of behavioural impulsivity in adolescent drug and alcohol use has focused on impairments in inhibitory control as a risk factor for developing hazardous patterns of alcohol and drug use. For example, inhibitory control at aged 16 has been found to prospectively predict substance abuse disorders as well as variation in drug use at age 19 (Tarter et al., 2003). In addition to predicting drug use, Wong et al. (2006) found that slower development of behavioural control was associated with increased alcohol use in childhood and adolescence. Although these studies suggest that inhibitory control may play a significant role in the development of hazardous alcohol use in adolescents and subsequent alcohol abuse problems in later life there are some important caveats to be considered. Firstly, although Nigg et al. (2006) found that poor response inhibition in childhood and early adolescence predicted alcohol use in mid to late adolescence (15-17), the amount of variance explained by inhibitory control deficits was relatively small in the sample as a whole (approximately 1%). In at risk participants, i.e. those with alcoholic parents, impairments in inhibitory control prospectively predicted significantly more variance in alcohol consumption (approximately 9%). Secondly, other studies (e.g. Harden & Pihl 1995) have found other aspects of executive functioning such as mental set shifting rather than inhibitory control, are impaired in at risk children compared to controls. Finally, there have also been notable failures to demonstrate an association between poor inhibitory control and risk behaviours such as smoking in adolescents (Galván et al., 2011; Reynolds et al., 2007). As with the evidence implicating delay discounting in

increased adolescent alcohol use, studies investigating the contribution inhibitory control to adolescent alcohol use are equivocal. It is possible that it is only in at risk adolescents that inhibitory control contributes significantly to hazardous patterns of alcohol use.

Although the evidence for the direct association between measures of behavioural impulsivity and alcohol use in adolescents is inconsistent, there is some evidence for the specific predictions of dual process models of addiction that the impact of automatic cognitive processes on drinking behaviour will be moderated by behavioural impulsivity/executive cognitive functioning in adolescents. Firstly, in a sample with a mean age of 16.34 (±1.34) Thush et al. (2008) found that automatic alcohol–active associations (assessed with an IAT) predicted alcohol use at one month follow-up in participants with poor working memory, whereas explicit alcohol outcome expectancies predicted drinking at follow up in participants with good working memory. Secondly, Grenard et al. (2008) assessed automatic alcohol and cigarette memory associations (using word association tests) and working memory in a sample of high school students (mean age 16.71 ±0.74). Performance on word association tests (giving drug-related responses to ambiguous cues) was directly associated with alcohol but not cigarette consumption, and working memory capacity was not directly associated with either alcohol or cigarette use. Significantly, the interaction between working memory capacity and word association test performance predicted alcohol and cigarette use, inasmuch that performance on the word association task predicted more variance in participants with poor working memory capacity. However, a different patterns of findings were found by van Hemel-Ruiter et al. (2011), who reported that in a sample aged 15.09 (±0.97), explicit attitudes, but not automatic approach responses to alcohol-related cues, predicted more variance in adolescent drinking in participants with poor working memory capacity than those with good working memory capacity.

Rather than assessing behavioural impulsivity, the aforementioned studies have measured working memory capacity (using the self ordered pointing task) as a hypothesised moderator of the impact of automatic cognitive processes (which were assessed with forms of word association tests in Grenard et al., 2008; Thush et al., 2008) on alcohol consumption. Furthermore, it is notable that the samples in these

studies may, to a greater or lesser degree, represent at risk populations. Those used by Grenard et al. (2008) were in continuation high schools (and explicitly described as at risk), and participants in Thush et al. (2008) and van Hemel-Ruiter et al. (2011) were taken from second tier Dutch secondary schools which have the highest prevalence of behaviour problems and substance use in the Dutch educational system.

The current study aimed to investigate the contribution of automatic processing of alcohol-related cues and behavioral impulsivity to hazardous alcohol use (and alcohol use frequency and volume) in adolescents. As in previous studies, aspects of cognitive bias that have been shown to have a causal relationship with drinking (attentional bias and automatic alcohol-approach responses), were assessed. A delay discounting task was used to measure impulsive decision making. Although study two of this thesis used a Go/No-Go task to measure inhibitory control, this study used a simple Stop-Signal task that has been validated for use in adolescents and children (see Bitsakou et al., 2008). As well as investigating the contribution of these cognitive processes to hazardous alcohol consumption in isolation, the specific predictions of dual process models of cognition, that the impact of automatic cognitive processes on alcohol use will be moderated by behavioural impulsivity, were also investigated. The adolescent sample (aged between 15 and 16) was taken from a broad range of schools in the Merseyside area. Therefore, unlike previous studies that have investigated dual process models of addiction in adolescents, the sample will not be taken from an at risk population. It is hypothesized that cognitive bias and behavioral impulsivity will be associated with increased alcohol use and hazardous drinking, with the impact of the former measures being moderated by the latter.

4.3. Method

Participants

256 participants (135 female) aged between 15 and 16 years (mean 15.31 ±0.32) participated in the experiment. The cross sectional data set reported here was taken from a larger longitudinal study in which changes in cognitive bias and behavioural impulsivity were measured at six month intervals over a two year period to investigate their contribution to patterns of adolescent drinking. The current data set was taken from the last wave of testing (wave five). Participants were recruited from five state schools in the Merseyside area; schools were selected to give a broad socio-demographic range of participants. Participants were excluded if their vision was not normal or corrected-to-normal. All participants provided informed consent, and as all participants were minors opt-out consent was given to parents in accordance with school policy. The study was approved by the University of Liverpool Ethics committee.

Design

A between subjects design was used to investigate whether non drinkers/infrequent drinkers differed from regular drinking adolescents on measures of cognition and socio-economic status. Furthermore, the experiment also had correlational design to investigate whether measures of automatic cognitive processing and behavioural impulsivity predicted variance in hazardous drinking (hazardous drinking index) and non-hazardous drinking behaviour (alcohol use index) in participants who had consumed an alcoholic drink. In addition to looking at these factors in isolation the interaction between measures of automatic cognition and behavioural impulsivity in the prediction of hazardous drinking was also investigated. Due to the large sample size all both measures of automatic cognition were assessed in the same regression models.

Materials

Pictorial stimuli

The SRC task and the visual probe used a picture set containing 14 alcohol-related pictures and 14 (matched) alcohol-unrelated pictures. These pictures differed from those used in studies one and two. A different picture set was utilised to reflect adolescent drinking behaviours and drink preference, for example instead of pictures of wine in wine glasses, pictures of alco-pops and cider were used. The control pictures were matched to the alcohol pictures on perceptual characteristics but did not contain any alcohol-related cues; instead they depicted stationery (e.g. someone holding a pen up to their mouth). All the pictures were 100mm high X 125mm wide.

Questionnaires

Family affluence scale (Boyce et al., 2006). This scale was used to assess the socio economic status of the adolescents. This questionnaire comprised of three items: number of holidays in the past year (no holiday = 1, one holiday = 2, two holidays = 3, three of more = 4), family car ownership (no car = 1, one car = 2, two or more = 3), and whether they have their own bedroom (shared room = 1, own room = 2). Responses for the three questions were added together to give an overall measure of socio-economic status, higher scores are indicative of greater familial wealth. This scale was developed for the WHO-Health Behaviour in School-aged Children (HBSC) survey, which assessed health in every European country. Boyce et al. (2006) found that when comparing mean country scores to GDP the kappa coefficients were in excess of .57, with mean country scores correlating with GDP which suggests this scale has good criterion validity. Inter item correlations for the family affluence scale indicate adequate internal validity, although it is worth noting that formative indexes do not necessarily need to have high inter-item correlations, as the construct measured (in this case family affluence) is a product of the factors chosen to assess it.

Modified Time Line Follow Back (TLFB based upon Sobell & Sobell 1990). The modified TLFB self report questionnaire was used to assess fortnightly alcohol consumption. Participants were asked to state on which days in the previous two weeks they had consumed alcohol, what type of alcohol, what brand, and what volume they had consumed. The experimenter used this to estimate the number of alcohol units consumed over the preceding fourteen days. In addition to this participants were asked to state how often they drank alcohol (almost every day, twice a week, once a week, once a fortnight, a few times a month, a few times a year, never), estimate the number of times that they had been drunk in the previous six months, and state how old they were when they had their first full alcoholic drink.

Alcohol problem index (Magar et al., 2008). The alcohol problem index was calculated as a measure of hazardous drinking in adolescents. This measure was used to assess problems as a result of alcohol consumption, participants were asked to state whether any of the following negative events had resulted after drinking: 'I got into an argument', 'I got into a fight', I had to be taken to hospital', 'I damaged my clothes or other items', 'I lost money or other items', 'I got into trouble with the police'. An overall alcohol problem index score was derived by adding answers to these questions to give a score from zero to six. These measures were found to have an acceptable Cronbach's alpha ($\alpha = 0.69$).

Cognitive bias tasks

Stimulus Response Compatibility (SRC) task. The SRC task was identical to that used in experiment two except for the pictorial stimuli used (see materials).

Visual probe task.This task was programmed in Inquisit version 1.33 (Millisecond software, 2002). Each trial of the visual probe task commences with a central white fixation cross presented on a black background for 500 ms. Immediately after this a pair of pictures was presented for 500 ms, one picture to the left of the fixation the other to the right, 60mm apart. Alcohol and neutral cues were presented for 500 ms. Immediately after picture offset, a probe (a white arrow on a black background, pointing up or down) appeared in one of the picture locations. Participants had to

respond to the orientation of the probe by pressing up or down on arrows on a laptop keyboard. There was an intertrial interval of 500 ms.

The visual probe task consisted of 68 trials in total. Participants first completed 10 practice trials in which neutral picture pairs were presented, following which instructions were re-iterated before they completed the main task. The main task consisted of 2 buffer trials (of neutral picture pairs) followed by 56 critical trails. Each of the 14 picture pairs appeared four times with the alcohol and control pictures appeared twice on the right side of the screen and twice on the left with probes replacing alcohol and control pictures an even number of times. Trials were presented in a new random order for each participant. Reaction time (the time taken to react to the orientation of the probe) was measured on each trial and the dependent variables were mean reaction time to congruent probes (those that appeared in the same position of the alcohol picture) compared to incongruent probes (those that appeared in the same position of the neutral picture). As before a reaction time bias score was derived from subtracting congruent probe reaction times from incongruent probe reaction times.

Impulsivity measures

Stop-Signal task (Bitsakou et al., 2008). This task was used to assess inhibitory control and was programmed in Visual basic 6.0. Each trial commenced with a central fixation cross (+) appearing in the centre of the screen for 500 ms followed by one of two visual cues. The cues were a large upper case "O" and a large upper-case "X" appearing for 1000 ms. Participants were instructed to respond to these cues by pressing the correspondingly labeled key on the keyboard. Participants were instructed to respond to the appearance of either cue on the screen by pressing the corresponding key as fast as possible, however, when an auditory tone (lasting 500 ms) was presented through the headphones they were told to inhibit their response. They were instructed that sometimes they would not be able to stop when they heard the tone and they should not wait to hear if the auditory tone was presented before pressing the key. There was an intertrial interval of 500 ms.

The Stop-Signal task consisted of 192 trials split into six blocks of 32 trials. The first block of the task was a practice block in which participants were informed if they made a commission error (responding after the presentation of the tone), with the message "Wrong! You pressed a key". The two visual cues appeared an even number of times in each block and in 25% of the trials the stop tone was presented. The latency of the first stop signal in each block was 250 ms which adjusted according to performance. If participants failed to inhibit their response (made a commission error) then 50 ms was taken off the stop signal delay (e.g. if the participant failed to inhibit their button press to the first auditory tone then the next stop signal delay would be 200 ms). Successful inhibition resulted in 50 ms added to the stop signal delay (e.g. successful inhibition of response to the first auditory tone resulted in the subsequent stop signal delay being 300 ms). Stop signal reaction time (SSRT; Band et al., 2003) was calculated by subtracting the mean stop signal latency from each block from the mean go latency (on go trials only) on each block. A mean task SSRT was then derived from the block SSRT's. The overall SSRT is an estimate of the mean time required for participants to inhibit their go responses, higher scores are indicative of poorer inhibitory control.

Delay discounting (Du et al., 2002). A computerised delay discounting task (programmed in Visual Basic 6.0) was used to assess impulsive decision making in response to monetary rewards. The delay discounting methodology was identical to the one used by Fernie et al. (2010). Participants were presented with the hypothetical choice of receiving £100 at a future date or receiving a smaller amount immediately. The size of the immediate reward was adjusted by either adding 50% of the last adjustment (if the delayed reward was selected) or subtracting 50% of the last adjustment (if the immediate reward was selected). This decreasing adjustment logarithm was used by Du et al. (2002). Participants made six choices for each delay period. Monetary choices were made for delays of one day, one week, two weeks, one month and six months. Indifference points for each of the seven delays were analysed by computing area under the curve (AUC) values (Myerson et al., 2001). Lower values of AUC indicate steeper delay discounting, or increased impulsive decision-making. For consistency with the other behavioural measures the AUC values were reversed so that higher values represent steeper discounting.

Procedure

Participants were tested in groups ranging from four to eight in size with at least one experimenter for every four participants. Participants provided informed consent before completing the questionnaire measures described above. Participants then completed a battery of cognitive tasks which included the SRC task, visual probe task, Stop-Signal task, and the delay discounting task. Participants also completed the Balloon analogue risk task (BART; Lejuez et al., 2007), the results of which are not reported here. Task order was counterbalanced across testing groups, with all participants within each testing group completing the tasks in the same order. The entire testing session lasted approximately 45 minutes. At the end of the experiment participants were thoroughly debriefed before receiving a £5 HMV voucher as compensation for their time.

Data analysis

The data analysis strategy for this study is generally consistent with the other cross sectional studies (as described in chapter two). In addition, because not all participants were regular alcohol consumers, differences between participants who reported being non-drinkers or infrequent drinkers and regular drinkers on all dependent variables were also investigated using a MANOVA. The dependent variables used in the regression analyses are however different than that in the previous two studies. The alcohol problem index was used as a validated measure of hazardous drinking in adolescents as one dependent variable. In another regression analysis an alcohol use index (AUI) was calculated to use as the dependent variable. This measure was calculated by converting fortnightly alcohol consumption, number of days drinking in the last fortnight, times drunk in the previous six months, length of time regularly drinking, and age of first drink into Z scores before adding them together. This measure was used so as to get an alcohol use variable that captured as

many aspects of adolescent drinking as possible as well as reducing the impact of inaccurate responding on the two week TLFB.

In this study neither the alcohol problem index or the alcohol use index were normally distributed (Kolmogorov – Smirnov test ps<.05). Therefore the data was log transformed so it met parametric assumptions (Kolmogorov – Smirnov test ps>.05).

4.4. Results

Sample Characteristics

Participants consumed a mean of 7.37 (±14.59; range 0-38.6) UK units of alcohol over the fourteen day period before testing (1 UK unit = 8g alcohol). Out of the full sample of 268 participants, 39 reported that they had never consumed an alcoholic drink. The mean number of UK units consumed in the fourteen day period before testing by the 'drinkers' was 8.62 (±14.44). For descriptive statistics and correlations between all questionnaire measures and cognitive tasks see Table 4.1.

Gender differences in economic status, alcohol use indices' and all cognitive tasks

Univariate ANOVAs revealed that males only significantly differed from females on problem index scores ($F(1,216) = 3.90$, $p = .048$, $\eta_p^2 = .02$). There was also a trend towards male adolescents having greater SSRT in the Stop-Signal task indicating that they had poorer inhibitory control compared to their female counterparts ($F(1,216) = 3.52$, $p = .062$, $\eta_p^2 = .02$) for descriptive statistics see Table 4.2.

Drinker status differences for all for all cognitive tasks

Participants were split into two groups of 'drinkers', those who have never consumed a whole alcoholic drink or who drank less than monthly were in the

abstainer-infrequent adolescent drinker category (N=142). Participants who drank monthly or more frequently were in the regular adolescent drinker category (N= 125). Univariate between subjects ANOVAs revealed a significant difference between groups in their attentional bias towards alcohol-related cues ($F(1,254)$ = 11.54, p = .001, η_p^2 = .04). Planned comparisons revealed that this was due to significantly greater attentional bias towards alcohol cues in the regular adolescent drinkers (17.30 ±57.63) compared to the abstainer-infrequent drinkers (-2.69 ±30.62; $t(259)$= -3.55, p < .001; see Figure 4.1). There was however no difference between regular drinking and abstaining-light drinking adolescents in SRC bias scores ($F(1,254)$ = 0.29, p >.1, η_p^2 = .00). There was also no difference between groups in either delay discounting AUC values ($F(1,254)$ = 0.06, p > .1, η_p^2 = .000) or SSRT, ($F(1,254)$ = 1.61, p >.1, η_p^2 = .006). Finally, there was no difference between groups in socio-economic status ($F(1,254)$ = 0.12, p > .1, η_p^2 = .00) for descriptive statistics see Table 4.3.

Predicting adolescent alcohol use and alcohol-related problems

Hierarchical regression analyses were conducted to investigate the contribution of gender, (age was excluded due to the sample being from the same school year), socio-economic status, cognitive bias, impulsivity and interactions between the latter two clusters of variables to adolescent alcohol use. Participants who had never consumed alcohol (N = 39) were excluded from these regression analysis giving a sample size of 229 (before outlier removal). Two regression analyses were conducted. The dependent variable in the first regression analysis was the alcohol use index (AUI), and the second analysis investigated which factors were related to problematic alcohol consumption as measured by the problem use index. Both these dependent variables were not normally distributed (in the one sample Kolomgorov-Smirov test both ps < .001, and the skewness statistic was twice the SEM for both variables). These variables were therefore log transformed so that they met parametric assumptions and were suitable dependent variables for the regression analyses.

The contribution of automatic cognitive processes and behavioural impulsivity to the alcohol use index (Table 4.4)

The full regression model predicted only 2% of the variance in AUI scores (R^2 = .06, R^2 adjusted = .02, $F(10, 207) = 1.33$, $p > .1$). There was no association between gender and AUI scores (β = -.06, $p > .1$), there was also no association between economic status and AUI scores (β = -.06, $p > .1$). The measures of cognitive bias predicted approximately 3% of variance in AUI scores after controlling for gender and economic status (R^2 change = 0.04, $F(2, 213) = 4.29$, p= .015). This effect was carried by the visual probe reaction time bias as this variable was found to be a significant predictor of the AUI scores (β=.19, p = .01), with greater attentional bias towards alcohol cues being associated with higher AUI scores. SRC bias scores were not associated with AUI scores (β=-.05, $p > .1$). There was no evidence of a direct association between AUC scores from the delay discounting task or SSRT from the Stop-Signal task and AUI scores (ps > .1). Finally, the interactions between automatic cognitive processes and impulsivity measures did not explain additional variance in AUI scores (ps > .1).

The contribution of automatic cognitive processes and behavioural impulsivity to the alcohol problem index (Table 4.5)

The full regression model predicted 6% of the variance in problem index scores (R^2 = .01, R^2 adjusted = .06, $F(10,210) = 2.36$, p = .011; Table 4.5). Gender was found to be a significant predictor of problem index scores (β=-.15, p = .031), with females having lower scores than males. Again, there was no association between economic status and problem index scores. Unlike the prediction of AUI scores, the cognitive bias variables did not significantly predict variance after controlling for gender and economic status (R^2 change = .01, $F(2, 216) = 1.65$, $p > .1$). There was a strong trend towards attentional bias predicting problem index scores (β=.14, p = .058), with greater attentional bias towards alcohol cues being associated with higher problem index scores. As before SRC bias sores failed to predict any variance in problem index scores (β=.02, $p > .1$). There was no evidence of a direct association between AUC scores from the delay discounting task or SSRT from the Stop-Signal task and

problem index scores (ps > .1). The interactions between cognitive bias and impulsivity were found to predict additional variance in problem index scores after controlling for gender, economic status, cognitive bias and impulsivity (R^2 change = .05, $F(4,210) = 2.71$, p = .031). Participants with greater attentional bias and greater AUC scores (elevated discounting of future rewards) had significantly higher problem index scores (β=.15, p = .029). There was no relationship between problem index scores and the interactions between attentional bias and SSRT, or SRC bias AUC scores or SSRT (all ps <.1).

Table 4.1: Descriptive statistics and inter-correlations for economic status, indices of alcohol consumption and experimental tasks

	Mean (±SD)	2	3	4	5	6	7	8	9	10	11
1. Economic status	3.68 (1.49)	-.02	-.02	-.02	.04	-.04	.12*	.10*	.07	-.12*	-.17**
2. 14 day alcohol consumption (UK units)	7.37 (14.59)	-	.77**	.43**	.54**	.67**	.16**	.21**	-.01	-.06	.06
3. Number of days drinking	0.87 (1.16)		-	.41**	.55**	.55**	.25**	.21**	-.01	-.05	0.9
4. Problem Index	0.78 (1.23)			-	.35**	.41**	.23**	.11*	.03	.02	.14*
5. Length of time regularly drinking (months)	6.60 (9.56)				-	.34**	.24**	.14*	.01	-.06	.07
6. Times drunk (6 months)	3.44 (8.26)					-	.15**	.19**	.00	-.03	.04
7. Age at first drink (years)	10.25 (4.65)						-	.07	.05	.03	.03
8. VP RT bias (ms)	6.80 (46.32)							-	.17**	-.09	.01
9. SRC bias (ms)	11.25 (83.52)								-	.00	.05
10. Inhibitory control (SSRT; ms)	259.29 (78.61)									-	.04
11. Delay discounting (AUC)	0.48 (0.26)										-

Economic status = Family affluence scale scored 0 (minimum) to 6 (maximum), higher scores are indicative of greater familial wealth; 14 day alcohol consumption (UK units), 1 unit = 8g alcohol; Number of days drinking = Number of days in the previous fourteen in which a full alcoholic beverage has been consumed; Problem index = Negative outcomes from drinking scores range from 0 (minimum) to 6 (maximum), higher scores are indicative of more negative outcomes; Length of time regularly drinking (months) = Number of months during which participants have classified themselves as regular drinkers; Times drunk (6 months) = Number of times that participants classified themselves as being intoxicated during the 6 month period before testing; Age at first drink = Age which participants consumed their first full alcoholic beverage; VP RT Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias; Inhibitory control (SSRT; ms) = Mean top latency minus mean go latency, averaged across the 5 experimental blocks, higher values indicate more poorer inhibitory control; Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. **p < .01, *p < .05

Table 4.2: Comparison of gender differences (Mean ±SD).

	Male	**Female**
	(N=133)	(N=134)
Economic status	3.76 (1.56)	3.60 (1.43)
14 day alcohol consumption (UK units)	9.27 (17.52)	5.49 (10.69)
Number of days drinking	1.02 (1.32)	0.72 (0.95)
Problem Index	0.96 (1.35)	0.59 (1.07)*
Length of time regularly drinking (months)	7.20 (6.69)	6.01 (9.42)
Times drunk (6 months)	4.08 (10.04)	2.81 (5.98)
Age at first drink	10.98 (4.15)	9.59 (5.03)
VP RT bias (ms)	5.92 (39.56)	7.67 (52.35)
SRC bias (ms)	4.30 (91.36)	18.10 (74.73)
Inhibitory control (SSRT; ms)	269.88 (72.29)	249.16 (83.21)
Delay discounting (AUC)	0.49 (0.27)	0.47 (0.25)

Economic status = Family affluence scale scored 0 (minimum) to 6 (maximum), higher scores are indicative of greater familial wealth; 14 day alcohol consumption (UK units), 1 unit = 8g alcohol; Number of days drinking = Number of days in the previous fourteen in which a full alcoholic beverage has been consumed; Problem index = Negative outcomes from drinking scores range from 0 (minimum) to 6 (maximum), higher scores are indicative of more negative outcomes; Length of time regularly drinking (months) = Number of months during which participants have classified themselves as regular drinkers; Times drunk (6 months) = Number of times that participants classified themselves as being intoxicated during the 6 month period before testing; Age at first drink = Age which participants consumed their first full alcoholic beverage; VP RT Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias; Inhibitory control (SSRT; ms) = Mean top latency minus mean go latency, averaged across the 5 experimental blocks, higher values indicate more poorer inhibitory control; Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. **p < .01, *p < .05

Table 4.3: Comparison of drinker status group differences (Mean ±SD).

	Abstainer-infrequent	**Regular**
	(N=142)	(N=125)
Economic status	3.53 (1.48)	3.85 (1.50)
VP RT bias (ms)	-2.69 (30.62)	17.30 (57.63)**
SRC bias (ms)	8.08 (75.51)	15.14 (92.20)
Inhibitory control (SSRT; ms)	265.49 (79.12)	251.54 (77.74)
Delay discounting (AUC)	0.49 (0.27)	0.48 (0.25)

Economic status = Family affluence scale scored 0 (minimum) to 6 (maximum), higher scores are indicative of greater familial wealth; VP RT Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias; Inhibitory control (SSRT; ms) = Mean top latency minus mean go latency, averaged across the 5 experimental blocks, higher values indicate more poorer inhibitory control; Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. **$p < .001$, *$p < .05$

Figure 4.1: Mean reaction times (±SEM) to congruent and incongruent probes in the Visual Probe task. Values shown separately for regular and abstainer-infrequent drinkers (*p<.01).

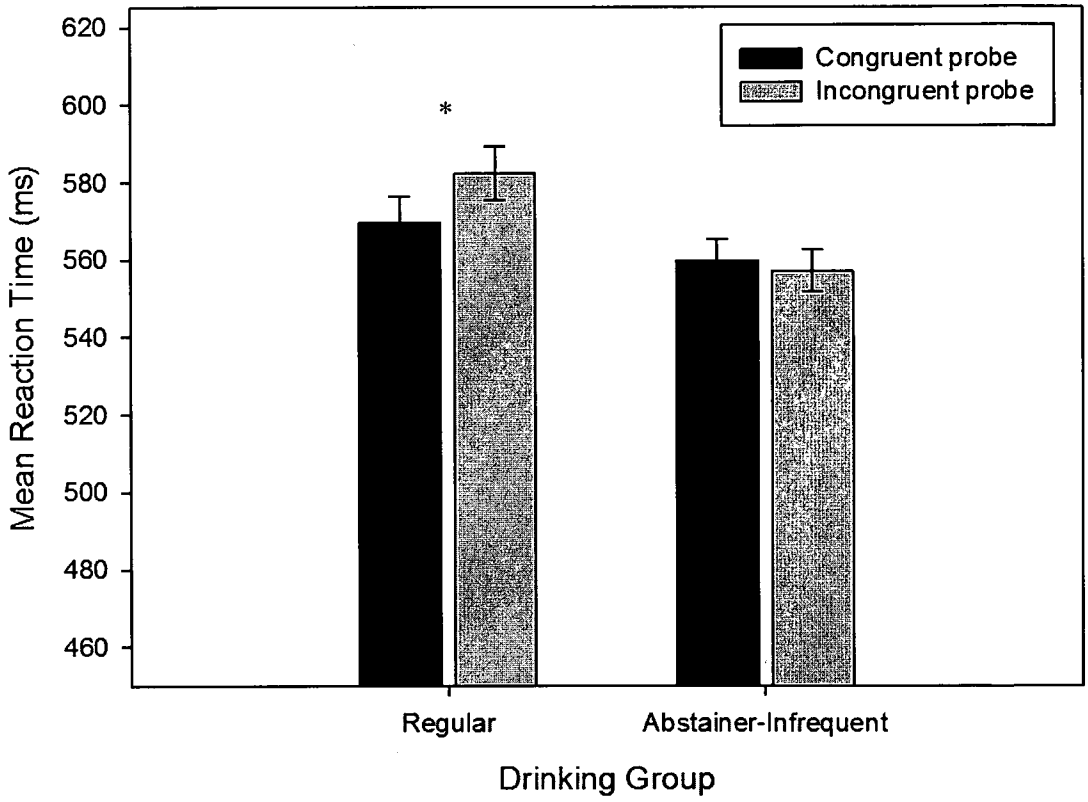


Table 4.4: Regression analysis showing demographics, behavioural impulsivity, and automatic cognitive processing, as predictors of alcohol use index scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Gender	.01	-.04	$F(2,215) = 0.57$	-0.38	0.47	-.06
Economic status				-0.12	0.15	-.06
Step 2						
VP RT Bias (ms)	.04	.03	$F(2,213) = 4.29$*	0.01	0.01	.19**
SRC bias (ms)				-0.00	0.01	-.05
Step 3						
Delay discounting (AUC)	.05	.02	$F(2,211) = 0.46$	0.93	0.93	.07
SSRT (ms)				-0.00	0.00	-.04
Step 4						
VP RT Bias (ms)XDelay discounting (AUC)	.06	.02	$F(4,207) = 0.68$	-0.01	0.02	-.04
VP RT Bias (ms)XSSRT (ms)				1.22†	0.01	-.01
SRC bias (ms)XDelay discounting (AUC)				-0.00	0.01	-.01
SRC bias (ms)XSSRT (ms)				4.69†	0.01	-.10

Economic status = Family affluence scale scored 0 (minimum) to 6 (maximum), higher scores are indicative of greater familial wealth . VP RT Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias; Inhibitory control (SSRT; ms) = Mean top latency minus mean go latency, averaged across the 5 experimental blocks, higher values indicate more poorer inhibitory control; Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Cognitive bias measureXbehavioural impulsivity measure = product of normalized variables. †= 10^-5, *p < .05, **p < .01

Table 4.5: Regression analysis showing demographics, behavioural impulsivity, and automatic cognitive processing, as predictors of the alcohol problem index

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Gender	.02	.01	$F(2,218) = 2.16$	-0.07	0.03	-.15*
Economic status				-0.01	0.10	-0.04
Step 2						
VP RT Bias (ms)	.03	.02	$F(2,216) = 1.61$	0.01	0.00	.14
SRC bias (ms)				4.14†	0.00	0.02
Step 3						
Delay discounting (AUC)	.06	.03	$F(2,214) = 2.38$	0.10	0.07	.10
SSRT (ms)				0.00	0.00	.08
Step 4						
VP RT Bias (ms)XDelay discounting (AUC)	.10	.06	$F(4,210) = 2.71$*	0.01	0.00	.15*
VP RT Bias (ms)XSSRT (ms)				7.54†	0.00	.10
SRC bias (ms)XDelay discounting (AUC)				0.01	0.01	.09
SRC bias (ms)XSSRT (ms)				-3.91†	0.00	-.11

Economic status = Family affluence scale scored 0 (minimum) to 6 (maximum), higher scores are indicative of greater familial wealth . VP RT Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias; Inhibitory control (SSRT; ms) = Mean top latency minus mean go latency, averaged across the 5 experimental blocks, higher values indicate more poorer inhibitory control; Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. Cognitive bias measureXbehavioural impulsivity measure = product of normalized variables. †= 10^-5, *p < .05, **p < .0.01

possible that social learning may have an impact on the development of attentional processes (Zucker et al., 1995) or that there is a degree of heritability as suggested by (Zetteler et al., 2006), that is not the case for automatic alcohol-approach responses. Future research could seek to elucidate as to which forms of cognitive bias in adolescents are influenced by different factors; social learning, genetics, specific conditioning history etc. This would allow the identification of at risk adolescents and the development of targeted interventions which could aim at reducing the impact of the specific forms of cognitive bias that are most associated with adolescent drinking.

Despite the considerable evidence base implicating both impulsive decision making (e.g. Field et al., 2007a; Fields et al., 2009a) and inhibitory control (e.g. Wong et al., 2006) as a risk factor for adolescent drug and alcohol use the current study found no direct association been these measures and hazardous drinking or the alcohol use index. It is notable that in the literature there are several examples in which impulsive decision making does not predict risk behaviours such as smoking (e.g. Reynolds et al., 2003). Furthermore, there is only one study to date that has shown that delay discounting is associated with increased alcohol consumption in adolescents (Field et al., 2007a). The recent meta-analysis into delay discounting and alcohol use by MacKillop et al. (2011) found that although there is evidence for a significant positive association between delay discounting and alcohol use in non-dependent populations this association is weak. It may simply be because the current sample did not contain participants with a significant enough drinking history to exhibit increased discounting of future rewards. Likewise, although impairments in inhibitory control prospectively predict alcohol and drug use (Tarter et al., 2003; Wong et al., 2006) it is notable that Nigg et al. (2006) found that inhibitory control deficits prospectively predicted a small (1%) amount of variance in alcohol consumption in a sample of adolescents who were not stated as being at risk. In at risk participants, inhibitory control deficits were found to predict more variance in alcohol use (9%). The current study and Nigg et al. (2006) suggests that little variance in alcohol use is accounted for by inhibitory control deficits in populations that are not defined as being at risk.

Although there was no evidence for a direct association between behavioural impulsivity and alcohol use in the current study, there was evidence that the impact of attentional bias on hazardous drinking was moderated by impulsive decision making. Attentional bias only predicted hazardous drinking in participants who discounted future rewards steeply, as predicted by dual process models of addiction. Significantly, attentional bias was directly associated with alcohol use (and was also greater in regular drinkers compared to infrequent/non-drinkers) but not hazardous drinking. This suggests that although attentional bias towards alcohol-related cues is related to alcohol use it is only specifically associated with hazardous drinking patterns in participants with elevated impulsive decision making. This is consistent with previous research that demonstrated that the impact of automatic alcohol cognitions on alcohol consumption is moderated by controlled process (i.e. working memory; Grenard et al., 2008; Thush et al., 2008). There was, however, no evidence for the hypothesis that the impact of attentional bias on drinking would be moderated by deficits in inhibitory control as would be predicted by dual process models of addiction (e.g. Goldstein & Volkow 2002). One speculative explanation for this is that aspects of behavioural impulsivity have been shown to follow different developmental trajectories, with a reduction in impulsive decision making developing later in adolescence compared to inhibitory control and other measures of executive cognitive functioning such as working memory (Prencipe et al., 2011). It is possible that within this age group inhibitory control was relatively well developed in the sample as a whole. This would suggest that within this age group of adolescents steep discounting of future rewards (along with attentional bias) may be particularly implicated in hazardous drinking. Interventions aimed at reducing impulsive decision making (e.g. Bickel et al., 2011) which have been shown to be efficacious in adults might therefore also be a suitable treatment for adolescents.

4.6. Chapter summary

The current study found that adolescent alcohol use was associated with attentional bias towards alcohol-related cues but not automatic approach tendencies. As in previous studies (studies one and two) there was no evidence for a direct association between either measure of behavioural impulsivity and alcohol use indices. One explanation being that impulsivity only has a significant association with drinking behaviour in older adults with significant drinking histories. It is possible that prolonged alcohol use increases impulsivity, therefore it will only be identifiable in samples of older adults. Although in isolation measures of behavioural impulsivity did not explain variance in drinking behaviour, the impact of attentional bias on hazardous drinking (but not the alcohol use index) was moderated by impulsive decision making. This suggests that increased attentional bias towards alcohol and elevated discounting of future rewards is a particular risk factor for hazardous drinking. It is possible that hazardous drinking is therefore the result of motivational responses to alcohol-related stimuli that the individual finds hard to resist due to a tendency to opt for immediate reward (e.g. a 'buzz' from drinking, peer approval) over long term benefits (e.g. improved school performance, reduced likelihood of getting in trouble with police and parents).

4.7. Acknowledgements

The current study represented the final wave of testing of a larger longitudinal study, comprising five assessments conducted over a two year period. This project was funded by a research grant from the Medical Research Council (reference G0601070) awarded to Dr Matt Field. My role in the study included contributions to the planning of the study protocol, the choice of appropriate measures, and assistance with data collection.

Chapter 5

The role of impulsive decision making, inhibitory control and automatic responses to alcohol-related cues in hazardous drinking among older adults.

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5.1. Abstract

Hazardous drinking is associated with both increased impulsivity and automatic responses to alcohol-related cues. However, impulsivity is a multi-factorial construct, and it is currently unclear if all components of impulsivity are associated with heavy drinking. Furthermore, emerging evidence suggests that the relationships between hazardous drinking and automatic alcohol cognitions may be moderated by individual differences in impulsivity. The current study aimed to investigate these associations in a sample of older adult drinkers (mean age 28.95 ±11.57). Ninety-seven social drinkers (65 female) completed questionnaire measures of self report impulsivity, alcohol consumption and hazardous drinking. Participants also completed computerised measures of automatic alcohol approach tendencies (stimulus-response compatibility (SRC) task), attentional bias (visual probe task) and two behavioural measures of impulsivity (Go/No-Go and delay discounting tasks) as well as measure of phonemic fluency (COWAT). Both measures of behavioural impulsivity and the measure of self report impulsivity predicted unique variance in hazardous drinking, although phonemic fluency did not. Automatic alcohol-approach tendencies were also associated with hazardous drinking, although the impact of this relationship was not moderated by impulsivity. There was however, no association between attentional bias and hazardous drinking. These results indicate that multiple components of impulsivity and automatic alcohol approach tendencies explain unique variance in hazardous drinking. Principal component analysis revealed that the two measures of behavioural impulsivity were distinct from each other and from self-reported trait impulsivity, although self-report non-planning impulsivity loaded on to two factors (trait impulsivity and delay discounting).

5.2. Introduction

Contemporary theories of addiction propose that both increased impulsivity and heightened salience of alcohol-related cues play a central role in alcohol use disorders. For example, elevated impulsivity has been closely linked with alcohol use disorders, and it may play a causal role in loss of control over drinking (de Wit 2009). Likewise, it has been shown that alcohol-related cues acquire conditioned incentive-motivational properties ('incentive salience'), causing those cues to capture attention and initiate approach behaviours automatically, ultimately leading to alcohol consumption (Robinson & Berridge 2001). Recent theoretical models (Goldstein & Volkow 2002; Jentsch & Taylor 1999; Wiers et al., 2007) make more detailed predictions about how impulsivity and incentive salience might interact during the development of alcohol use disorders. For example, Wiers et al. (2007) suggested that approach behaviour automatically elicited by alcohol cues should result in increased alcohol consumption, but this effect should be moderated by individual differences in impulsivity, with highly impulsive individuals more sensitive to the incentive properties of alcohol cues. In support of this model, recent experimental studies have demonstrated that measures of impulsivity and executive (dys)function moderate the association between automatic processing of alcohol-related cues and individual differences in drinking behaviour (for a review, Stacy & Wiers 2010).

Although impulsivity has commonly been associated with alcohol and other drug use findings are generally inconsistent. Indeed, the previous three studies in this thesis have not shown any evidence for a direct association between measures of impulsive decision making or inhibitory control and hazardous drinking in student or adolescent samples as would be predicted by de Wit and Richards (2004) and Olmstead (2006). One possible reason for this lack of direct association between these measures and hazardous drinking is the samples used. The previous studies have utilised young adult (students) and adolescent samples. Impulsivity may be the consequence of heavy drinking; in young adult samples impulsivity may not be elevated due to limited drinking histories. By recruiting a sample of older adults the

current study aims to look at the association between behavioural impulsivity and heavy drinking in a sample in which heavy drinking would be more maladaptive and inappropriate due to employment, family commitments etc. It is important to note that impulsivity is generally defined as 'maladaptive or inappropriate behaviours' (de Wit 2009 p 23), within younger samples heavy drinking may not be maladaptive or inappropriate, it may be socially normative. Indeed, Littlefield et al. (2009) found that impulsivity was associated with adults who did not 'mature out' of the pattern of problematic alcohol use that is associated with the late teenage years and early twenties. In addition an older adult sample would have a much greater drinking history, so it would be expected that there would be a stronger association between behavioural impulsivity and alcohol use.

With regard to impulsive decision-making, in which individuals are over-sensitive to immediate rewards but insensitive to delayed rewards or negative consequences, there is some evidence that heavy social drinkers and alcoholics show an increased rate of delay discounting (i.e. preference for smaller immediate rewards) compared to light-drinker controls (Field et al., 2007a; Petry 2001; Vuchinich & Simpson 1998). However, several studies have revealed no association between delay discounting rate and individual differences in alcohol consumption or alcohol-related problems (Fernie et al., 2010; Kirby & Petry 2004; MacKillop et al., 2007). In addition, it is notable that those studies which have shown increased rates of discounting in student populations (Vuchinich & Simpson 1998) have used very large samples (527 and 380 participants in experiments one and two respectively), and still only found a relatively weak association between drinker status and discounting. In a recent meta-analysis MacKillop et al. (2011) found that although there is evidence for elevated discounting of future rewards in clinical samples of alcohol abusers, it is much less pronounced in non-clinical samples. This indicates that non-clinical samples may not contain enough participants with a significant drinking history to be exhibiting increased discounting of future rewards. Furthermore, many negative findings are in experiments conducted on samples of largely undergraduate drinkers (e.g. Fernie et al., 2010; MacKillop et al., 2007). Undergraduates have been shown to be motivated to drink by social factors (Faulkner et al., 2006; Wicki et al., 2010); therefore, undergraduate alcohol

consumption may not be associated with individual differences in impulsivity, but a decision facilitated by conformity to a social norm. By recruiting older adults it would be expected that the associations between impulsive decision making and hazardous drinking would be stronger due to greater drinking histories (which would be consistent with the findings of MacKillop et al., 2011).

The second, independent, component of impulsivity defined by de Wit and Richards (2004) and Olmstead (2006) is deficient inhibitory control. Inhibitory control refers to the ability to control or suppress pre-potent responses. Previous studies have assessed this with behavioural tasks including Go/No-Go (Newman & Kosson 1986) and Stop-Signal (Logan et al., 1984) tasks, both of which involve the suppression of prepotent motor responses. Although none of the previous studies have found evidence for an association between inhibitory control and hazardous drinking, some recent studies suggest that heavy drinking and alcoholism are associated with failures of inhibitory control on these tasks (Go/No-Go task: Colder & O'Connor 2002; Stop-Signal task: Goudriaan et al., 2006). In addition, Jones et al. (2011) found that induction of a disinhibited state resulted in increased alcohol consumption, relative to a control manipulation, which suggests a causal effect of disinhibition on alcohol-seeking. However, as with the delay discounting data, the findings are not consistent across studies. For example, Kamarajan et al. (2005) did not detect impairments in response inhibition among alcoholics compared to controls in a study that utilised a Go/No-Go task, and Fernie et al. (2010) found no association between alcohol consumption and performance on a Stop-Signal task among young adult social drinkers. These inconsistent findings may also be due to the samples used. If the participants are not trying to control their behaviour then there is no reason to assume they will be trying to inhibit a prepotent response to drink. Furthermore it may be that drinking is only associated with inhibitory control in pathological samples and that only weak associations are evident non-dependent student drinkers, as found for delay discounting (MacKillop et al., 2011). Indeed, it is possible that inhibitory control deficits only become apparent after chronic alcohol use has directly impaired inhibitory control due to its effects on the prefrontal cortex (as argued by Lyvers 2000), this would indicate that only within a sample with a considerable drinking history will impairments in inhibitory control become

apparent. If this is the case then it would be expected that the sample recruited for this study would be more likely to show deficits in inhibitory control than previous samples.

In addition to behavioural measures of impulsivity, self-report measures (that treat impulsivity as a stable personality trait) have also been developed. These measures, in both cross-sectional and longitudinal studies suggest some correspondence between elevated self report impulsivity and increased alcohol consumption or alcohol problems (chapter three of the current thesis; Fernie et al., 2010; Gunnarsson et al., 2008; McAdams & Donnellan 2009; Von Diemen et al., 2008; Von Knorring et al., 1987). Indeed, evidence for the association between self report impulsivity and alcohol use is more compelling than for either measure of behavioural impulsivity.

There is a significant body of evidence demonstrating a clear dissociation between measures of impulsive decision making and inhibitory control. Reynolds et al. (2006a) used principal component analysis to investigate the independence of measures of behavioural impulsivity and found Stop-Signal and Go/No-Go tasks loaded on to a separate factor (impulsive disinhibition) from delay discounting and risk-taking tasks (impulsive decision making). Likewise, Swann et al. (2002) found two distinct components of behavioural impulsivity: 'rapid-response' and 'reward-delay impulsivity'. The relationship between these two behavioural measures of impulsivity and trait measures is less clear. White et al. (1994) found trait impulsivity to be a separate construct from behavioural impulsivity, and Reynolds et al. (2006a) also found that behavioural and trait measures were distinct. Swann et al. (2002) reported significant correlations between trait impulsivity and rapid-response impulsivity. Furthermore, de Wit et al. (2007) reported a significant correlation between the non-planning subscale of the BIS-11 and impulsive decision making and suggested that both these measures reflect insensitivity to delayed rewards. It is currently unclear how much unique variance each of the discussed types of impulsivity contributes towards hazardous drinking in non-dependent adult drinkers. It is possible that individuals develop an awareness of their behavioural impulsivity which is reflected in self report measures.

Although the literature regarding associations between impulsivity and heavy drinking is inconsistent, the literature concerning incentive-motivational properties of alcohol cues is much clearer. Indeed, the previous three studies have all found some association between automatic processing of alcohol related stimuli and hazardous drinking (or the alcohol use index in the case of adolescents) and previous research has also shown increased attentional bias among alcoholics and heavy social drinkers (Stetter et al., 1995; Townshend & Duka 2001; for a recent review, see Field & Cox 2008). Furthermore, individual differences in attentional bias for alcohol-related cues also prospectively predict alcohol use among heavy drinking University students (Fadardi & Cox 2008), and the degree of attentional bias predicts relapse and treatment dropout among treatment-seeking alcoholics (Cox et al., 2002). Finally, studies have shown that experimentally manipulating attentional bias can increase the urge to drink in the laboratory (Field & Eastwood 2005), suggesting a causal relationship between attentional bias and drinking.

With regard to overt behavioural approach elicited by alcohol cues, there was an association between hazardous drinking and automatic approach responses in studies one and two (albeit a relatively weak association), although SRC bias scores were not associated with adolescent drinking. Previous research using the SRC task has revealed that heavy, but not light drinkers, respond more rapidly during the 'approach alcohol' block compared to the 'avoid alcohol' block, which suggests that alcohol-related cues elicit an automatic approach tendency among such heavy drinkers (Field et al., 2008; Field et al., 2011). Palfai and Ostafin (2003), using an IAT, and Wiers et al. (2009) using an AAT, obtained comparable findings. In a recent manipulation study, Wiers et al. (2010) found that these automatic approach tendencies, like attentional bias, had a causal influence on the motivation to drink alcohol in the laboratory. In addition, Wiers et al. (2011) found that retraining alcoholics to avoid alcohol-related cues led to improved treatment outcomes. This suggests that automatic approach bias, like attentional bias, can drive alcohol consumption and is not merely an epiphenomenon of heavy drinking. It is notable that the only other study to date that has assessed automatic approach responses towards alcohol-related cues in adolescents found that they were associated with lower rates of alcohol consumption (van Hemel-Ruiter et al., 2011). Taken together,

the results concerning automatic approach responses and attentional bias suggest that attentional bias develops relatively quickly (and may even precede the onset of drinking; O'Connor et al., 2007) while automatic approach responses develop more slowly as a result of continued drinking. If this is the case it would be expected that there will be robust associations between hazardous drinking and both attentional bias and automatic approach responses among older adult heavy drinkers due to their significant conditioning histories.

Finally, as predicted by dual process models of addiction (e.g. Goldstein & Volkow 2002; Wiers et al., 2007) associations between other measures of automatic alcohol cognitions and drinking behaviour are moderated by individual differences in inhibitory control (Houben & Wiers 2009b), as well as other aspects of executive cognitive function such as working memory (Thush et al., 2008). These studies revealed that drinking behaviour of individuals with better executive functioning is weakly associated with, or unrelated to, automatic processing of alcohol-related cues, whereas these relationships are much stronger in individuals with poor executive functioning. It would therefore be expected that within this sample of older adults there would be support for dual process models of addiction as one would expect participants to be more likely to be trying to control their behaviour (i.e. engage controlled processes). Furthermore, as previously suggested the current sample may reveal greater associations between behavioural impulsivity and hazardous drinking as well as more robust associations between automatic processing of alcohol-related cues due to the significant drinking history of this sample. These factors may result in the predictions of dual process models of addiction becoming more apparent in this sample.

The current study investigated the relative contribution of automatic approach responses elicited by alcohol-related cues (as assessed with the SRC task) and attentional bias (measured by the visual probe task) to individual differences in hazardous drinking in a community sample. Furthermore, the contribution of behavioural impulsivity (inhibitory control and delay discounting) as well as a measure of phonemic fluency (COWAT) to hazardous drinking was assessed. By utilizing a community sample it was hoped that participants with longer drinking histories would be recruited and that within this sample there would be a reduced

likelihood of participant drinking being facilitated by a social norm. The hypothesis was that alcohol approach tendencies, attentional bias and the measures of impulsivity would explain unique variance in hazardous drinking. The current study also explored the prediction derived from Goldstein and Volkow (2002) and Wiers et al. (2007), which was that behavioural impulsivity measures would moderate the association between approach tendencies and attentional bias, and hazardous drinking. Finally, principal component analysis was used to investigate the independence of behavioural (delay discounting and a Go/No-Go task) trait measures of impulsivity (the BIS-11) and phonemic fluency. It was predicted that this analysis would identify two distinct components of behavioural impulsivity, with trait impulsivity emerging as a distinct factor and phonemic fluency also being an additional distinct factor.

5.3. Method

Participants

97 participants (65 female) aged between 18 and 59 years (mean 28.95 ±11.57) participated in the experiment (age groups break down as follows, 18-28= 58%; 28-38= 17%; 38-48= 14%; 48+ =11%). Participants were recruited from the staff and student population (although they were not undergraduates) of the University of Liverpool as well as workplaces within the Liverpool area. Employers were approached by the experimenter and consent to recruit on the premises was sought from a senior member of staff. Potential participants were excluded if they self-reported a current or past alcohol use disorder, or if their vision was not normal or corrected-to-normal. All participants provided informed consent, and the study was approved by the University of Liverpool Ethics committee.

Design

The current experiment used a correlational design to investigate whether measures of automatic cognitive processing and behavioural impulsivity predicted variance in hazardous drinking (AUDIT scores). In addition to looking at these factors in isolation the interaction between measures of automatic cognition and behavioural impulsivity in the prediction of hazardous drinking was also investigated. As measures of automatic cognition have been shown to be quantitatively distinct from each other the impact of each measure of automatic cognition (as well as its interaction with multiple measures of behavioural impulsivity) were assessed separately.

Materials

Questionnaires

Time Line Follow Back (TLFB).

The Alcohol Use Disorders Identification Test (AUDIT).

Barratt Impulsiveness Scale (BIS-11).

Temptation and Restraint inventory (TRI; Collins & Lapp 1992). This scale measures preoccupation with and attempts to limit drinking, and consists of two factors 'Cognitive and Emotional Preoccupation' (CEP) and 'Cognitive and Behavioural Control' (CBC). The TRI consists of 15 questions scored from 1 to 9 on a Likert scale. Recent evaluation of the psychometric properties of the TRI by (MacKillop et al., 2006) confirmed the two factor structure of the scales. It also revealed that, in a sample of hazardous drinkers, the overall internal reliability of the TRI (α= .87), and that of the CEP (α= .85) and the CBC subscales (α= .83), was good. Significantly, in harmful drinkers these properties were identical (except for the CBC subscale which had slightly lower α of .80).

Pictorial stimuli

The picture set was identical to that used in chapter three.

Cognitive bias tasks

Stimulus Response Compatibility (SRC) task.

The visual probe task. The visual probe task was identical to that used in study three except for the pictorial stimuli were presented for 2000 ms. Attentional Bias at this SOA was correlated with AUDIT scores in study two and revealed the largest effects in Field et al. (2004b)

Impulsivity/Executive cognitive function measures

Go/No-Go task (Newman & Kosson 1986). As used in study two

Delay discounting (Du et al., 2002). As used in study three.

Phonemic fluency. As used in study two.

Procedure

All participants were tested in a quiet testing cubicle at the University of Liverpool, or a quiet room within their workplace. Upon arrival, participants provided informed consent before providing a breath sample (all participants provided a zero breath alcohol reading). Participants then completed the questionnaire battery (including TLFB, AUDIT, BIS-11 and TRI) followed by the complete battery of cognitive tasks (SRC, Visual probe, Go/No-Go, delay discounting and COWAT); task order was counterbalanced across participants. Once participants had completed the questionnaire battery, they were thoroughly debriefed. Participants received £10 as compensation for their time.

5.4. Results

Sample Characteristics

Participants consumed a mean of 23.31 (±20.36; range 0-93) UK units of alcohol per week (1 UK unit = 8g alcohol), and the sample mean AUDIT score was 13.14 (±6.73). For descriptive statistics and correlations between all questionnaire measures and cognitive tasks see Table 5.1.

Gender differences in alcohol use, self report impulsivity and all cognitive tasks

Regarding the alcohol use indices males consumed significantly more UK units of alcohol in the previous week ($F(1,86)$ = 15.91, p < .001, η_p^2 = .16), had higher AUDIT scores ($F(1,86)$ = 6.42, p = .013, η_p^2 = .07) and scored higher on the CEP subscale of the TRI, ($F(1,86)$ = 4.86, p = .03, η_p^2 = .05). Males also had significantly higher self report non-planning impulsivity ($F(1,86)$ = 6.14, p = .015, η_p^2 = .07). There were also trends for males having higher BIS-11 total scores ($F(1,86)$ = 3.52, p = .067, η_p^2 = .04) and elevated AUC values compared to their female counterparts ($F(1,86)$ = 3.12, p = .081, η_p^2 = .04). See Table 5.2 for descriptive statistics broken down by gender.

Principle component analysis for dimensions of impulsivity

The PCA was conducted on the behavioural measures of impulsivity, phonemic fluency and BIS-11 subscales with oblique rotation (oblimin). The sampling adequacy was deemed to be acceptable (KMO = .62) and Bartlett's test of sphericity demonstrated that correlations between items were large enough for PCA ($\chi^2(15)$ = 88.18, p < .001). The PCA revealed four components that explained 85.98% of variance. Table 5.3 shows the factor loadings following rotation, which suggests that cluster one represents self report impulsivity, cluster two represents impulsive decision making, cluster three represents phonemic fluency and cluster four represents inhibitory control. Significantly, the non-planning subscale of the BIS-11

loaded onto the self report impulsivity component and the impulsive decision making component.

Predicting hazardous drinking: Attentional bias and behavioural impulsivity (Table 5.4)

The full regression model predicted 26% of the variance in AUDIT scores ($R^2 = .35$, R^2 adjusted = .26, $F(10,77) = 4.07$, $p < .001$). Age was a trend predictor of AUDIT scores (β=-.19, $p < .1$), with older drinkers having lower scores. In the simultaneous regression model gender was not a significant predictor of AUDIT scores. Participants with higher scores on the BIS-11 also had significantly greater AUDIT scores (β=.28, $p = .011$). Unexpectedly, attentional bias in the visual probe task was not associated with increased AUDIT scores (β=-.02, $p > .1$). There was a significant association between delay discounting and AUDIT scores inasmuch that participants with steeper discounting of future rewards had significantly greater AUDIT scores (β=.28, $p = .009$). Although there was a trend towards an association between No-Go errors and AUDIT scores (β=.17, $p < .1$), COWAT switches did not predict a significant amount of variance in AUDIT scores in the simultaneous model. Finally, the interactions between attentional bias and measures of delay discounting or inhibitory control / ECF did not explain additional variance in AUDIT scores (ps > .1).

Predicting hazardous drinking: Automatic approach responses and behavioural impulsivity (Table 5.5)

The full regression model predicted 32% of the variance in AUDIT scores ($R^2 = .39$, R^2 adjusted = 0.32, $F(10,77) = 5.00$, $p < .001$). Age was a trend predictor of AUDIT scores (β=-.19, $p <. 1$), with older drinkers having lower scores. In the simultaneous regression model gender was not a significant predictor of AUDIT scores. Participants with higher scores on the BIS-11 also had significantly greater AUDIT scores (β=.31, $p = .003$). SRC bias was a significant predictor of AUDIT scores (β=.32 $p = .008$), as stronger automatic approach tendencies elicited by alcohol-

related cues were associated with higher AUDIT scores. There was a significant association between No-Go errors and AUDIT scores, with more commission errors being associated with greater AUDIT scores (β=.21, p = .033). There was also a significant association between delay discounting and AUDIT scores in that participants with steeper discounting of future rewards had significantly greater AUDIT scores (β=.28, p = .006). COWAT switches (β=-.04, p > .1) did not predict a significant amount of variance in AUDIT scores. Finally, the interactions between SRC bias and measures of inhibitory control / ECF did not explain additional variance in AUDIT scores (ps > .1).

Table 5.1: Descriptive statistics and inter-correlations for age, weekly alcohol consumption, questionnaire measures and experimental tasks.

	Mean (±SD)	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age	28.39 (11.20)	-.14	-.32**	-.02	-.13	-.26**	-.30**	-.23*	-.31**	-.03	.07	.16	-.21**	.33**
2. Weekly alcohol consumption	23.64 (20.83)	-	.68**	.55**	.23*	.33**	.14	.24*	.31**	.14	.10	.09	.33**	-.05
3. AUDIT	11.87 (6.71)		-	.61**	.38**	.45**	.26**	.39**	.45**	.15	.24*	.04	.38**	-.14
4. TRI CEP	23.55 (12.33)			-	.44**	.36**	.21*	.24*	.34**	.11	.14	.02	.17	-.07
5. TRI CBC	17.62 (9.98)				-	.26**	.17	.19*	.27**	.02	-.01	-.05	.25*	-.12
6. BIS-11 Non-Planning	25.17 (5.44)					-	.41**	.49**	.82**	.20*	.10	-.16	.43**	-.19*
7. BIS-11 Motor	23.33 (4.80)						-	.56**	.81**	.02	.05	-.05	.11	-.15
8. BIS-11 Attentional	18.12 (3.18)							-	.79**	.21*	.14	-.04	.11	.03
9. BIS-11 Total	66.40 (10.66)								-	.18	.11	-.17	.25*	-.15
10. VP RT bias (ms)	-0.92 (39.48)									-	.29**	-.17	-.11	-.03
11. SRC bias (ms)	35.40 (99.30)										-	-.14	.04	.08
12. Inhibitory control (No-Go errors)	11.36 (6.40)											-	-.20*	-.17
13. Delay discounting (AUC)	0.24 (0.23)												-	-.11
14. COWAT Switches	28.61 (8.62)													-

Weekly alcohol consumption in UK units, 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol TRI = Temptation and Restraint inventory, range of TRI subscale scores (minimum to maximum); Cognitive and Emotional Preoccupation (CEP) 9 to 81, Cognitive and Behavioural Control (CBC) 6 to 54. Range of BIS-11 subscale scores are (minimum to maximum); Non-Planning 12 to 48, Motor 10 to 40, Attentional 8 to 32. SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. Inhibitory control (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function **p < .01, *p < .05

Table 5.2: Comparison of gender differences (Mean ±SD).

	Male	**Female**
	(N=32)	(N=65)
Age (years)	28.30 (11.41)	29.43 (11.87)
14 day alcohol consumption in UK units	33.11 (26.77)	18.19 (13.69)*
AUDIT	14.32 (6.27)	10.54 (6.31)*
TRI CEP	26.97 (13.00)	21.30 (11.06)*
TRI CBC	19.24 (9.67)	16.21 (9.67)
BIS-11 Non-Planning	26.79 (6.13)	24.25 (4.56)
BIS-11 Motor	23.54 (5.15)	23.13 (4.48)
BIS-11 Attentional	18.61 (6.13)	17.68 (3.33)
BIS-11 Total	68.94 (11.31)	65.06 (10.14)
Visual probe RT bias (ms)	1.04 (41.32)	-1.95 (38.78)
SRC bias (ms)	48.02 (96.32)	31.94 (101.13)
Inhibitory control (No-Go errors)	12.61 (7.78)	11.27 (6.23)
Delay discounting (AUC)	0.27 (0.25)	0.22 (0.22)
COWAT Switches	28.72 (6.43)	28.56 (9.21)

Weekly alcohol consumption in UK units, 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol TRI = Temptation and Restraint inventory, range of TRI subscale scores (minimum to maximum); Cognitive and Emotional Preoccupation (CEP) 9 to 81, Cognitive and Behavioural Control (CBC) 6 to 54. Range of BIS-11 subscale scores are (minimum to maximum); Non-Planning 12 to 48, Motor 10 to 40, Attentional 8 to 32. SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. Inhibitory control (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function **p < .01, *p < .05

Table 5.3: Principle component analysis for behavioural measures of impulsivity and BIS-11 subscales (N=95)

	Rotated components			
	1	2	3	4
Eigenvalues	2.19	1.24	0.90	0.82
Variance (%)	36.52	20.73	14.99	13.74
BIS-11 Attentional	**0.89**	0.03	-0.18	0.01
BIS-11 Motor	**0.86**	0.10	0.17	0.01
BIS-11 Non-planning	**0.51**	**0.56**	0.06	0.02
Go/No-Go	0.00	0.01	0.00	**1.00**
Delay discounting	0.11	**0.96**	0.02	0.02
COWAT Switches	-0.01	0.02	**0.98**	0.01

Note: Factors highlighted load above 0.512 and are considered significant (Stevens, 2002).
Range of BIS-11 subscale scores are (minimum to maximum); Non-Planning 12 to 48, Motor 10 to 40, Attentional 8 to 32. Inhibitory control (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function

Table 5.4: Regression analysis showing trait impulsivity, behavioural impulsivity measures, and maintenance of attention (visual probe task reaction time, VP bias), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.14	.12	$F(2,85) = 6.98^{**}$	-0.10	0.06	-.19
Gender				-1.17	1.37	.08
Step 2						
BIS-11 total score	.26	.23	$F(1,84) = 12.99^{**}$	0.17	0.07	.28**
Step 3						
VP Bias (ms)	.26	.22	$F(1,83) = 0.04$	-0.01	0.03	-.02
Step 4						
Go\No-Go (No-Go errors)	.33	.28	$F(3,80) = 3.10^{*}$	0.18	0.11	.17
Delay discounting (AUC)				-8.23	3.09	.28**
COWAT switches				0.01	0.08	.13
Step 5						
VP Bias (ms)XGo\No-Go (No-Go errors)	.35	.26	$F(3,77) = 0.47$	-0.00	0.00	-.02
VP Bias (ms)XDelay discounting (AUC)				-0.06	0.09	-.08
VP Bias (ms)XCOWAT switches				0.00	0.01	-.16

BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum).VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation.. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting.. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. Cognitive bias measureXInhibitory control/ECF measure = product of normalized variables. *p < .05, **p < .01

Table 5.5: Regression analysis showing trait impulsivity, behavioural impulsivity measures, and automatic approach responses (SRC bias), as predictors of AUDIT scores

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Age	.14	.12	$F(2,85) = 6.98^{**}$	-0.11	0.06	-.19
Gender				-1.32	1.31	.09
Step 2						
BIS-11 total score	.26	.23	$F(1,84) = 12.99^{**}$	0.18	0.06	.31**
Step 3						
SRC Bias (ms)	.29	.26	$F(1,83) = 4.45^{*}$	0.02	0.01	.32**
Step 4						
Go\No-Go (No-Go errors)	.37	.32	$F(3,80) = 3.35^{*}$	0.22	0.10	.21*
Delay discounting (AUC)				8.16	2.86	.28**
COWAT switches				0.03	0.08	-.04
Step 5						
SRC Bias (ms)XGo\No-Go (No-Go errors)	.39	.32	$F(3,77) = 0.09$	0.00	0.00	.10
SRC Bias (ms)X Delay discounting (AUC)				0.04	0.03	.12
SRC Bias (ms)XCOWAT switches				0.00	0.01	.14

BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum).VP Bias = Mean reaction time to incongruent probes minus mean reaction time to congruent probes, higher values indicate increased attentional bias; ms represent duration of cue presentation.. Go / No-Go (No-Go errors) = Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC) = Area under the curve, values range from 0 to 1, with higher values indicating a steeper rate of delay discounting.. COWAT switches = Total number of switches between word groups in the phonemic fluency task (reversed scores), with high scores indicating poorer executive cognitive function. Cognitive bias measureXInhibitory control/ECF measure = product of normalized variables. *p < .05, **p < .

5.5. Discussion

The current study investigated the relative contribution of measures of behavioural impulsivity as well as automatic responses to alcohol-related cues (approach bias and attentional bias), and interactions between the two, as predictors of hazardous drinking in older adults. In addition the independence of behavioural and trait measures of impulsivity were also investigated. It was hypothesised that the different measures of impulsivity would explain unique variance in hazardous drinking as would automatic responses elicited by alcohol-related cues. The specific hypotheses of dual process models of addiction (e.g. Wiers et al., 2007), that the association between hazardous drinking and measures of automatic alcohol cognitions would be moderated by behavioural impulsivity, was also tested. Finally, it was hypothesised that the PCA would reveal impulsive decision making, inhibitory control and self report measures of impulsivity to be independent from each other. Results were generally supportive of the hypotheses. Delay discounting, inhibitory control, self report impulsivity and automatic approach bias all explained unique variance in hazardous drinking, although attentional bias and phonemic fluency did not. The interactions between both measures of automatic responses to alcohol-related cues and the measures of behavioural impulsivity did not explain additional variance in the regression model. Furthermore, PCA revealed three components of impulsivity (inhibitory control, delay discounting, and trait impulsivity) as predicted, although the non-planning subscale of the BIS-11 loaded onto both trait impulsivity and impulsive decision making, and as expected phonemic fluency loaded onto a unique component.

Unlike in previous studies both components of behavioural impulsivity – inhibitory control and delay discounting – as well as trait impulsivity, predicted unique variance in hazardous drinking. The finding that trait impulsivity is associated with alcohol use is consistent with studies one and two as well as a large body of previous research (e.g. McAdams & Donnellan 2009; Von Diemen et al., 2008). Associations between heavy drinking and both components of behavioural impulsivity have also been reported in previous studies (delay discounting, see Vuchinich & Simpson 1998

and Petry 2001; poor inhibitory control, see Colder & O'Connor 2002 and Goudriaan et al., 2006). However, there have been numerous failures to replicate these associations, particularly among non-dependent 'social' drinkers (e.g. Fernie et al., 2010; MacKillop et al., 2007, as well as studies one and three of the current thesis). The recent meta-analysis by MacKillop et al. (2011), along with the current study suggests that these findings may be at least partly attributable to the samples investigated. MacKillop et al. (2011) reported that although delay discounting is elevated in clinical samples, the effect size (referring to the relationship between delay discounting and drinking habits) is smaller in non-dependent populations. If non-clinical studies recruited young adult social drinkers, particularly University students, who had no reason to attempt to control their drinking behaviour, then one would expect an inconsistent pattern of associations between impulsivity and heavy drinking. The longer drinking histories of the current sample may also have contributed towards the significant associations between behavioural impulsivity and hazardous drinking. Chronic alcohol use has been shown to have a detrimental effect on the prefrontal cortex (Lyvers 2000), therefore the greater drinking histories of the current sample may have resulted in increased impulsivity, although extensive longitudinal research would be required to test this assertion.

Significantly, although behavioural impulsivity predicted AUDIT scores phonemic fluency did not. This suggests that it is not deficits in all aspects of executive cognitive function, (specifically updating working memory) but rather specific increases in impulsivity that are associated with hazardous drinking. One clinical implication is that novel interventions which aim to reduce impulsive decision making, or improve inhibitory control, may be suitable targets for the treatment of alcohol use disorders, particularly within older adults. Recent research suggests that training working memory reduces rates of delay discounting (see Bickel et al., 2011); this along with the current research suggests that treatments targeting reductions in delay discounting may reduce hazardous drinking among older adults.

The finding that automatic approach responses towards alcohol-related stimuli measured by the SRC task were associated with increased alcohol consumption replicates previous research using this task (Field et al., 2008; Field et al., 2011), and it is also consistent with similar findings obtained from related tasks, all of which

measure the strength of associations between the concepts of 'alcohol' and 'approach' (Ostafin & Marlatt 2008; Ostafin & Palfai 2006; Ostafin et al., 2003; Wiers et al., 2009). Recently, Wiers et al. (2010) demonstrated that behavioural training which aimed to reduce the strength of automatic approach tendencies elicited by alcohol-related cues led to reductions in ad-lib alcohol consumption which suggests that strong automatic alcohol-approach associations may be a fundamental driving force behind alcohol use and a suitable target for treatment (see Wiers et al., 2011). Although studies one and two demonstrated an association between automatic approach responses towards alcohol-related cues and hazardous drinking the findings in this study were considerably stronger than in previous studies. Indeed this is the first study in which the association between automatic approach responses and hazardous alcohol use was apparent in the simultaneous regression model. The pattern of findings in these cross sectional studies suggests that as individual's age and gain more cumulative experience with alcohol, automatic approach responses and their associations with alcohol use are strengthened.

Despite the association between approach responses and hazardous drinking there was no association between attentional bias and hazardous drinking. This was unexpected as previous research has shown that attentional bias is associated with heavy drinking (e.g. Field et al., 2004b; Miller & Fillmore 2010; Townshend & Duka 2001). The current thesis also found associations between attentional bias assessed by the visual probe task and hazardous drinking in students, as well as a robust association between this measure and adolescent alcohol consumption in study three. One possible explanation for this is that there is a subtle change in the automatic cognitive processes that are associated with drinking over an individual's lifetime. Conditioned approach responses may have a greater impact in older adulthood, which in turn, reduces the impact of attentional bias which is important early on in an individual's drinking career. Overall, the results concerning automatic approach responses suggest that alcohol-related cues possess incentive-motivational properties among hazardous drinkers, and as such they provide support for incentive-motivational theories of addiction and alcohol abuse (e.g. Robinson & Berridge 2001).

The specific predictions made by Wiers et al. (2007) and Goldstein and Volkow (2002), that the association between automatic responses elicited by alcohol cues and drinking behaviour would be moderated by measures of behavioural impulsivity were also investigated. However, the results did not provide any support for this prediction. One explanation for this finding is that automatic alcohol cognitions were assessed using an SRC and a visual probe task rather than an IAT as used by Houben and Wiers (2009b) and Thush et al. (2008). Another possible explanation for the failure to replicate previous research is that Houben and Wiers (2009b) assessed inhibitory control using a Stroop task, and Thush et al. (2008) investigated other aspects of executive cognitive function (specifically working memory). It is possible these tasks may have differing sensitivity to impairments in executive functioning/automatic alcohol cognitions, which may account for the current findings.

The finding that the two measures of behavioural impulsivity were distinct from each other is consistent with arguments made by de Wit and Richards (2004) and Olmstead (2006) inasmuch as two separate components of behavioural impulsivity, deficient inhibitory control and impulsive decision making, were found. Likewise the demonstration that phonemic fluency was independent from behavioural impulsivity also supports analyses on the independence of different forms of executive cognitive functioning (e.g. Miyake et al., 2000), as well as study two. The findings of the PCA regarding measures of behavioural impulsivity also serve to replicate and expand upon those of Reynolds et al. (2006a), who also found that a Go/No-Go task (and a Stop-Signal task) measured an aspect of impulsivity ('impulsive disinhibition') that was distinct from a second factor including delay discounting ('impulsive decision making'). In addition, it was shown that trait measures of impulsivity are partially independent from behavioural impulsivity, with all three subscales of the BIS-11 loading onto a third factor (trait impulsivity). Although these results are largely consistent with Reynolds et al. (2006a) it was found that non-planning impulsivity also loaded onto the impulsive decision making factor obtained from the PCA. One explanation for this is that participants have some insight into their impulsive decision making, which influences their response to questions which are concerned with a lack of planning for future events (possibly reflecting their decreased

sensitivity to future rewards, de Wit et al., 2007). This relationship is in contrast to that observed by Swann et al. (2002) who found that although trait impulsivity (particularly the non-planning of subscale of the BIS-11) was related to impulsive decision making a much stronger relationship was found between trait impulsivity (again, specifically non-planning) and measures of disinhibition. This, along with the current findings, suggest more research is required to investigate how trait measures of impulsivity are associated with behavioural measures.

5.6. Chapter summary

The current study found that increased impulsivity and automatic approach responses elicited by alcohol-related cues were associated with individual differences in hazardous drinking in older adults. Significantly, this is the first study in the current thesis to show a direct association between behavioural impulsivity and hazardous drinking. There was no evidence for the moderation of the association between hazardous drinking and approach responses by behavioural impulsivity as predicted by dual process models of addiction. Furthermore, the results suggest that behavioural impulsivity consists of two distinct components with self report impulsivity being an additional component. Interestingly, it was found that non-planning impulsivity loaded onto both the self report and decision making components of impulsivity which suggests that self report measures may reflect self knowledge of a tendency to make impulsive decisions.

Chapter 6

The pharmacological and anticipated effects of alcohol on automatic cognitive processes, behavioural impulsivity, executive cognitive functioning and subsequent alcohol seeking.

6.1. Abstract

Acute alcohol administration leads to biased processing of alcohol related cues, and impairs executive cognitive function. The current study aimed to investigate the effects of an alcohol prime (0.65 g/kg) on these processes and their relationship to alcohol-seeking behaviour. Thirty one social drinkers (19 female) completed three experimental sessions in which they received either 0.65 g/kg alcohol, a placebo, or a control beverage before they completed a battery of cognitive tasks including measures of automatic responses to alcohol-related cues (SRC and visual probe task), behavioural impulsivity (Cued Go/No-Go, and delay discounting) and a general measure of executive functioning (COWAT). At the end of each session participants also completed a bogus taste test in order to assess ad-lib drinking. Results indicated that COWAT performance was impaired after alcohol, and on the SRC task automatic alcohol-approach tendencies were pronounced after both alcohol and placebo compared to the control beverage; none of the other measures were significantly affected by alcohol or placebo administration. Ad-lib alcohol consumption increased after the alcohol prime. Importantly, increases in ad-lib drinking after alcohol administration were mediated by performance on the COWAT, but not automatic cognitive processes. The effects of the experimental condition on SRC performance were found to be mediated by impairments in executive cognitive function. Results suggest that the effect of 0.65g/kg alcohol on ad-lib drinking may be mediated by impairments in executive function rather than by increases in automatic approach tendencies elicited by alcohol-related cues.

6.2. Introduction

Acute alcohol intoxication has been found to increase the likelihood of engaging in a variety of potentially harmful behaviours, such as risky sexual practice (Conner et al., 2008; George & Stoner 2000), aggressive behaviour (Bushman & Cooper 1990; Subra et al., 2010) and cigarette smoking (Burton & Tiffany 1997; Epstein et al., 2007). In addition, alcohol administration increases subsequent alcohol seeking behaviour, resulting in loss of control over drinking. Laboratory studies have shown that priming doses of alcohol increase subsequent alcohol consumption in social drinkers (de Wit & Chutuape 1993; Rose & Duka 2006) and voluntary alcohol self-administration in alcohol-dependent individuals (Ludwig et al., 1974). This alcohol priming effect (de Wit 1996) may be a fundamental driving force behind repeated alcohol binges as well as other negative consequences of alcohol consumption. The current chapter aims to investigate the cognitive processes that may mediate the acute effects of alcohol on subsequent alcohol-seeking behaviour in social drinkers.

Several models of addiction (Goldstein & Volkow 2002; Jentsch & Taylor 1999; Wiers et al., 2007) propose that both mesolimbic reward mechanisms and behavioural impulsivity/executive cognitive functioning play important roles in addictive behaviour. These theories propose that conditioned responses elicited by alcohol-related stimuli can influence drinking behaviour, but executive processes such as inhibitory control and working memory can moderate their impact, as well as having a direct influence on drinking behaviour. For example, an individual may have a conditioned appetitive response to alcohol cues that increases the motivation to drink, but executive processes can moderate the impact of this response, thereby enabling an individual to refrain from drinking. Recent studies have shown that the impact of automatic appetitive associations on drinking behaviour is indeed moderated by working memory (Thush et al., 2008) and inhibitory control (as measured by Stroop interference; Houben & Wiers 2009b). Specifically, the drinking behaviour of individuals with better executive functioning is only weakly associated with automatic processing of alcohol-related cues, whereas associations are stronger in individuals with poor executive functioning. In a recent paper, Field et al. (2010)

proposed similar roles for automatic processes and executive function as cognitive mediators of the alcohol priming effect, i.e. the ability of initial doses of alcohol to prime further drinking behaviour.

Considering the evidence on automatic appetitive responses to alcohol-related cues Robinson and Berridge (1993; 2001) proposed that after chronic heavy drinking, alcohol-related stimuli acquire conditioned incentive-motivational properties. As a consequence, those stimuli are able to capture and hold attention, and elicit behavioural approach automatically; ultimately, this leads to a loss of control over drinking behaviour. Tests of selective attention have shown that alcohol cues capture and hold the attention of heavy, but not light, drinkers (Bruce & Jones 2004; Field & Cox 2008; Field et al., 2004b; for a recent review see Field & Cox 2008). This attentional bias may be associated with the risk of relapse to heavy drinking among individuals with alcohol dependence (Cox et al. 2002) and it may play a causal role in drinking behaviour (Field & Eastwood 2005; Schoenmakers et al., 2010). There is also evidence that alcohol-related cues can automatically elicit behavioural approach responses in heavy drinkers. For example, study four of the current thesis and Field et al., (2008) used a stimulus response compatibility (SRC) task in which participants were required to rapidly categorise alcohol-related and neutral pictures by moving a manikin towards alcohol pictures and away from neutral pictures in one block of the task, but vice versa in a different block. Hazardous drinkers (but not light drinkers) were faster to categorise alcohol-related pictures by making the symbolic approach response rather than the symbolic avoidance response. Research using related tasks to probe automatic associations between alcohol and concepts related to approach has yielded similar findings (Palfai & Ostafin 2003; Lindgren et al., 2009; Wiers et al., 2009). As with the literature on attentional bias, there is evidence that the automatic approach tendencies elicited by alcohol cues may play a causal role in drinking behaviour (Wiers et al., 2010), and novel treatments aimed at extinction of these responses may reduce the risk of relapse in individuals with alcohol dependence (Wiers et al., 2011).

A limited number of studies have investigated acute effects of alcohol on automatic appetitive responses to alcohol cues. Recently, Adams et al. (2011) found that a 0.4 g/kg priming dose of alcohol increased attentional bias as measured by a visual

probe task. However it seems likely that there is a non-linear relationship between alcohol dose and changes in attentional bias, for example, Duka and Townshend (2004) found that 0.3 g/kg of alcohol led to an increase in attentional bias for alcohol-related pictures, compared to placebo, although attentional bias was not increased after a higher (0.6 g/kg) dose. Similarly, Miller and Fillmore (2011) found no significant increase in attentional bias compared to placebo following 0.64 g/kg dose of alcohol, (although they also found no increase in attentional bias following a 0.32 g/kg dose). Schoenmakers et al. (2008) reported increased attentional bias after 0.3 g/kg of alcohol compared to placebo. The latter study also examined effects of a 0.3 g/kg dose on performance on the alcohol SRC task, but found no significant difference from placebo. Farris and Ostafin (2008) found some evidence that alcohol administration strengthened alcohol-approach associations relative to pre-drink baseline, however, participants were not given a uniform dose of alcohol, and there was no comparison with a placebo beverage. Therefore any conclusions on the effect of alcohol on the accessibility of alcohol-approach associations made from this study are speculative. Consideration of these findings raises the possibility that doses of alcohol higher than the 0.3 g /kg used in the Schoenmakers et al. (2008) study may activate automatic alcohol approach associations, but an additional possibility is that these effects may reflect the anticipated, rather than the pharmacological, effects of alcohol. Collectively, these studies do suggest dose-dependent effects of alcohol on automatic appetitive responses to alcohol-related cues, but none of the previous studies have examined if these effects are related to the increased alcohol-seeking behaviour that is seen during alcohol intoxication (i.e. the alcohol priming effect).

A more substantial body of research has investigated effects of acute and chronic alcohol on tasks which assess executive cognitive functioning and behavioural measures of impulsivity. Executive cognitive function refers to a set of inter-connected cognitive abilities which subserve the ability to inhibit pre-potent responses, hold information in working memory, and switch between different mental sets (Miyake et al., 2000). Impulsivity is commonly defined as rash or risky behaviours which do not take into account future consequences and are exemplified by a lack of planning (e.g. Dawe & Loxton 2004). As such the concept of impulsivity has considerable overlap with the notion of executive cognitive

(dys)function. Deficits in executive cognitive functioning and increased impulsivity have been found in alcohol dependent patients (Lawrence et al., 2009; Mitchell et al., 2005) as well as adolescent and young adult heavy drinkers (Colder & O'Connor 2002; Field et al., 2007a). Regarding acute alcohol effects, moderate (≥0.4 g/kg) doses of alcohol have been found to reliably impair inhibitory control when assessed with Cued Go/No-Go tasks (Marczinski et al., 2005; Marczinski et al., 2007) and Stop-Signal tasks (de Wit et al., 2000; Fillmore & Vogel-Sprott 1999; for a review see Field et al., 2010).

Impairment in inhibitory control following moderate alcohol consumption (0.65 g/kg) has been associated with measures of ad-lib drinking, such that participants who were more impaired by alcohol subsequently consumed more beer when given ad-lib access in a subsequent testing session (Weafer & Fillmore 2008). This important study suggests that alcohol induced impairments in inhibitory control may be associated with loss of control over drinking behaviour. However, the study does not reveal if alcohol-induced impairments in inhibitory control are associated with priming effects, i.e. alcohol-induced increases in ad-lib drinking after administration of a priming dose of alcohol. Furthermore, other aspects of executive functioning such as working memory and mental set shifting have also been shown to be impaired after alcohol administration. Balodis et al. (2007) and Grattan-Miscio and Vogel-Sprott (2005) found that working memory was significantly impaired following 0.7 g/kg and 0.62 g/kg of alcohol respectively. Recently, Guillot et al. (2010) found that mental set shifting, assessed by the trail making test and the Wisconsin card sorting task, was impaired following a priming dose of alcohol. Other measures of executive cognitive functioning, such as phonemic fluency tasks (e.g. controlled oral word association test, COWAT; Benton 1968) are also impaired following high doses (1.0 g/kg) of alcohol (Peterson et al., 1990). Measures of phonemic fluency are particularly sensitive to impairment in prefrontal cortex functioning, and have been used extensively to assess impaired executive cognitive functioning in clinical samples (e.g. Troyer et al., 1998a; Troyer et al., 1998b). It has been argued that phonemic fluency tasks assess a cluster of executive cognitive functions: working memory, inhibition and mental set switching (Abwender et al., 2001; Troyer et al., 1997). If the alcohol priming effect is related to impairments in

diverse executive functions, then the COWAT may be a particularly useful tool to investigate executive dysfunction as a cognitive mediator of the alcohol priming effect. However it is notable that recent principal component analyses have found that phonemic fluency tasks largely measure updating working memory, although factor loadings are not as high as for other working memory tasks such as letter-number sequencing (Verdejo-Garcia & Perez-Garcia 2007). If the COWAT is more representative of updating working memory then it would still be expected that performance would be impaired by a moderate priming dose of alcohol (e.g. Balodis et al. 2007;Grattan-Miscio & Vogel-Sprott 2005). Despite the relative sensitivity of measures of inhibitory control and associated measures of executive cognitive functioning to alcohol primes delay discounting has been found to be unaffected by moderate or higher priming doses of alcohol (Reynolds et al., 2006b; Richards et al., 1999), indeed Ortner et al. (2003) found a 0.7 g/kg of alcohol reduced delay discounting.

In their integration of the findings regarding the effects of alcohol on automatic cognitive processes and executive cognitive functioning Field et al. (2010) proposed a mechanism for the alcohol priming effect that was consistent with dual process models of addiction. They suggested that alcohol induced impairments in executive cognitive function may result in increased behavioural control by automatic cognitive processes, with this resulting in the alcohol priming effect. Therefore it would be expected that impairments in executive cognitive function/increased impulsivity would mediate increased accessibility of automatic cognitive processes. This increase in automatic cognitive processing would, in turn, mediate the alcohol priming effect.

Although priming studies have been informative on the pharmacological effects of alcohol on these processes studies that investigate the acute effects of alcohol usually adopt a methodology in which participants are administered alcohol in one session and placebo in another; any differences can then be attributed to the pharmacological effects of alcohol. None of the aforementioned studies included an additional control condition in which participants consumed an inert substance with the knowledge that the substance would have no effects. As a consequence, existing studies are uninformative regarding possible effects of the belief that alcohol was consumed,

which might occur independently of, or may mask, the pharmacological effects of alcohol. Consider the following, simplistic, example: acute alcohol, when given above a threshold dose, impairs choice reaction time (RT; Tzambazis & Stough 2000). If a study fails to show an effect of alcohol on choice RT (e.g. Fagan et al., 1987; Jääskeläinen et al., 1996), there are several possible explanations for this. For example, the response to a placebo may mimic the pharmacological effects of alcohol (Stewart et al., 1984), and therefore reaction time would be slow after administration of both alcohol and placebo; if these expectancy effects are large but the actual pharmacological effects of alcohol are relatively small, there would be no difference between alcohol and placebo and researchers may (erroneously) conclude that this specific dose of alcohol did not influence choice RT. Alternatively, the response to a placebo may be a form of compensatory response, which runs counter to (opposes) the pharmacological effects of alcohol (Siegel 1999; 2005). Again, if this compensatory response is large in comparison to a (relatively small) pharmacological effect of alcohol, there would be no difference between alcohol and placebo, and researchers could inappropriately conclude that there is no difference between alcohol and placebo.

If a study were to administer alcohol and a placebo and contrast those with a third beverage which participants knew to be pharmacologically inactive, this would help to clarify and distinguish between the pharmacological versus anticipated effects of alcohol. If the placebo response to alcohol does in fact mimic the pharmacological effect, choice RT would be slower after both alcohol and placebo, compared to after the control beverage. If the placebo response to alcohol counteracts the pharmacological effect (RT slowing), choice RT would be fastest after placebo, slightly slower after the control beverage, and slowest after alcohol. This is not just a practical issue. Arguably, comparing the effects of alcohol with a control beverage rather than a placebo would offer a more ecologically valid assessment of the acute effects of alcohol, as responses to alcohol in naturalistic settings (e.g. Friday night in a bar) will inevitably reflect the combined pharmacological and anticipated effects of the drug. In this sense, when seeking to understand the consequences of alcohol intoxication for health-related behaviours such as binge drinking, comparisons

between alcohol and placebo are unlikely to be representative of alcohol effects in naturalistic settings.

A meta-analysis on the pharmacological and anticipated effects of alcohol (Hull & Bond 1986), found that the anticipated effects of alcohol tended to affect the 'social' aspects of behaviour (aggression, sexual arousal) but have a very limited effect on non-social aspects of behaviour (e.g. memory and motor performance). A more recent series of studies highlighted the role of specific outcome expectancies on the anticipated effects of alcohol on motor skill tasks. Fillmore and Vogel-Sprott (1995) found impaired performance on motor skill tasks after both alcohol and placebo administration if participants expected to experience behavioural impairment. In addition, Fillmore et al. (1994) instructed participants that alcohol would either improve or impair performance on a pursuit rotor task. Those expecting impairment performed relatively better than participants who expected improvement, perhaps due to a compensatory response. To my knowledge, no previous studies have explored the role of expectancy effects on automatic cognitive processing of alcohol cues, although there are demonstrations that mere exposure to alcohol-related cues (Lindgren et al., 2009; Schulze & Jones 1999) and induction of positive mood (Birch et al., 2004) can influence automatic alcohol-related cognitions. It is possible that the alcoholic smell and taste of a placebo, as well as the expectation of receiving alcohol, may also influence automatic alcohol-related cognitions.

Although there is evidence for both drug-like and compensatory responses to placebo on tasks that assess psychomotor function, no previous studies have investigated the anticipated effects of alcohol on aspects of cognition associated with loss of control over drinking (Field et al., 2010). In addition, recent studies into the alcohol priming effect have also neglected to investigate the anticipated effects of alcohol on craving and ad-lib alcohol consumption. There is, however, evidence that the anticipated effects of alcohol have an important role in the alcohol priming effect. Using a balanced placebo design, Marlatt et al. (1973), demonstrated that if participants were informed that drinks contained alcohol, both a priming dose of alcohol and a placebo increased voluntary alcohol consumption in a subsequent taste test, compared to if participants were informed that drinks did not contain alcohol. These effects were seen in both current alcoholics and social drinkers.

The present study investigated the pharmacological and anticipated effects of alcohol, alone and in combination, by assessing behaviour after ingestion of alcohol (0.65 g/kg), placebo, and a control beverage which participants knew contained no alcohol. After drink administration, participants completed measures of impulsivity/executive cognitive function (Cued Go/No-Go, delay discounting and the COWAT), attentional bias (visual probe task) automatic approach tendencies elicited by alcohol-related cues (SRC task), and alcohol-seeking behaviour (ad-lib drinking during a bogus taste test). The primary hypothesis was that impulsivity would be increased (and therefore executive function would be impaired), attentional bias and automatic approach tendencies would be increased, and ad-lib alcohol consumption would be increased, after alcohol compared to both placebo and control beverages. Furthermore, it was hypothesised that the effects of alcohol on ad-lib drinking would be partially mediated by impairments in executive function and increases in attentional bias and automatic approach tendencies. It was also investigated if any increases in attentional and approach bias were mediated by increases in impulsivity/impairments in executive cognitive functioning. This experimental design also permitted the contrasting of responses to the placebo and the control beverage, in order to examine if the anticipated effects of alcohol, uncontaminated by its pharmacological effects, would influence executive cognitive function, automatic approach tendencies, attentional bias, and ad-lib drinking. It was hypothesised that the anticipated effects of alcohol would have drug-like effects on automatic cognitive processing, self reported craving and beer consumption, but have compensatory (drug-opposite) effects on behavioural impulsivity and executive cognitive functioning, as participants would compensate for anticipated impairment.

6.3. Method

Participants

Thirty one participants (19 female) aged between 18 and 40 years (mean 21.03 ± 4.12) were recruited from the University of Liverpool via word of mouth and intranet

advertisements. Inclusion criteria were fluency in English, and normal or corrected to normal vision. Participants were invited to take part if they self-reported consuming at least 15 units of alcohol (females) or 22 units (males) each week, which is in excess of the UK government guidelines for safe drinking (Edwards 1996). Exclusion criteria included current or past self-reported alcohol use disorder, current or recent illness which may increase sensitivity to alcohol (e.g. colds and flu), taking medication that is contraindicated for alcohol (e.g. antidepressants, anxiolytics), and aversion or allergy to any of the drink constituents (vodka, tonic water, or tabasco sauce). Additional exclusion criteria for female participants included current breastfeeding or pregnancy; the latter was confirmed with a pregnancy test at the beginning of the first session. All participants provided informed consent before taking part in the study, which was approved by the University of Liverpool Research Ethics Committee. Participants received either course credits or £30 as compensation for their travel expenses and time.

Design

The study utilized a within-subjects partially balanced placebo design. After a familiarization session, participants attended the laboratory for three sessions, with an interval of at least two days between sessions. During the three sessions, participants consumed an alcoholic drink, a placebo drink, and a control drink. A full balanced placebo design (i.e. includes a fourth session when alcohol is consumed but instructions are non-alcohol) was not used because the pharmacological effects of 0.65 g/kg alcohol render the deception ineffective (Sayette et al., 1994). The design enabled us to contrast the pharmacological effects of alcohol with its anticipated effects (alcohol vs. placebo), to identify the purely anticipated effects of alcohol (placebo vs. control), and to examine the combined influence of pharmacological and anticipated effects of alcohol (alcohol vs. control). All drinks were administered double blind by a second experimenter who also took breath alcohol samples. Session order was counterbalanced across participants.

Materials

Drink preparation

The alcoholic drink contained vodka (Smirnoff Red, 37.5% ABV); the dose was calculated as 0.65 g of pure alcohol per kg of body weight, up to a maximum of 200ml of vodka. The drink was mixed with chilled tonic water in the ratio one part vodka to three parts tonic. The placebo drink consisted of chilled tonic water only (identical total volume to the alcoholic drink). For both alcoholic and placebo drinks, a few drops of Tabasco sauce were added, vodka was smeared on the rim of the glass, and an atomiser was used to spray vodka mist on the surface of the drink. The control drink consisted of chilled water only, in the same total volume of alcoholic and placebo drinks.

Questionnaires

Time Line Follow Back (TLFB)

The Alcohol Use Disorders Identification Test (AUDIT).

Barratt Impulsivity Scale (BIS-11).

Desire for Alcohol Questionnaire – brief version (DAQ; Love et al., 1998). The DAQ is 14-item multidimensional alcohol craving scale that yields scores on four different factors of craving: Positive and Negative Reinforcement (six items), Strong Desires and Intentions (four items), Mild Desires and Intentions (two items), and Perceived Control Over Drinking (two items). Scores on each question range from 1 to 7, with higher scores on this scale being indicative of higher craving. Cronbach's α for the subscales of the DAQ range between (.95 and .97). Notably, all factors have highly significant loadings ($\geq$ .90) onto a single craving factor suggesting that the total score of the DAQ gives a reliable overall measure of craving.

Subjective intoxication scales (SIS; Duka et al., 1998). The SIS consisted of six 100mm visual analogue scales which assessed subjective feelings of 'light headed', 'irritable', 'stimulated', 'alert', 'relaxed' and 'contented'.

Perceived alcohol content. Participants were asked to 'Estimate how many standard 25ml shots of Vodka you have consumed' by circling a number from 1 to 9+ on a likert scale.

Pictorial stimuli

The picture set was identical to that used in chapter three.

Eye Tracking

Eye movements were recorded during the visual probe task using the Eyetrace 300x system (Applied Science Laboratories, Bedford, MA, USA) as described in chapter three, study two.

Cognitive bias tasks

Stimulus Response Compatibility (SRC) task.

The visual probe task. The visual probe task was identical to that used in study four although in addition to utilising the reaction time measure of attentional bias participants lateral eye movements were measured, using the Orbit Eyetrace software. Using this participants gaze position was assessed every 8.33 ms during the 2000 ms picture presentation. Gaze dwell times on alcohol pictures and control pictures were measured as a second dependent variable for the visual probe task. A bias score for gaze dwell times was derived by subtracting dwell times on control pictures from dwell times on alcohol pictures, with higher scores being indicative of increased attentional bias.

Impulsivity/Executive cognitive function measures

Cued Go/No-Go (GNG; Weafer and Fillmore 2008). The Cued Go/No-Go was programmed in Inquisit version 1.33 (Millisecond software, 2002). Each trial commenced with a fixation point for 800 ms followed by a blank screen for 500 ms. A cue was then presented for a variable duration (100 ms, 300 ms, 300 ms, 400 ms or 500 ms); following which a target (Go target or No-Go target) was presented until the participant responded or until 1000 ms had elapsed. There was an intertrial interval of 700 ms.

The cues consisted of white rectangles with a black outline 7.5cm x 2.5cm in size. Cues were presented horizontally ('Go' cue; 7.5cm wide 2.5cm tall) or vertically ('No-Go' cue; 2.5cm wide 7.5cm tall). The Go and No-Go targets were green and blue (respectively) and filled the interior of the cues following the variable SOA. Participants were instructed to respond to a green 'Go' target by pressing the spacebar as rapidly as possible and withhold responses to the blue 'No-Go' target.

The Cued Go/No-Go task consisted of 250 trials split into 5 blocks each containing 50 trials. Go cues and No-Go cues were presented in 125 trials each as were go and no-go targets. Horizontal cues ('Go cues') preceded a go target in 80% of trials and a no-go target in 20% of trials; vertical cues ('No-Go cues') preceded Go targets in 20% of trials and No-Go targets in 80% of trials. Each block consisted of 20 Go targets preceded by Go cues, 20 No-Go targets preceded by No-Go cues, 5 No-Go targets preceded by Go cues and 5 Go targets preceded by No-Go cues. The five cue SOAs were utilised in each cue target combination an equal number of times within each cue target combination. At the end of each block participants were informed of their average reaction time in order to encourage rapid responding. Number of errors of commission (responding to No-Go targets) and reaction times were recorded. In order to contrast the effect of the cue type a proportion of commission errors was calculated for No-Go trials that followed a 'Go' cue and No-Go trials that followed a 'No-Go' cue.

Delay discounting (DD; Du, et al., 2002). As used in study three.

Phonemic Fluency. The COWAT and scoring of the COWAT were identical to the previous studies. To reduce practice effects, participants were given different letters/letter combinations in each session (F, A and S; P, L and W; C, F and L) the order of which was counterbalanced across conditions. These letter combinations were found to produce a similar number of words in previous studies (Ross et al., 2007). In this analysis the COWAT switches were not reversed - high scores are therefore indicative of better executive cognitive functioning.

Bogus Taste Test

The taste test was based on that used by Field and Eastwood (2005). Participants were given a 275ml bottle of Becks non-alcoholic beer and a 275ml bottle of Orange and Passion fruit J^{2}0. The labels from both bottles were removed and participants were not informed that the beer was non-alcoholic. Participants were asked to taste the two drinks and rate them according to four continuums (unpleasant-pleasant, tasteless-strong tasting, bitter-sweet, flat-gassy) using 100 mm visual analogue scales. Participants were informed that they were allowed to drink as much of either drink as they needed to make accurate ratings. At the end of the session, the volume of each drink consumed was recorded. Informal debriefing indicated that none of the participants were aware that the beer was non-alcoholic.

Procedure

Testing sessions took place between 12 pm and 6 pm in a laboratory in the School of Psychology. Participants were asked to consume a high carbohydrate, low fat meal the night before and a light meal (e.g. a sandwich) an hour before each experimental session. Participants were also asked to avoid drinking alcoholic drinks before each session, and to avoid heavy drinking the night before each session. All participants provided a zero breath alcohol reading before each session (Lion Alcometer 500,

Lion Laboratories, Barry, UK). Participants initially attended the laboratory for a familiarization session (cf. Weafer & Fillmore, 2008) in which they completed a questionnaire battery (demographics, TLFB, AUDIT, and BIS-11) before the cognitive test battery. These data are not reported here as the purpose of the familiarization session was simply to enable participants to complete the tasks while sober.

During experimental sessions, participants initially provided a breath sample before completing the DAQ and SIS questionnaires. Drinks were then provided and participants were instructed to consume the drink within 10 minutes, before a 10 minute absorption period in which participants were provided with magazines to read. This pattern of alcohol administration yields a peak BAC approximately 65 minutes after consumption of 0.65 g/kg alcohol, therefore all cognitive tasks were completed during the ascending limb of the blood alcohol curve (Fillmore & Vogel-Sprott 1998).

After the absorption period, participants completed further DAQ and SIS questionnaires before being breathalysed by a second experimenter. Participants then completed a battery of cognitive tasks; the SRC task, a visual probe task with eye movement monitoring, Cued Go/No-Go task, delay discounting task and COWAT as described above. The entire cognitive test battery took approximately 40 minutes to complete. Participants then completed additional DAQ and SIS questionnaires and provided a further breath alcohol sample, before completing a short questionnaire in which they estimated the number of pub measures of vodka (one measure = 25ml vodka) that were in the drink. Finally, participants completed the bogus taste test procedure.

Participants were advised to remain in the laboratory until their BAC had declined to 0.39g/100ml (approximately half the UK drink drive limit). At the end of the final session, participants were fully debriefed before being discharged.

Data Analysis

Unlike in previous experiments repeated measures ANOVAs were used to analyse the data in the current experiment due to the use of three within-subject experimental conditions (alcohol, placebo, control). Planned comparisons using *t*-tests were used to assess any significant interactions further. In order to investigate whether the effect of experimental condition on any of the cognitive tasks mediated the alcohol priming effect the ANOVA conducted on the taste test data was repeated with additional covariates (i.e. a series of ANCOVAs were conducted). Covariates were determined by taking the variables that were significantly affected by the experimental condition(s) and creating a priming effect score, which was derived by subtracting values from the control condition from the alcohol condition. This value was added as a covariate to the taste test ANOVA. If the inclusion of a covariate eliminated the main effect of condition on the taste test this is indicative of the covariate at least partially mediating the alcohol priming effect. Similarly, in order to investigate whether alcohol-induced increases in impulsivity and/or impairments in executive cognitive functioning were partially mediating any alcohol-induced increase in the automatic responses to alcohol-related cues, ANCOVAs were run on the measures of cognitive bias that were significantly affected by condition. The covariates for these ANCOVAs were the priming effect scores of measures of behavioural impulsivity and/or executive cognitive functioning (if affected by the experimental condition). As the mediation analysis consists of repeated testing with different covariates an intermediated correction was utilised, with $p<.01$ being deemed significant (Sankoh et al., 1997). A full bonferroni correction was not utilised as performance on the tasks was intercorrelated.

6.4. Results

Sample Characteristics

Participants consumed an average of 39.00 (± 17.29, range 15-77) UK units of alcohol (1 unit = 8g alcohol) in the last week. Average AUDIT scores were above the cut off for hazardous drinking (16.06 ±5.32).

Self-reports and Breath Alcohol Levels (See Table 6.1).

Subjective intoxication scales. VAS ratings for 'lightheaded', 'irritable', 'stimulated', 'alert', 'relaxed', and 'contented' were analysed using a series of repeated measures ANOVAs, with factors of session (alcohol, placebo, control) and time (pre-drink, post-drink, and end of session). A session by time interaction was found for lightheaded ($F(4,120) = 42.03$, $p < .001$, $\eta_p^2 = .74$). Post hoc tests revealed that in the alcohol session there was a significant increase between pre-drink and post-drink ($t(30) = -9.13$, $p < .001$) and pre-drink and end of session ($t(30) = -8.34$, $p < .001$), although post-drink and end of session did not differ from each other ($p > .1$). In the placebo condition participants also reported feeling more lightheaded at post-drink compared to pre-drink ($t(30) = -6.24$, $p < .001$) and at end of session compared to pre-drink ($t(30) = -4.28$, $p < .001$) but again, there was no difference between time post-drink and end of session ($p > .1$). No change in lightheaded ratings was found in the control condition. Light headedness was found to be higher at post-drink ($t(30) = 6.70$, $p < .001$) and end of session ($t(30) = 8.06$, $p < .001$) in the alcohol compared to the placebo session.

A session by time interaction was also found for alert ($F(4,120) = 8.81$, $p < .001$, $\eta_p^2 = .22$). In the alcohol session there was a significant decrease between pre-drink and post-drink ($t(30) = 6.19$, $p < .001$) and pre-drink and end of session ($t(30) = 6.17$, $p < .001$), although post-drink and end of session did not differ from each other ($p > .1$). The placebo condition also produced significant decreases in alert ratings, both pre-drink and post-drink ($t(30) = 2.60$, $p = .015$) and pre-drink and end of session

($t(30) = 2.25$ p = .032), although post-drink and end of session did not differ from each other (p > .1). No significant changes were reported in the control condition. Alert ratings were not significantly different post-drink (p > .1) or at end of session (p > .1) in the alcohol compared to the placebo session.

Finally, a significant session by time interaction was found for contented ($F(4,120) = 3.01$, p = .04, $\eta_p^2 = .09$). In the alcohol condition there was a trend towards increased contented ratings between pre-drink and end of the session ($t(30) = -1.93$, p = .063), although pre-drink and post-drink (p > .1) and post drink and end of session (p > .1) did not significantly differ. In the placebo condition participants reported feeling significantly less contended post-drink compared to the end of session ($t(30) = 2.14$, p = .04), although pre-drink and post-drink (p > .1) and pre-drink and end of session (p > .1) did not significantly differ. No significant changes in 'Contented' ratings were observed in the control condition (p > .1). Contended ratings were higher at end of session in the alcohol compared to the placebo condition, ($t(30) = 3.56$ p = .001) , although post-drink ratings did not significantly differ (p > .1). Data are shown in Table 6.1.

Perceived alcohol content. A 3 way repeated measures ANOVA was used to assess the estimated number of alcohol units in the drinks in the different sessions (alcohol, placebo and control). A significant main effect of session was found ($F(2,60) = 229.08$, p < .001, $\eta_p^2 = .88$). Participants estimated that they consumed more alcohol in the alcohol session compared to the placebo session ($t(30) = 10.49$, p < .001) and in the placebo session compared to the control session ($t(30) = 10.45$, p < .001). Data are shown in Table 6.1.

Craving. A 3 x 3 repeated measures ANOVA was used to check differences in mean DAQ scores between sessions (alcohol, placebo and control) at the three time points within test sessions (pre-drink, post-drink, and end of session). There was a significant session by time interaction ($F(4,120) = 8.13$, p < .001, $\eta_p^2 = .21$). After alcohol, post hoc comparisons revealed a significant increase in craving between pre-

drink (2.63) and post-drink (3.14; $t(30) = -3.74$, $p < .001$) and between pre-drink and end of session (3.25; $t(30) = -4.61$, $p < .001$), although post-drink and end of session did not differ from each other ($p > .1$). For placebo, craving increased between pre-drink (2.32) and post-drink (2.78; $t(30) = -2.30$, $p = .031$), but craving then declined from post-drink to the end of session (2.11; $t(30) = 2.09$, $p = .045$); pre-drink and end of session did not differ ($p > .1$). There were no changes in craving after administration of the control drink ($ps > .1$). Contrasts between different drinks revealed that craving was significantly higher post-drink after alcohol compared to placebo ($t(30) = 2.63$, $p = .013$) and after placebo compared to the control drink ($t(30) = 2.73$, $p = .01$). End of session craving was also significantly higher after alcohol compared to placebo ($t(30) = 5.88$, $p < .001$) and after placebo compared to the control drink ($t(30) = 2.42$, $p = .022$). Data are shown in Table 6.1.

Breath alcohol concentration. All participants had a BAC of 0 g/100ml when assessed at the beginning of all sessions. In the placebo and control sessions all other BAC readings were 0 g/100ml. In the alcohol session, the mean BAC was 0.89 g/100ml (±0.16) at the post-drink assessment and this increased to 0.96 g/100ml (±0.11) at the end of the session (although this increase was not statistically significant, $t(30) = 1.53$, $p > .1$).

Cognitive tasks and beer consumption in the taste test

For descriptive statistics of all task dependent variables shown separately for each condition see Table 6.2.

SRC task (See Figure 6.1)

A repeated measures ANOVA was used to analyse the SRC task reaction times, with session (alcohol, placebo, control) and SRC task block (approach alcohol, avoid alcohol) as within subjects variables. The interaction between session and block was

significant ($F(2,60) = 3.59$, p = .034, $\eta_p^2 = .11$). Planned comparisons revealed that reaction times in the approach alcohol block were faster than reaction times in the avoid alcohol block in the alcohol and placebo sessions (alcohol; $t(30) = -3.59$, $p < .001$; placebo; $t(30) = -3.89$, $p < .001$) there was also a trend in this direction in the control session ($t(30) = -1.66$, p = .054). To investigate the significant session by task block interaction, 'approach bias' scores were calculated separately for each session, by subtracting reaction times during the approach alcohol block from reaction times during the avoid alcohol block, such that higher scores indicate speeded approach elicited by alcohol-related cues. Paired samples *t*-tests revealed that approach bias scores were larger in both the alcohol ($t(30) = 1.89$, p = .035) and placebo sessions ($t(30) = 2.48$, p = .009), compared to the control session. However, approach bias scores in the alcohol and placebo sessions did not differ from each other ($t(30) = -0.66$, $p > .1$).

Visual probe task: Reaction times

A repeated measures ANOVA was used to analyse the visual probe task reaction times, with session (alcohol, placebo, control) and trial type (congruent, incongruent) as within subjects variables. There was no main effect of session ($F(2,60) = 1.62$, $p > .1$, $\eta_p^2 = .05$), trial type ($F(1,30) = 1.05$, $p > .1$, $\eta_p^2 = .03$), or an interaction between trial and session ($F(2,60) = 0.26$, $p > .1$, $\eta_p^2 = .01$).

Visual probe task: Gaze dwell times

A repeated measures ANOVA was used to analyse the visual probe gaze dwell times, with session (alcohol, placebo, control) and picture type (alcohol, control) as within subjects variables. There was a significant main effect of session ($F(2,60) = 3.92$, p = .025, $\eta_p^2 = .12$), planned comparisons revealed this was due to participants holding their gaze on the location of either picture for significantly longer in the

placebo (1236.78 ±393.38) compared to the alcohol (1116.43 ±428.05; $t(30)$=-4.163, $p < .001$) and control condition (1114.78 ±445.80; $t(30) = -2.134$, $p = .041$). There was however no significant difference in overall gaze dwell times in the alcohol compared to the control condition ($t(30) = 0.03$, $p > .1$). There was also a significant effect of picture type ($F(1,30) = 5.98$, $p = .021$, $\eta_p^2 = .17$), this was due to significantly longer gaze dwell times on alcohol (893.68 ±318.42) compared to control pictures (840.31 ±279.62) regardless of condition ($t(30) = 2.45$, $p = .021$). There was however no significant interaction between picture type and session ($F(2,60) = 0.29$, $p > .1$, $\eta_p^2 = .01$).

Delay Discounting

A repeated measures ANOVA was used to analyse the AUC values from the delay discounting task, with session (alcohol, placebo, control) as the within subjects factor. There was no significant main effect of session ($F(2,60) = 2.08$, $p > .1$, $\eta_p^2 = .15$).

Cued Go/No-Go

A repeated measures ANOVA was used to analyse the proportion of errors on No-Go trials, with condition (alcohol, placebo, control) and cue type (No-Go cue, Go cue) as within subjects variables. There was no significant main effect of session, ($F(2,54) = 0.23$, $p > .1$, $\eta_p^2 = .01$). There was however, a significant main effect of cue, ($F(2,54) = 43.80$ $p < .001$, $\eta_p^2 = .62$). This was the result of a significantly greater proportion of errors following 'Go' cues (0.70 ±0.57) than following 'No-Go' cues (0.06±0.05; $t(27) = 6.10$, $p < .001$) regardless of the experimental condition. There was no significant interaction between condition and cue ($F(2,54) = 1.18$ $p > .1$, $\eta_p^2 = .04$).

COWAT (See Figure 6.2)

A repeated measures ANOVA was used to analyse the number of switches produced in the COWAT, with session (alcohol, placebo, control) as the within subjects factor. There was a significant main effect of session ($F(2,60) = 14.41$, $p < .001$, $\eta_p^2 = .32$), as participants produced significantly fewer switches in the alcohol session compared to both placebo ($t(30) = -3.74$, $p < .001$) and control sessions ($t(30) = -5.42$, $p < .001$). There was no significant difference between the number of switches in the placebo and control sessions ($t(30) = -1.33$, $p > .1$).

Beer consumption during bogus taste test (See Figure 6.3)

A repeated measures ANOVA was used to analyse beer consumed (as a percentage of total fluid consumed), with session (alcohol, placebo, control) as the within subjects factor. There was a significant main effect of session ($F(2,60) = 12.62$, $p < .001$, $\eta_p^2 = .30$). Participants consumed significantly more beer in the alcohol session compared to both placebo ($t(30) = 4.46$, $p < .001$) and control sessions ($t(30) = 3.95$, $p < .001$). However, the amount of beer consumed did not differ between placebo and control sessions ($t(30) = 1.26$, $p > .1$).

Mediation of effects on beer consumption by the SRC and COWAT.

In order to assess which alcohol-induced changes in cognition are associated with the alcohol priming effect, 'priming effect scores' for SRC bias and COWAT switches were added as a covariate to the repeated measures ANOVA that analysed beer consumed in the taste test. Only SRC bias and COWAT switches priming effect scores were used as covariates as these variables were the only two that were significantly affected by the alcohol prime and/or placebo condition.

The beer consumption data was reanalysed using a repeated measures ANOVA, with a within-subjects factor of session (alcohol, control), which revealed a highly significant main effect of session ($F(1,30) = 15.62$, $p < .001$, $\eta_p^2 = .34$). This analysis was repeated with the addition of the SRC bias priming effect score (approach bias

score in alcohol session minus approach bias score in control session) as a covariate. The main effect of session on beer consumption remained statistically significant ($F(1,29) = 12.74$, $p < .001$, $\eta_p^2 = .31$). However, when the analysis was repeated with the COWAT priming effect score (COWAT switches in control session minus COWAT switches in alcohol session) as a covariate, the main effect of session on beer consumption was no longer statistically significant ($F(1,29) = 2.86$, $p > .01$, $\eta_p^2 = .09$). These results suggest that the alcohol priming effect was mediated by alcohol induced impairments in executive cognitive functioning, but not by the alcohol-induced increase in automatic approach tendencies elicited by alcohol-related cues.

Mediation of the effects of alcohol on cognitive bias by the COWAT

In order to investigate whether the effects of alcohol dose on SRC bias scores (i.e. the increase after the placebo that was maintained following the alcohol prime) were mediated by effects of the experimental condition on executive cognitive functioning the SRC bias scores were re-analysed using the COWAT priming effect score (as described above) as a covariate.

The re-analysis of SRC bias scores used a repeated measure ANOVA with a within subjects factor of condition (alcohol, control). Then the alcohol COWAT priming effect score (COWAT switches in control session minus COWAT switches in alcohol session) was added as a covariate. There was the expected main effect of condition on SRC bias scores ($F(2,60) = 3.59$, $p = .034$, $\eta_p^2 = .11$). When the COWAT priming effect score was added to the ANOVA the effect of condition on SRC bias was no longer statistically significant $F(2,58) = 0.35$, $p > .1$, $\eta_p^2 = .01$). This suggests that the effect of condition on changes in SRC bias was (at least) partially mediated by impairments in executive cognitive functioning as measured by the COWAT.

Table 6.1: Between and within session comparisons of subjective intoxication, alcohol craving and unit estimates (values shown are mean ±SD).

	Alcohol			Placebo			Control					
Variable	Time 1	Time 2	Time 3	Time 1	Time 2	Time 3	Time 1	Time 2	Time 3	Session (F)	Time (F)	Interaction (F)
DAQ Mean score	2.63 (0.93)	3.14 (1.08)	3.25 (1.02)	2.32 (1.87)	2.78 (1.02)	2.11 (1.16)	2.50 (0.94)	2.48 (0.99)	2.38 (0.93)	9.53**	4.91*	3.14*
SIS Light-headed	3.75 (7.09)	46.97 (25.22)	44.64 (27.23)	7.42 (11.98)	21.48 (18.38)	18.81 (20.45)	4.10 (5.19)	4.00 (5.73)	4.52 (7.32)	48.16**	59.70**	42.03**
SIS Irritable	6.97 (8.99)	6.94 (10.51)	9.35 (15.18)	7.48 (9.07)	7.06 (9.52)	13.42 (14.21)	7.42 (14.72)	6.71 (10.90)	6.87 (10.22)	1.14	2.80	1.36
SIS Stimulated	34.06 (22.23)	34.84 (21.88)	37.23 (23.81)	34.97 (22.05)	38.90 (18.61)	34.19 (21.81)	34.90 (23.34)	36.77 (22.65)	35.90 (22.00)	0.02	0.37	0.70
SIS Alert	60.87 (15.01)	38.00 (19.03)	35.52 (23.01)	49.84 (17.50)	42.00 (15.94)	40.32 (20.78)	56.23 (15.92)	55.61 (19.72)	52.51 (21.61)	5.82**	22.37**	8.81**
SIS Relaxed	59.53 (18.54)	65.13 (21.36)	66.90 (23.21)	59.81 (22.03)	64.03 (19.31)	62.29 (21.67)	58.32 (19.20)	59.00 (15.94)	58.10 (15.38)	1.94	1.57	1.05
SIS Contented	63.26 (15.60)	65.39 (20.45)	68.03 (17.47)	58.71 (17.25)	63.32 (17.43)	56.23 (18.52)	62.68 (17.97)	61.35 (15.72)	59.39 (18.87)	4.55*	0.90	3.01*
Unit estimate	-	-	5.23 (1.28)	-	-	2.26 (1.20)	-	-	0.0 (0.0)	229.08**	-	-

DAQ mean score = Desires for alcohol questionnaire mean score, values range from 7 (minimum) to 98 (maximum). SIS = Subjective Intoxication Scale, values range from 0 (minimum) to 100 (maximum) for all subscales. Unit estimate = Participants estimate as to the number of U.K. units of Vodka they consumed at the start of the experimental session (one unit = 25ml of 37.5% ABV vodka).

**$p < .001$, *$p < .05$

Table 6.2: Between session comparisons of all cognitive tasks and beer consumed in the taste test (values shown are mean ±SD).

Variable	Alcohol	Placebo	Control
SRC Bias (ms)	45.66(60.62)	38.53(58.53)*	10.44 (45.90)
VP reaction time bias (ms)	-1.41(28.68)	-4.00 (22.06)	-2.54(26.10)
VP gaze dwell time bias (ms)	62.30(140.26)	27.71(137.37)	29.34(100.55)
Delay discounting (AUC)	0.68(0.29)	0.67(0.28)	0.70(0.27)
COWAT Switches	26.61 (7.13)**	31.07(7.87)	33.21(7.88)
Go-No-Go Errors (proportion)	0.24 (0.23)	0.23(0.18)	0.24 (0.23)
Beer consumed (%)	53.16 (28.81)**	36.47(19.00)	32.40(21.47)

SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. VP reaction time bias (ms) = reaction time to incongruent probes minus reaction times to congruent probes, higher values represent increased attentional bias. VP gaze dwell time bias (ms) = Dwell times on Group pictures minus gaze dwell times on control pictures, higher values indicate greater attentional bias. Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. COWAT Switches = Number of switches between word groups on the controlled oral word association test, higher scores are indicative of better executive cognitive functioning. Go-No-Go error (proportion) = Proportion of commission errors on following a Go cue on No-Go trials in the Cued Go/No-Go task, higher scores are indicative of increased impulsive responding. Beer consumed (%) = Percentage of beer out of total liquid consumed in the taste test. *p<.05, **p<.001

Figure 6.1: Mean reaction times (milliseconds) for approach alcohol and avoid alcohol blocks of the SRC task. Reaction times are shown separately for alcohol, placebo and control conditions. (Values are mean ±SEM). *p<.001

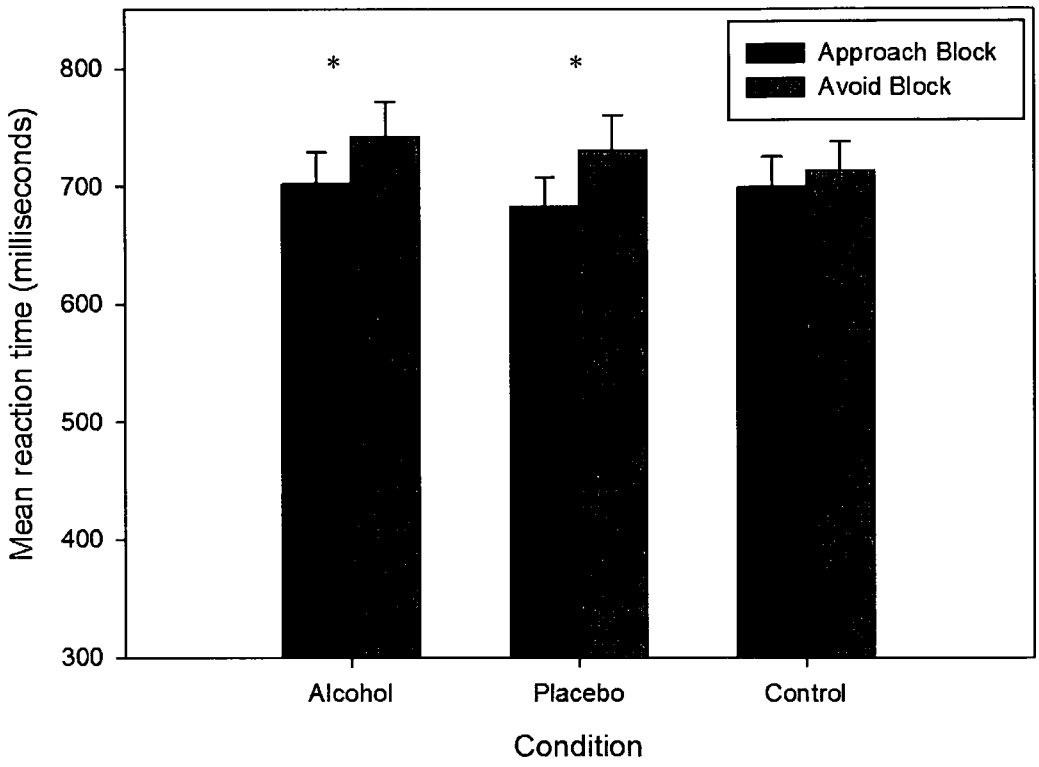


Figure 6.2: Total number of switches made on the COWAT. Switches are shown separately for alcohol, placebo and control conditions. (Values are mean ±SEM). *p<.001

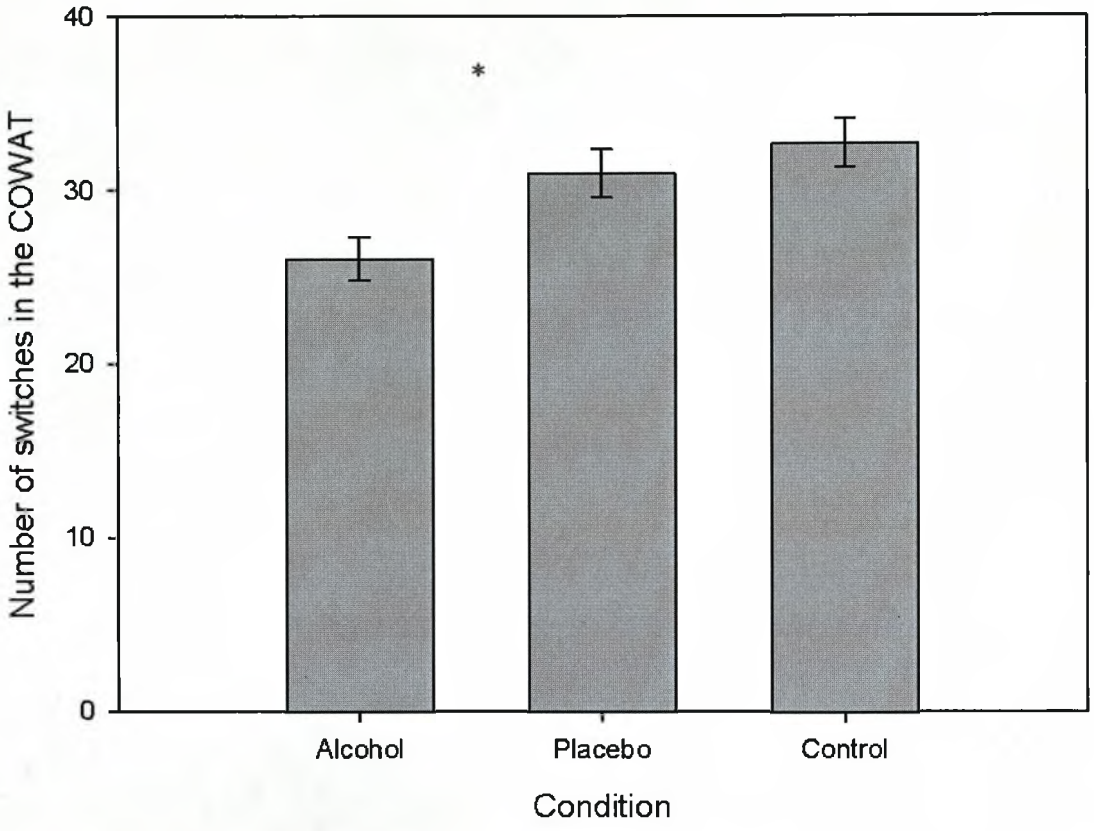
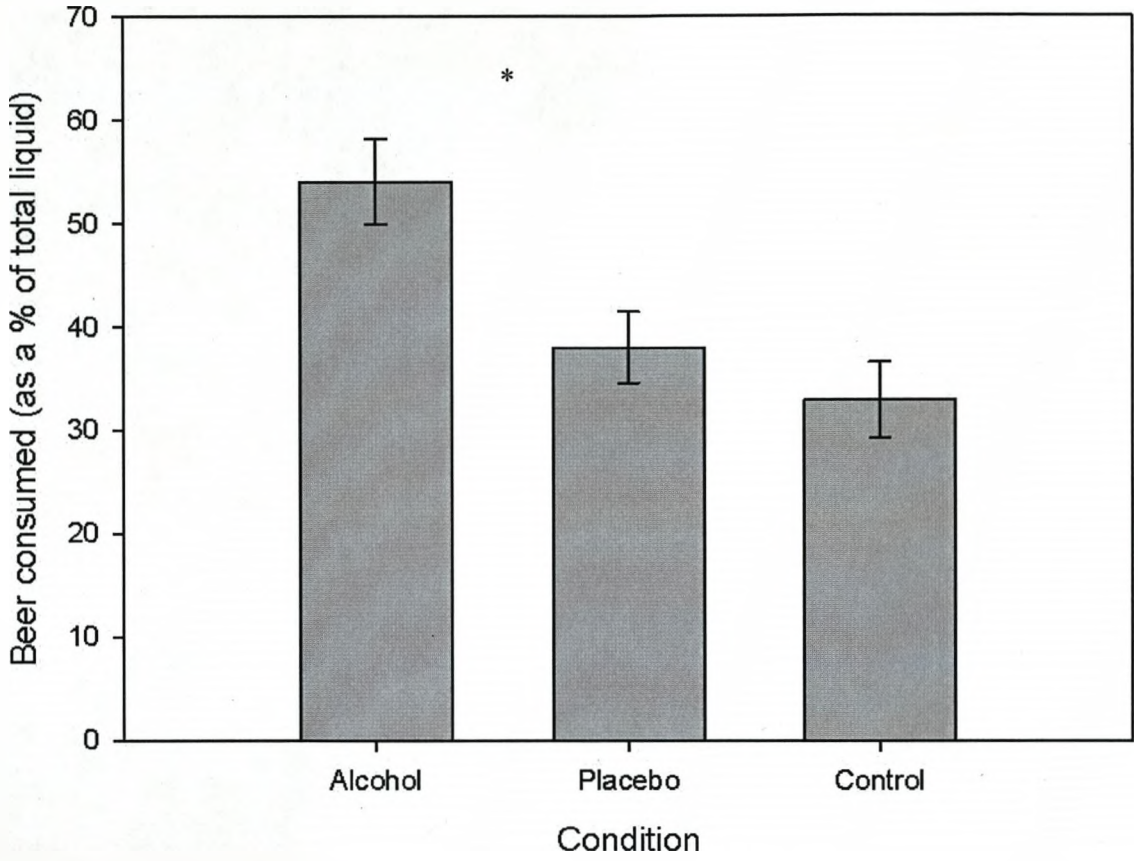


Figure 6.3: Beer consumption as a percentage of total fluid during the taste test, shown separately for alcohol, placebo and control conditions. (Values are mean ±SEM). *p<.001



6.5. Discussion

The current study investigated the effects of 0.65 g/kg alcohol and the anticipated effects of alcohol on attentional bias, automatic approach tendencies elicited by alcohol-related stimuli, measures of behavioural impulsivity and executive cognitive functioning. Furthermore, the association between these measures and the effect of alcohol on ad-lib drinking was also investigated. It was hypothesised that the alcohol prime would strengthen attentional bias and automatic approach tendencies and increase behavioural measures of impulsivity/impair executive function, and that all of these changes in cognitive processes would be associated with increased beer consumption. In addition, it was hypothesised that there would be a drug-like responses to the placebo in the visual probe and SRC task and a drug-opposite response to the placebo in the impulsivity/executive cognitive function tasks. Regarding performance on the SRC task, it was found that automatic approach tendencies elicited by alcohol-related cues were strengthened in the placebo compared to the control session, but 0.65 g/kg alcohol did not significantly increase approach bias beyond that found in the placebo session. Furthermore, approach tendencies were not associated with increased alcohol consumption during the bogus taste test. There was no effect of alcohol or placebo on attentional bias (assessed by reaction times or gaze dwell times). Surprisingly, there was also no effect of alcohol on performance on the cued Go/No-Go task, although the alcohol prime significantly impaired phonemic fluency, with this impairment being associated with increased beer consumption in the taste test. Impulsive decision making was however unaffected by the alcohol prime or placebo. Finally, there was evidence that increases in automatic approach responses were mediated by impairments in executive functioning (phonemic fluency).

'Approach bias' – the tendency to categorise alcohol-related pictures more quickly when required to make a symbolic approach movement towards, rather than a symbolic avoidance movement away from the pictures - was significantly larger after administration of alcohol and placebo compared to the control drink, although alcohol and placebo did not differ from each other. This finding may explain the

absence of an effect of alcohol on the SRC task as reported by Schoenmakers et al. (2008), who compared alcohol with placebo but did not include a further control drink which participants knew contained no alcohol. Interestingly, Farris and Ostafin (2008) reported an alcohol priming effect on a different measure of automatic alcohol approach tendencies, but their study did not use either a placebo or control condition. Taken together, these results suggest that although automatic approach tendencies can be increased by expectation of an alcoholic drink, the pharmacological effects of either low (0.3 g/kg; Schoenmakers et al., 2008) or higher (0.65 g/kg; present study) doses of alcohol do not contribute to this effect. This finding highlights the importance of the placebo effect on automatic approach tendencies. This indicates that future studies, which assess the effects of acute alcohol on automatic approach tendencies, should compare alcohol with both placebo and control beverages, in an attempt to replicate the present findings.

Although previous research (Adams et al., 2011; Duka & Townshend 2004; Schoenmakers et al., 2008) has found that alcohol primes increase attentional bias among social and heavy drinkers, these studies have utilised lower priming doses (0.4 g/kg or less). Indeed, studies utilising higher priming doses have generally not found subsequent increases in attentional bias (Duka & Townshend 2004; Miller & Fillmore 2011). This supports the assertion that there is not a linear relationship between priming doses of alcohol and attentional bias, with lower doses of alcohol increasing, and moderate to high doses of alcohol (in access of 0.6 g/kg) not increasing, attentional bias. It is notable that in all conditions of this experiment there was evidence of attentional bias (as measured by gaze dwell times), which suggests that moderate priming doses do not reduce attentional bias due to satiety, as suggested by Duka and Townshend (2004). Futhermore, there was no evidence of a drug-like response to the aniticipated effects of alcohol in the visual probe task. Taken together, these results indicate that moderate alcohol primes do not increase the automatic processing of alcohol-related cues, and that the anticipated effects of alcohol increase the accessability of automatic approach responses but not attentional bias. This suggests that although both these processes are hypothesised to be the result of the same neurological sensitisation process, they are differentially affected by alcohol and anticipated effects of alcohol.

The finding that phonemic fluency was significantly affected by a moderate alcohol dose adds to the growing evidence base demonstrating that acute alcohol administration impairs executive cognitive functioning. Although the majority of current research has focused on impairments in inhibitory control (Marczinski et al., 2005; Marczinski et al., 2007), the present results suggest that other aspects of executive functioning are equally impaired by moderate to high doses of alcohol. Previous research has also found working memory (Balodis et al., 2007) and mental set shifting (Guillot et al., 2010) to be impaired by acute alcohol administration. Combined with the results from the current study, this suggests that all domains of executive functioning may be impaired by alcohol. Indeed, it may be that working memory should be targeted more for investigation as phonemic fluency tasks have recently been shown to measure updating working memory and working memory has been suggested to be the central executive cognitive function (e.g. Friedman & Miyake 2012). The lack of a significant placebo effect on phonemic fluency (i.e. no difference between placebo and control sessions) suggests that, unlike approach bias, the effect of alcohol on executive functioning is the result of the direct pharmacological effects of alcohol consumption, rather than the anticipated effects. In addition, unlike performance on pursuit rotor tasks (Fillmore et al., 1994), there was no evidence of improved performance following the placebo which would be expected if participants tried to compensate for anticipated alcohol-induced deficits in performance. Interestingly, there was evidence that phonemic fluency mediated the effects of dose on SRC performance, inasmuch as that when deficits in executive cognitive functioning were controlled for, the effect of dose on SRC bias also disappears. This supports dual process models of addiction as when controlled processes were impaired there was a subsequent increase in automatic approach responses. However, although alcohol-induced impairments in executive cognitive function were found to mediate the increase in the accessibility of automatic alcohol-approach responses this did not mediate the priming effect as predicted by Field et al. (2010).

The finding that the alcohol prime had no effect on the proportion of inhibition errors in the Cued Go/No-Go task was unexpected. A substantial body of evidence shows that this task is particularly sensitive to moderate priming doses of alcohol (e.g.

Marczinski et al., 2005; Marczinski et al., 2007), so it is unclear why this effect was not replicated in the present study. One possible explanation is that the sample in the current study consisted of particularly heavy drinkers who may have responded differently to the prime, although the aforementioned studies do not give comparable data for this to be confirmed. It is likely that different patterns of drinking in the UK compared to the US contributed to the unexpected null findings in the current study.

The findings regarding delay discounting were less surprising; generally impulsive decision making, as assessed by non-experiential delay discounting, is not consistently affected by priming doses of alcohol, with most studies showing no effects (e.g. Reynolds et al,. 2006b; Richards et al., 1999), and another study (Ortner et al., 2003) demonstrating a decrease in impulsive decision making. These results suggest that delay discounting is relatively resilient to the effects of priming doses of alcohol. Furthermore, the anticipated effects of alcohol had no effects on impulsive decision making.

In line with other research (e.g. Rose & Grunsell 2008; de Wit & Chutuape 1993) alcohol administration significantly increased self-reported craving and alcohol-seeking behaviour, as measured by the taste test. This suggests the pharmacological, rather than anticipated effects of alcohol, are primarily responsible for alcohol priming effects on the motivation to drink. However, it was also found that the placebo produced a significant increase in alcohol craving, although this increase was small and transient compared to the increase seen after alcohol administration. Previous studies have generally shown small but non-significant increases in craving following placebo administration (e.g. Rose & Duka 2006; Schoenmakers et al., 2008). In contrast to Marlatt's (1973) seminal study there was no evidence that increased voluntary beer consumption was the result of the anticipated, rather than pharmacological, effects of alcohol. One explanation for this is that the alcohol-like effect of the placebo on self reported craving in the current study was relatively short lived. In Marlatt et al., (1973) participants completed the taste test immediately after the placebo; in the current study participants completed the battery of cognitive tasks before completing the taste test, and at this point the placebo-induced increases in craving had dissipated.

The effect of alcohol on the taste test was associated with the effect of alcohol on executive function, but not with the effect of alcohol on automatic approach tendencies. Similar to the findings of Weafer and Fillmore (2008) there was an association between impairments in executive cognitive function and beer consumption in a taste test. This suggests that disinhibited alcohol consumption during a binge may not specifically be the result of impairments in inhibitory control but due to global impairments in executive function. Although much of the previous research has concentrated upon inhibitory control, the current study is, to my knowledge, the first to show that impairments in other measures of executive function (specifically, phonemic fluency) may also be associated with disinhibited drinking. This indicates that future research should investigate what other aspects of executive functioning are associated with the alcohol priming effect. As working memory (Balodis et al., 2007) and switching (Guillot et al., 2010) tasks have both been shown to be sensitive to moderate alcohol primes, investigations concentrating on the role of these processes in the alcohol priming effect could offer greater insight into the specific neurocognitive mechanisms associated with disinhibited drinking. Increasing awareness of how alcohol consumption impairs behavioural control may help individuals better understand how plans to drink moderately will be adversely affected a few hours after drinking commences, increasing the likelihood of an unplanned binge.

6.6. Chapter summary

The current study indicates that there is a differential effect of alcohol primes and the anticipated effects of alcohol on automatic cognitive processes, impulsivity, and executive cognitive function. Automatic approach responses were increased by the placebo with this increase being maintained following the alcohol prime, while attentional bias was not increased by either alcohol or placebo. Furthermore increases in automatic approach responses were mediated by impairments in phonemic fluency. Although both measures of behavioural impulsivity were not affected by the pharmacological or the anticipated effects of alcohol, there was a

significant (detrimental) effect of the alcohol prime on phonemic fluency. With regard to the alcohol priming effect, there was the expected increase in alcohol seeking following the alcohol prime. In addition, the alcohol prime and the anticipated effects of alcohol increased self report craving. Importantly, the increase in alcohol seeking during the taste test was following the alcohol prime (not after the placebo) and this increase was mediated by impairments in phonemic fluency, but not automatic approach tendencies. These results have important implications for our understanding of the specific cognitive mechanisms that may mediate the ability of alcohol to prime drinking behaviour.

Chapter 7:

Ego Depletion Increases Ad-Lib Alcohol Consumption:

Investigating Cognitive Mediators and Moderators

A briefer version of this chapter has been published as- Christiansen P, Cole JC, Field M (2011) Ego depletion increases ad-lib alcohol consumption: Investigating cognitive mediators and moderators. *Experimental and Clinical Psychopharmacology*. doi: 10.1037/a0026623. See Appendix 16

7.1. Abstract

When self-control resources are depleted ("ego depletion"), alcohol-seeking behaviour becomes closely associated with automatic alcohol-related processing biases (e.g. Ostafin et al., 2008). The current study aimed to replicate and extend these findings, and also to investigate whether the effects of ego depletion on drinking behaviour would be mediated by temporary impairments in executive function or increases in impulsivity. Eighty heavy social drinkers (46 female) initially completed measures of automatic approach tendencies (stimulus response compatibility (SRC) task) and attentional bias (visual probe task) elicited by alcohol-related cues. Participants were then exposed to either an ego depletion manipulation or a control manipulation, before completing a bogus taste test in order to assess ad-lib alcohol consumption. In a subsequent testing session, the effects of the ego depletion manipulation (vs. control manipulation) on three aspects of controlled processes (inhibitory control, phonemic fluency, and delay discounting) were examined. Results indicated that the ego depletion manipulation increased ad-lib drinking, relative to the control manipulation. Automatic approach tendencies, but not attentional bias, predicted ad-lib drinking, although this effect was not moderated by ego depletion. Ego depletion had inconsistent effects on measures of executive function and impulsivity, and none of these measures mediated the effect of ego depletion on ad-lib drinking. However, the effect of ego depletion on ad-lib drinking was mediated by self-reported effort in suppressing emotion and thoughts during the manipulation. Implications for the effects of self-control strength on drinking behaviour, and cognitive mediators of these effects, are discussed.

7.2. Introduction

Humans display a remarkable capacity to inhibit unhealthy urges. For example, despite temptation to eat chocolate and respond to feelings of hunger a dieter may choose not to eat in order to achieve a long term goal of losing weight and improving health. Likewise, a newly abstinent smoker may have a strong desire for nicotine, but may be able to resist these urges to maintain the long-term goal of abstinence. In a variety of domains, humans are able to direct behaviour towards future goals at the expense of satisfying immediate urges and desires.

Despite this capacity for self-regulation there are numerous examples in which self control lapses. Risky sexual behaviour, overeating, drug and alcohol abuse, and aggression are all consequences of a failure to regulate behaviour (Baumeister et al., 1993; Quinn & Fromme 2010; Stinson et al., 2008). Indeed, a large number of social and health problems are likely to have failures in self regulation at their core (Baumeister & Heatherton 1996). Recent research into self regulation has focused on the "strength" model (Baumeister et al., 1998). This model proposes that self control resources are finite; if demands on self control are minimal then an individual will have the resources available to successfully regulate their behaviour. If demands on self control are very high and/or demands have been maintained over a prolonged period then self control resources will be diminished and subsequent self control will be impaired. This state of depleted self control resources has been termed 'ego depletion' (Baumeister et al., 1998; Muraven et al., 1997). Recent investigations have shown that exerting self control reduces the ability to regulate behaviour in subsequent self control tasks, even when the domains of the self control tasks are different. For example, Stucke and Baumeister (2006) found that controlling urges to eat reduced the ability to regulate aggressive behaviour in response to negative comments by an experimenter. Both Hofmann et al. (2007) and Zyphur et al. (2007), reported increased candy consumption after ego depletion manipulations (for a recent review see Hagger et al., 2009). With regard to alcohol consumption Muraven et al. (2002) found that ego depletion resulted in increased beer consumption in a bogus taste test, and this effect was seen despite a financial incentive to limit

consumption. In addition, Muraven et al. (2005) investigated ego depletion in naturalistic settings, by assessing participants' daily self control demands and alcohol consumption over a three week period using ecological momentary assessment. The primary finding was that individuals were more likely to drink in excess of their self imposed limits on days when they had experienced a higher than average level of self-control demands.

Despite this emerging evidence, the specific mechanisms that underlie effects of ego depletion are less clear. A recent meta-analysis found that ego depletion effects were associated with the degree of self-reported effort that was exerted during self-control tasks, as predicted by the strength model of self control (Hagger et al., 2010). This suggests that participants have some awareness of the amount of effort expended during depleting tasks, with increased effort resulting in reduced self-control resources. However, recent studies suggest that the perception of being in a state of depleted self-control, rather than actual resource depletion, accounts for the ego depletion effect. Indeed, Ackerman et al. (2009) found that simulating self control results in self control depletion, while observing self control in others actually increases self control in the observer. Manipulating beliefs about the availability of self control resources can also protect against ego depletion effects. For example if individuals are primed to believe that self control resources are available (Clarkson et al., 2010), or that self control is unlimited (Job et al., 2010), they are impervious to ego depletion manipulations. Furthermore other factors, such as the unconscious priming of persistence (Alberts et al., 2007) can mitigate against the effects of ego depletion.

Another possibility is that the depletion of self control resources is mediated by fatigue in specific brain areas involved in behavioural regulation, most likely the pre-frontal cortex (Gailliot & Baumeister 2007). The taxing of self-control resources may temporarily impair aspects of executive cognitive function involved in behavioural regulation, such as inhibitory control, set-shifting and working memory (Miyake et al., 2000). Related to this effect, demands on self-control resources may temporarily increase components of behavioural impulsivity, including disinhibition and impulsive decision-making (de Wit 2009). There is some limited evidence to suggest that ego depletion does have a detrimental effect on executive function, e.g.

decreases in verbal and figure fluency (Schmeichel et al., 2006), although the effects of ego depletion on behavioural measures of impulsivity have not yet been studied.

Dual process models such as Deutsch and Strack (2006) and Wiers et al. (2007) provide a theoretical framework for understanding the effects of ego depletion on alcohol consumption. These models argue that alcohol consumption is determined by the interplay between automatic alcohol-related processing biases (hereafter referred to as cognitive biases), on the one hand, and controlled processes, such as executive cognitive functioning, on the other. The strength model (Baumeister et al., 1998), states that after self-control resources have been depleted the ability to control subsequent behaviour is diminished; ego depletion should therefore lead to a state in which behaviour is strongly influenced by cognitive biases. Theoretically, cognitive biases develop because alcohol-related stimuli gain incentive-motivational properties after chronic heavy drinking (Robinson & Berridge, 2001), which results in increased attentional allocation and initiation of approach responses in response to alcohol-related cues (Franken 2003). For example alcohol cues capture and hold the attention of alcoholics and heavy social drinkers (Field et al., 2007a; Stetter et al., 1995; Townshend & Duka, 2001; for a recent review, see Field & Cox, 2008). Heavy drinkers also direct automatic approach responses towards alcohol cues as revealed by the stimulus response compatibility (SRC) task, (study four of the current thesis, Field et al., 2011; Field et al., 2008). Importantly, both alcohol attentional bias (Field & Eastwood, 2005; Schoenmakers et al., 2010) and automatic alcohol approach tendencies (Wiers et al., 2011; Wiers et al., 2010) may have causal effects on subsequent drinking behaviour. As predicted by dual-process models, the association between cognitive biases and drinking behaviour is stronger in participants with poor executive cognitive functioning, specifically working memory (Thush et al., 2008) and inhibitory control (Houben & Wiers, 2009b). If ego depletion affects behaviour because it fatigues areas of the brain associated with behavioural control (as argued by Gailliot & Baumeister 2007) then we would expect ego depletion to increase the association between cognitive biases and alcohol consumption. Indeed, recent studies have demonstrated that cognitive bias, assessed with an implicit association test, predicts ad-lib drinking after ego depletion but not after a control manipulation (Friese et al., 2008; Ostafin et al., 2008). This suggests

that ego depletion facilitates the ability of automatic processes to control behaviour. Dual process models of addiction would suggest that ego depletion is therefore mediated by impairments in controlled processes. If this is the case ego depletion would increase behavioural impulsivity and impair executive cognitve functioning thereby faciliating the ability of automatic cognitive processes to control behaviour.

The primary aim of this experiment was to extend the findings reported by Friese et al. (2008) and Ostafin et al. (2008), who demonstrated that the association between cognitive biases and alcohol consumption was moderated by ego depletion: following ego depletion, the association between cognitive biases and ad-lib drinking became stronger. The current experiment investigated if these findings would generalise to other aspects of cognitive bias that have been demonstrated to have a causal influence on drinking behaviour, that is automatic approach tendencies and attentional bias (Schoenmakers et al., 2010; Wiers et al., 2010; Wiers et al., 2011). The secondary aim was to investigate the specific mediators of the ego depletion effect, from the perspective of dual process models of addiction. Gailliot and Baumeister (2007) argue that ego depletion affects behaviour because it produces temporary impairments in executive cognitive functioning. Previous investigations of the effects of ego depletion on ad-lib drinking did not investigate whether these effects were mediated by changes in executive cognitive functioning or impulsivity. Furthermore, other studies (e.g. Clarkson et al., 2010) suggest that ego depletion effects are driven by the perception of self-control, so this may also be partially responsible for changes in ad-lib drinking following ego depletion. In the present study, executive cognitive functioning, impulsivity, and perceived self-control resources were investigated as possible mediators of the effects of ego depletion on ad-lib drinking.

This study consisted of two different experimental sessions. In the first session cognitive biases were assessed using measures of attentional bias (visual probe task) and automatic approach tendencies (SRC task). Following this participants were exposed to either an ego depletion manipulation (involving both emotional and cognitive suppression, as recommended by Ostafin et al., 2008), or a non-depleting control manipulation. Finally, participants completed a bogus taste test in order to assess their ad-lib drinking. In a second session, participants were again exposed to

the ego depletion manipulation or the control manipulation, before completing a short battery of executive function and impulsivity tests comprising measures of inhibitory control, delay discounting, and phonemic fluency. These tasks were administered in a separate session in the light of evidence suggesting that completion of executive function tasks can itself be ego-depleting (Govorun & Payne 2006; Webb & Sheeran 2003); if so, this would have compromised the anticipated effect of the ego depletion manipulation on ad-lib drinking in the first session.

Firstly, it was hypothesised that participants in the ego depletion condition would consume more beer than participants in the control condition, which would serve to replicate previous reports (e.g. Muraven et al., 2002). The second hypothesis was that the associations between attentional bias for alcohol-related cues, automatic approach tendencies elicited by those cues, and ad-lib beer consumption would be moderated by the experimental manipulation, with the strongest associations seen in the ego depletion group. The third hypothesis was that the ego depletion manipulation would impair all three measures of executive function / impulsivity in the second session, and individual differences in the extent of this impairment, together with effort expended during ego depletion would be associated with, and would mediate, the effects of ego depletion on ad-lib drinking.

7.3. Method

Participants

Eighty participants (46 female) aged between 18 and 40 years (mean 22.08 ± 4.53) were recruited via word of mouth and intranet advertising from the University of Liverpool. Inclusion criteria were fluency in English, and normal or corrected-to-normal vision. Participants were invited to take part if they self-reported consuming at least 15 units of alcohol (females) or 22 units (males) each week, which is in excess of the UK government guidelines for safe drinking (Edwards 1996). Furthermore it was made clear in advertisments and the participant information sheet that all participants must regularly drink beer, as tasting beers was a part of the

procedure. Exclusion criteria included current or past self-reported alcohol use disorder, blood injury phobia, current or recent illness which may increase sensitivity to alcohol (e.g. colds and flu), taking medication that is contraindicated for alcohol use (e.g. antidepressants, anxiolytics). Additional exclusion criteria for female participants included current breastfeeding or pregnancy; the latter was confirmed with a pregnancy test at the beginning of the first session. All participants provided informed consent before taking part in the study, which was approved by the University of Liverpool Research Ethics Committee. Participants received either course credits or £20 as compensation for their travel expenses and time.

Design

The study used a between subjects design in which participants either completed an ego depletion manipulation of a non-depleting control manipulation. Participants were in the same condition for both experimental sessions (in which different tasks were completed). The impact of ego depletion on beer consumption and the predictive utility of automatic cognitive processing (after ego depletion) on beer consumption were investigated using a correlational design. In addition a second session was used to investigate the effects of ego depletion on executive cognitive functioning and whether impairments in these processes as a result of ego depletion mediated any increases in beer consumption following ego depletion in the first session.

Materials

Questionnaires

Time Line Follow Back (TLFB).

The Alcohol Use Disorders Identification Test (AUDIT).

Temptation and Restraint inventory (TRI).

Barratt Impulsivity Scales (BIS-11).

Desires for Alcohol Questionnaire – brief version (DAQ).

Brief Mood Introspection Scale (BMIS; Mayer & Gaschke 1988). The BMIS is a self-report measure of mood and arousal. The BMIS consists of 16 adjectives which are responded to on 4 point Likert scales ranging from 'definitely do not feel' (1) to 'definitely feel'(4). Four underlying mood factors are derived: Pleasant-Unpleasant, Arousal-Calm, Positive-Tired, and Negative-Relaxed. Factor scores are derived by adding or subtracting scores from relevant items. For example, the Pleasant-Unpleasant factor is computed by subtracting values for unpleasant adjectives (e.g. Grouchy, Sad) from scores derived from pleasant adjectives (e.g. Content, Happy). Cronbach's alpha for the subscales of the BMIS range between 0.76 and 0.83 (and as such, are acceptable to good).

Video stimuli for ego depletion manipulation

An 18 minute section from the film Audition (2002, Dir. Takashi Miike) was selected to use for the ego depletion manipulation. The entire 18 minute section contained numerous flashbacks which were cut, so the final edited clip was 10 minutes long. The clip contained graphic depictions of torture (for example, a character has his feet amputated with wire). This clip was used as disgust is more easily and ethically manipulated than emotions such as happiness or sadness (Gross & Levenson 1993). As the experiment consisted of two sessions the scene was split into two five minute segments, the presentation of which was counterbalanced across sessions.

Pictorial stimuli

As used in study one.

Bogus Taste Test (based on Weafer & Fillmore, 2008)

Participants were provided with three numbered glasses each containing 255ml of beer. They were instructed to taste the beers and rate each one according to nine different dimensions, for example 'how sweet tasting is the drink?' by marking 15 point Likert scales with anchors 'not at all' and 'very much'. Participants were also asked to rank the beers in order of preference and order of alcohol content, and attempt to identify the beer brands. Participants were informed that they could drink as much of each beer as they wished in order to make accurate assessments, and they were given 30 minutes to do so. The beers provided were Hoegaarden wheat beer (4.9% ABV), Carlsberg lager (4.2% ABV) and Golden Champion summers ale (5% ABV). Beers with distinctly different tastes were selected to encourage participants to think more about their responses. Responses to the taste ratings, perceived alcohol content and beer preference questions were not analysed. The dependent variable was the total volume of beer consumed.

Cognitive bias tasks (session 1 only)

The Stimulus Response Compatibility (SRC) task.

The visual probe task. As used in study 5.

Impulsivity/Executive cognitive function measures (session 2 only)

Go/No-Go task (Newman & Kosson 1986). As used in study two

Delay discounting (Du et al., 2002). As used in study three.

Phonemic fluency. Identical to study 2, although scores were not reversed, so high scores are indicative of better executive cognitive functioning.

Procedure

Testing sessions took place between 12 pm and 6 pm in laboratories at the University of Liverpool. Each participant attended the laboratory for two separate sessions, with a gap of at least two days between sessions (mean duration between session was 4 days). The first session lasted approximately 75 minutes; the second session lasted approximately 25 minutes. Diagrams illustrating key features of both experimental sessions are shown in Figure 1. During the first session, participants provided informed consent before being breathalysed using a Lion Alcometer 500 (Lion Laboratories, Barry U.K.) to confirm a baseline breath alcohol content (BAC) of zero (no participants provided a positive BAC before either session). They then completed a short questionnaire battery (TLFB, AUDIT, TRI, BIS-11 and DAQ). Participants were then seated 1m away from the computer monitor before completing the SRC and visual probe tasks, in a counterbalanced order.

Participants were randomly assigned to either the ego depletion or the control condition. Both groups of participants were told that they were to watch a film clip. Participants in the ego depletion condition were informed that they should try not to respond to the clip in any way (no facial expressions or turning away), and that they should suppress any thoughts, feelings or emotions that they may experience while watching the clip. Participants in the control condition were given no instructions before watching the clip. The experimenter remained in the laboratory throughout the experiment in order to observe participants' emotional expressions and to remind them of the task instructions if necessary. Once the clip finished, participants were asked to complete a manipulation check questionnaire. Participants were asked to rate perceived effort put into suppression, perceived difficulty of suppression, emotionality of the task, and feelings of being emotionally drained and tiredness on 25 point scales. Participants then completed a cognitive suppression task; participants in the ego depletion group were told to write down any thoughts that came to mind over the next five minutes but not to think about anything that they had just seen in the clip. Participants in the control condition were told to write down any thoughts that they had over the next five minutes, but no reference was made to the film clip. Upon completion of the cognitive suppression task participants completed a second manipulation check as well as the BMIS and DAQ.

Participants were then informed that they would have to taste and rate different beers for 30 minutes, before being asked to complete an additional reaction time task. Participants did not actually complete this final task but were informed that it was a 'short but difficult reaction time task on which good performance would be rewarded with a £10 bonus payment, although performance on this task is very likely to be negatively affected by alcohol consumption'. This was to encourage participants to limit their drinking, similar instructions were utilised by Muraven et al. (2002) and Ostafin et al. (2008). The experimenter left the participant for thirty minutes to complete the taste test. The experimenter then returned, breathalysed the participant and informed them that there would be no reaction time test. Participants were then discharged and reminded to return for the second session.

In the second session participants were again breathalysed before completing the experimental manipulation (ego depletion or control) in the same manner as they did in the first session (including all manipulation checks except for the DAQ). All participants viewed a different clip from that seen in the first session. Upon completion of the manipulation participants completed the Go-No/Go, COWAT and delay discounting tasks in a counterbalanced order. An informal debrief revealed that participants were unaware of the links between the cognitive tasks, experimental manipulations and beer consumption in the taste test; Participants were then fully debriefed on the nature of the study before being released. For an overview of the experimental procedures for session one and two see Figure 7.1.

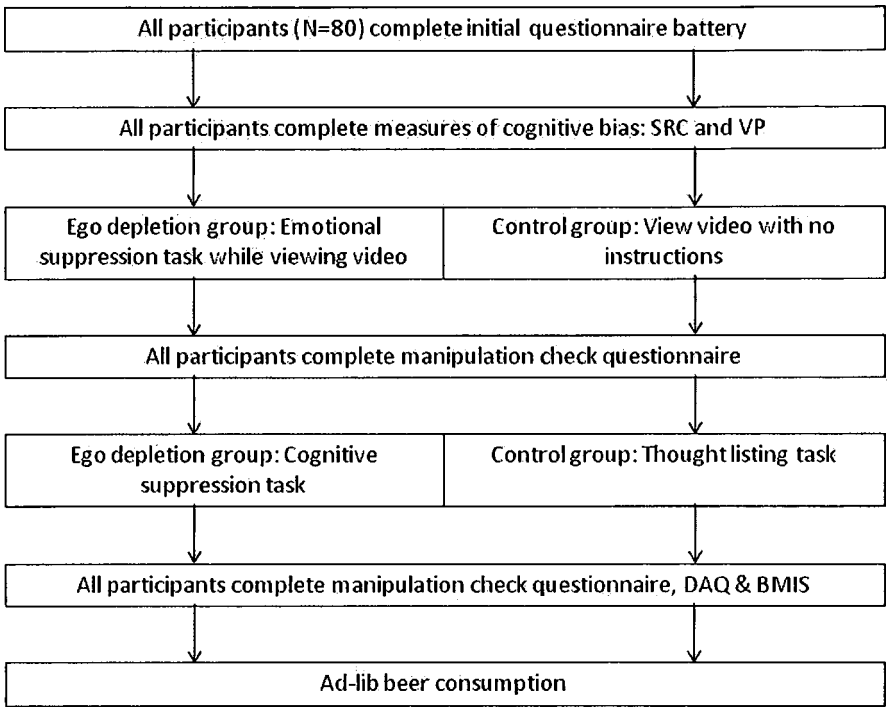
Data analysis

A simple power analysis indicates that a sample size of approximately 80 has sufficient power to detect a medium effect size (power = 0.8, $f^2 = 0.14$, $\alpha = 0.05$). These figures are based on previous research using the same experimental design (Ostafin et al., 2008). Group differences in demographics, alcohol use indices, self-report impulsivity and self-report craving were assessed (the latter was assessed before and after the manipulation) to ensure both groups were matched upon these variables as they are likely to influence beer consumption in the taste test.

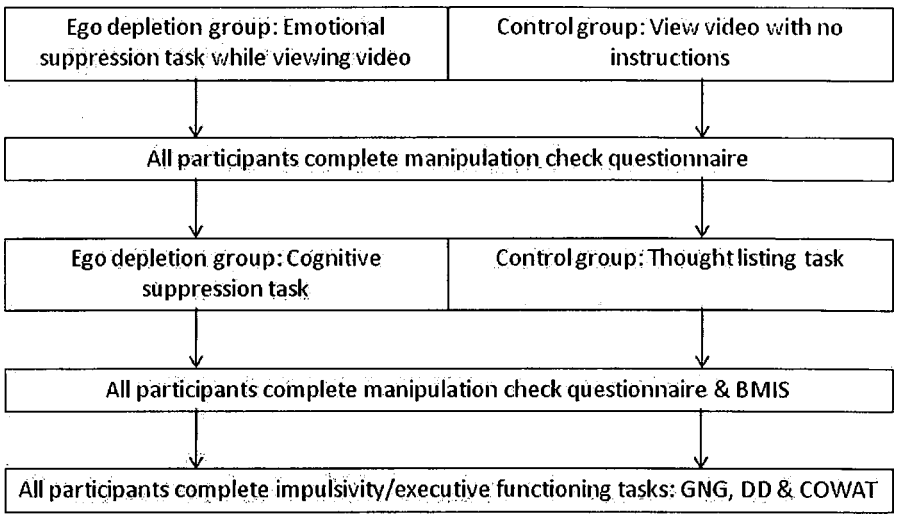
A series of mediation analyses were conducted to investigate whether the anticipated ego depletion effect on beer consumption was a result of impaired executive cognitive function. In addition, mediation analyses were also used to assess the hypothesis that ego depletion effects were mediated by effort put into the depletion tasks. These analyses were conducted on variables that were shown to be significantly affected by the ego depletion manipulation. There has been considerable debate as to which methodologies best evaluate mediation in the social sciences. MacKinnon et al. (2002), investigated 14 methods for mediation analysis and found the joint significance test to be the best method due to its statistical power (in sample sizes similar to the current sample) and because it does not suffer from inflated Type I error rates. This method involves testing the statistical significance of the relationship between the IV (ego depletion) and the proposed mediator (α path), and the relationship between the proposed mediator and the DV (ad-lib beer consumption; β path). If both these relationships are statistically significant there is evidence of mediation. In addition, the PRODCLIN program (see MacKinnon et al., 2007) was used to generate 95% confidence intervals by using the α and β coefficients and their standard errors ($\sigma\alpha$, $\sigma\beta$) to compute the asymmetric confidence interval for the mediated effect.

Figure 7.1: Schematic overviews of experimental procedures in Session 1 (panel 1) and Session 2 (panel 1)

Panel 1



Panel 2



7.4. Results

Group characteristics

Table 7.1 shows summary statistics for the questionnaires that participants completed at the beginning of the session (weekly alcohol consumption, AUDIT, BIS-11 total score and TRI subscales). A multivariate ANOVA (MANOVA) revealed no significant main effect of experimental condition ($F(6,73) = 0.77$, $p > 0.1$, $\eta_p^2 = .04$). Therefore ego depletion and control groups did not differ on any of these measures. There was also no significant difference in the proportion of males and females in the experimental conditions, χ^2 (1, N= 80) = .21, $p > .1$.

Effects of ego depletion on subjective alcohol craving (session 1 only)

A 2 x 2 mixed model ANOVA was used to investigate the effect of experimental condition (ego depletion, control) and time (pre manipulation vs. post-manipulation) on mean DAQ scores. There was a significant main effect of time, with DAQ scores higher at post-manipulation (2.85 ± 0.94) compared to pre-manipulation (2.65 ± 0.86; $F(1,78) = 10.37$, $p = .005$, $\eta_p^2 = .12$). The main effect of experimental condition ($F(1,78) = 2.45$, $p > .1$, $\eta_p^2 = .03$), and the experimental condition x time interaction, were not statistically significant ($F(1, 78) = 2.15$, $p > .1$, $\eta_p^2 = .03$). Therefore, the ego depletion manipulation had no significant effect on self-reported alcohol craving.

Effects of ego depletion on manipulation check questions and mood (sessions one and two)

In order to assess the success of the ego depletion manipulation and whether the effects of the manipulation were consistent across sessions 1 and 2, the manipulation check and BMIS data were analysed using a series of 2 x 2 mixed model ANOVAs, with a within subjects factor of session (session one, session two) and a between subjects factor of experimental condition (ego depletion, control). Participants in the ego depletion condition reported significantly more effort put into both emotional

suppression ($F(1,78) = 15.99$, $p < .001$, $\eta_p^2 = .24$), and cognitive suppression ($F(1,78) = 12.28$, $p < .001$, $\eta_p^2 = .14$). Furthermore, participants in the ego depletion condition also found it significantly more difficult suppressing emotions ($F(1,78) = 7.66$, $p < .001$, $\eta_p^2 = .10$), although there was no significant difference between conditions in self reported difficulty in suppressing thoughts ($F(1,78) = 1.44$, $p > .1$, $\eta_p^2 = .01$). There was also a significant condition X session interaction for both effort put into ($F(1,78) = 4.10$, $p < .05$, $\eta_p^2 = .06$), and difficulty of ($F(1,78) = 4.76$, $p < .05$, $\eta_p^2 = .07$), emotional suppression. This interaction was due to decreases in effort and difficulty ratings between session one and two in the ego depletion condition only. There were no significant main effects of condition or session, and no significant condition X session interactions, for the BMIS subscales, self-reports of emotionally drained and tiredness, and the perceived emotionality of the film clip. Summary data for manipulation check and mood variables are shown in Table 7.2.

Relationship between cognitive bias measures and ad-lib drinking in ego depletion and control conditions

The primary hypothesis of this study was that the association between cognitive biases and ad-lib alcohol consumption would be moderated by the ego depletion manipulation. To test this three separate regression analyses were conducted in which the relationships between automatic approach tendencies, the reaction time index of attentional bias, and gaze dwell time index of attentional bias, with ad-lib beer consumption in the taste test were assessed. Firstly, a multivariate ANOVA (MANOVA) was performed to test for pre-existing group differences in cognitive bias, which revealed no significant differences in cognitive bias between the two groups ($F(3,74) = 1.92$, $p > .1$, $\eta_p^2 = .03$). An independent samples *t*-test was used to investigate gender differences in beer consumption. As males were found to drink more than females, ($t(78) = -4.29$, $p < .001$) gender was added as the first step of in the regression equation. In the second step of the regressions the cognitive bias measure and experimental group (ego depletion or control) was added. In the third step the interaction between the normalised cognitive bias variable and experimental

condition was added to the regression model (with ego depletion coded as 2 and control coded as 1).

In the regression model which assessed automatic approach tendencies as the cognitive bias measure (Table 7.5), the overall model was significant, as the predictors accounted for approximately 31% of the variance in beer consumption (R^2 = .59, R^2 *adjusted* = .31, $F(4,73) = 9.83$, $p < .001$). Firstly, gender was a significant predictor of beer consumed in the taste test with males (469.59 ml ± 235.40) consuming more beer than females (274.02 ml ± 180.31; $\beta = .40$, $p < .001$). Participants with stronger automatic approach tendencies elicited by alcohol-related cues consumed more beer during the taste test ($\beta = .35$, $p = .018$). Experimental condition was also a significant predictor ($\beta = .29$, $p = .003$), with the ego depletion group consuming more beer (429.25ml ± 212.77) than the control group (285.03ml ± 218.27). However, the interaction between automatic approach tendencies and experimental condition was not statistically significant ($\beta = .06$, $p > .1$), which indicates that the association between automatic approach tendencies and beer consumption was not moderated by the ego depletion manipulation.

Regarding attentional bias, although the regression models for both the reaction time index ($R^2 = .10$, R^2 *adjusted* = .07, $F(3,76) = 2.94$, $p < .05$; table 7.3) and gaze dwell time index ($R^2 = .11$, R^2 *adjusted* = .07, $F(3,76) = 3.09$, $p < .05$; Table 7.4) of attentional bias were significant, this was carried by the effect of gender and of ego depletion on beer consumption. Neither the reaction time index ($\beta = .10$, $p > .1$) or the gaze dwell time index ($\beta = .09$, $p > .1$) were significant predictors of beer consumed. Furthermore, the interactions between reaction time index of attentional bias and condition ($\beta = -.02$, $p > .1$) and the gaze dwell time index of attentional bias and condition ($\beta = -.02$, $p > .1$), were not significant.

Effects of ego depletion on executive function / impulsivity

A multivariate ANOVA (MANOVA) revealed that there was a trend towards a main effect of condition on the measures of executive cognitive function / impulsivity, ($F(3,76) = 2.39$, $p < .1$, $\eta_p^2 = .08$). Ego depletion (0.22 ± 0.03) and control groups (0.20

± 0.03) did not differ in discounting rates (AUC values) obtained from the delay discounting task ($F(1,78) = 0.19$, $p > .1$, $\eta_p^2 = .00$). Regarding COWAT switches, there was no significant difference between ego depletion compared to the control group (28.32 ± 8.06 vs. 25.43 ± 8.44; $F(1,78) = 2.47$, $p > .1$, $\eta_p^2 = .03$). However, there was a trend towards increased commission errors (impaired inhibitory control) on the Go/No-Go task in the ego depletion group compared to participants in the control group, (9.10 ± 5.47 vs. 6.85 ± 5.74; $F(1,78) = 3.22$, $p < .1$, $\eta_p^2 = .04$).

Investigating mediators of the effect on ego depletion on beer consumption

As the joint significant test requires the proposed mediator to be affected by the IV, the mediation analysis was only conducted using proposed mediators that were affected by the ego depletion manipulation (i.e. commission errors on the Go/No-Go task and perceived effort put into emotional and cognitive suppression tasks).

There was a trend towards an effect of ego depletion on commission errors (α path; $r^2 = .04$, $\beta = .20$, $p < .1$), although commission errors were not associated with beer consumption (β path; $r^2 = .01$, $\beta = .10$, $p > .1$). This analysis indicates that impairments in inhibitory control did not mediate the effect of ego depletion on alcohol consumption.

To investigate a possible mediating role for perceived effort put into the ego depletion tasks, scores from the two manipulation check variables assessing how much effort participants put into the emotional and cognitive suppression tasks were combined into a single 'effort' variable. The effect of the ego depletion manipulation on effort put into suppression was significant (α path; $r^2 = .25$, $\beta = .50$, $p < .001$) as was the association between effort put into suppression and beer consumption (β path; $r^2 = .05$, $\beta = .22$, $p = .04$). PRODCLIN revealed that the upper and lower 95% confidence limits for the indirect effect of ego depletion on beer consumed were > 1, therefore indicating statistically significant mediation of the ego depletion effect on beer consumed by perceived effort put into the tasks [$CL_{.95} = 1.82, 17.03$].

Table 7.1: Group difference in alcohol use indices and self report impulsivity (Mean ± SD).

	Control	**Ego depletion**
	(N=40)	(N=40)
Gender (M:F)	(16:24)	(18:22)
Age (years)	22.73 (5.56)	21.42 (3.62)
Past 14 day alcohol consumption (UK units)	55.35 (25.93)	60.65 (29.98)
AUDIT	13.50 (5.05)	13.33 (5.06)
TRI CEP	24.38 (11.24)	23.38 (10.23)
TRI CBC	18.38 (8.34)	16.63 (8.20)
BIS-11 Total scores	69.45 (9.83)	73.82 (12.41)

Past 14 day alcohol consumption in UK units, 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). TRI = Temptation and Restraint Inventory, range of TRI subscale scores (minimum to maximum); Cognitive and Emotional Preoccupation (CEP) 9 to 81, Cognitive and Behavioural Control (CBC) 6 to 54. BIS 11 total scores = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum).

Table 7.2: Group and session comparisons of manipulation check and mood variables Mean (SD).

	Session one		Session two				
Variable	Ego depletion	Control	Ego depletion	Control	Condition (F)	Session (F)	Interaction (F)
Emotional suppression							
Effort suppressing emotions	15.68 (5.67)	9.63(5.99)	13.65 (6.85)	9.92 (5.58)	15.99**	2.56	4.10*
Difficulty suppressing emotions	13.80 (6.09)	8.92(6.09)	12.57 (.18)	10.22 (6.12)	7.66**	0.01	4.76*
Emotionality of clip	13.82 (7.08)	14.02 (7.12)	12.82 (6.86)	13.67 (6.09)	0.14	1.23	0.29
Emotionally drained	8.47 (6.56)	8.67 (5.36)	8.02 (6.20)	8.92 (5.45)	0.21	0.03	0.42
Tiredness	8.70 (6.14)	7.32 (5.61)	7.90 (5.92)	7.15 (5.66)	0.38	0.88	0.36
Cognitive suppression							
Effort suppressing thoughts	12.32 (7.12)	7.65 (5.35)	11.67 (7.07)	7.27 (5.18)	12.28**	0.85	0.06
Difficulty suppressing thoughts	11.02 (7.10)	9.07 (6.43)	10.75 (7.00)	9.55 (6.49)	1.44	0.89	0.25
Emotionally drained	6.32 (6.18)	6.60 (5.18)	7.00 (6.07)	7.42 (5.80)	0.08	1.31	0.01
Tiredness	7.92 (6.64)	7.92 (6.84)	7.35 (5.74)	7.15 (5.74)	0.94	0.97	0.02
BMIS							
Pleasant-unpleasant	4.50 (7.24)	3.60 (8.04)	4.77 (7.35)	3.70 (7.33)	0.46	0.05	0.01
Arousal-calm	14.65 (3.71)	14.92 (3.22)	15.50 (2.79)	15.12 (2.79)	0.01	1.36	0.52
Positive-tired	6.22 (3.43)	6.25 (3.49)	6.95 (3.71)	6.52 (3.30)	0.09	1.44	0.29
Negative-relaxed	5.85 (3.56)	6.30 (7.76)	6.32 (4.33)	6.35 (3.13)	0.11	0.37	0.24

All manipulation check scores for emotional and cognitive suppression scores range from 1-25. BMIS subscale scores range from; Pleasant-unpleasant (-) Arousal-calm (-) Positive-tired (-) Negative-relaxed (-) **p < .01, *p < .05

Table 7.3: Regression analysis showing the relationship between visual probe reaction time bias, ego depletion and beer consumption

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Gender	.43	.19	$F(1,78) = 17.72$**	193.41	44.92	.43**
Step 2						
VP Reaction time bias (ms)	.53	.28	$F(2,76) = 5.15$**	0.53	1.10	.10
Ego depletion group				135.85	44.06	.30**
Step 3						
VP reaction time bias x Group	.53	.28	$F(1,75) = 0.01$	-0.09	1.09	-.02

VP reaction time bias (ms) = reaction time to incongruent probes minus reaction times to congruent probes, higher values represent increased attentional bias. VP reaction time bias x Group = product of normalised variables. **p < .01

Table 7.4: Regression analysis showing the relationship between visual probe gaze dwell time bias, ego depletion and beer consumption

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Gender	.43	.19	$F(1,78) = 17.72$	189.42	44.30	.42**
Step 2						
VP gaze dwell time bias (ms)	.53	.28	$F(2,76) = 5.02$	0.10	0.30	.09
Ego depletion group				127.35	45.18	.28**
Step 3						
VP gaze dwell time x Group	.53	.28	$F(1,75) = 0.01$	-0.02	0.27	-.02

VP gaze dwell time bias (ms) = Dwell times on Group pictures minus gaze dwell times on control pictures, higher values indicate greater attentional bias. VP Gaze Dwell time x Group = product of normalised variables. **p < .01

Table 7.5: Regression analysis showing the relationship between automatic approach responses, ego depletion and beer consumption

	Cumulative			Simultaneous		
Variable	R^2	ΔR^2	*F*-change	B	SE	β
Step 1						
Gender	.44	.19	$F(1,76) = 18.22$	181.19	43.09	.40**
Step 2						
SRC Bias (ms)	.59	.35	$F(2, 74) = 8.61$	0.79	0.44	.35**
Ego depletion group				132.56	.42.82	.29**
Step 3						
SRC Bias x Group	0.59	.35	$F(1, 73) = 0.50$	-0.31	0.45	-.14

SRC bias (ms) = Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. SRC bias x Group = product of normalised variables.

**$p < 0.01$

7.5. Discussion

The current study investigated the effects of ego depletion on ad-lib alcohol consumption, and the role of cognitive processes in these effects. In an initial session participants completed two tasks that assessed cognitive biases for alcohol cues (automatic approach tendencies and attentional bias) followed by either an ego depletion manipulation or a control manipulation, before they completed a taste test to measure their ad-lib alcohol consumption. In a subsequent testing session participants underwent ego depletion or the control manipulation, and then completed tasks assessing executive cognitive functioning and impulsivity. It was hypothesised that the ego depletion manipulation would increase ad-lib drinking (beer consumption in the taste test), and also predicted that the associations between cognitive biases and ad-lib drinking would be moderated by the experimental manipulation, with stronger associations in the ego depletion group compared to the control group. It was also predicted that the ego depletion manipulation would influence executive function and impulsivity, and that these changes, together with self-reported effort expended during ego depletion, would mediate the effects of ego depletion on ad-lib drinking. The current results provided partial support for these hypotheses. As predicted, the ego depletion manipulation resulted in increased beer consumption. Individual differences in automatic approach tendencies elicited by alcohol-related cues (as assessed with the SRC task) were associated with ad-lib drinking, although this association was not moderated by the ego depletion manipulation as predicted. Neither measure of attentional bias was associated with beer consumption, regardless of ego depletion. Contrary to expectations, the effects of ego depletion on measures of executive function and impulsivity were inconsistent across different measures, and none of these measures appeared to mediate the effect of ego depletion on ad-lib drinking. Finally, the effect of ego depletion on ad-lib drinking was mediated by self-reported effort expended during ego depletion.

The main effect of ego depletion on beer consumption supports the strength model of self control and directly replicates previous studies such as Muraven et al. (2002): relative to a control (non-depleting) manipulation, beer consumption was increased

following ego depletion, even though participants were given an incentive to refrain from drinking. Therefore, this finding adds to a growing body of evidence which suggests that ego depletion leads to a reduction in self-control resources which can be detected in a variety of domains, including increased alcohol consumption, smoking, overeating, emotion regulation, and expended physical and mental effort (Hoffman et al., 2007; Muraven et al., 1997; Muraven et al., 2002; Shmueli & Prochaska 2009; Stucke & Baumeister, 2006).

Predictions derived from dual process models (e.g. Deutsch & Strack 2006; Wiers et al., 2007), namely that ad-lib drinking would be more strongly associated with cognitive biases for alcohol cues after ego depletion, due to the reduced availability of self control resources, were also investigated. The results did not support these predictions: there were no associations between attentional bias and ad-lib drinking, and although there was an association between automatic approach tendencies and ad-lib drinking, this association was not moderated by the ego depletion manipulation. Although previous studies have found stronger associations between implicit alcohol cognitions and ad-lib drinking after ego depletion when alcohol versions of the implicit association test (IAT) were used (Friese et al., 2008; Ostafin et al., 2008) the current study failed to extend these findings using different measures of cognitive biases for alcohol cues. In the present study, automatic approach tendencies elicited by alcohol-related cues (with the SRC task) and attentional bias for alcohol cues (with the visual probe task) were measured. Given recent experimental findings which suggest that both attentional bias (Field & Eastwood, 2005; Schoenmakers et al., 2010) and automatic approach tendencies (Wiers et al., 2011; Wiers et al., 2010) appear to exert a causal influence on subsequent drinking behaviour, one implication of these results is that the impact of these cognitive biases on drinking behaviour does not seem to be further exacerbated when self-control resources are depleted. This finding is problematic for dual process models (e.g. Wiers et al., 2007; Deutsch & Strack 2006), which posit that individual differences in self-control resources should increase the impact of automatic alcohol cognitions on subsequent drinking behaviour.

It was also hypothesised that the effects of ego depletion on ad-lib drinking would be mediated by executive function and impulsivity, as was predicted by Gailliot and

Baumeister, (2007). Contrary to hypotheses, ego depletion had inconsistent effects on these measures: inhibitory control was mildly impaired, but phonemic fluency and delay discounting were unaffected. Furthermore, performance on these tasks after ego depletion did not mediate the effects on subsequent drinking behaviour. One explanation for these findings is that the effects of ego depletion on ad-lib drinking were assessed in an initial testing session, but the effects of ego depletion on executive function were assessed in a subsequent testing session. This experimental design, rather than assessing effects of ego depletion on executive function, impulsivity, and ad-lib drinking in a single session, was opted for as there was a concern that completion of the executive function and impulsivity measures would serve to deplete self-control resources (e.g. see Govorun & Payne 2006), which may have obscured effects of the ego depletion manipulation on ad-lib drinking, and prevented accurate testing of the primary hypothesis. Unfortunately, analysis of the manipulation check data revealed that the ego depletion manipulation appeared to be less effective in the second session compared to the first, in that participants' self-reported effort, and perceived difficulty in suppressing emotions, was lower during the second session compared to the first. This is consistent with results from a meta-analysis, which found that ego depletion effects tend to decline in magnitude after training on depleting tasks (Hagger et al., 2010). Therefore, in order to conduct an appropriate test of the hypothesis that behavioural effects of ego depletion are partially mediated by changes in executive function and impulsivity, future studies should assess these variables in a single session, in order to prevent practice effects from weakening the effectiveness of the manipulation. It is also possible that participants became fatigued due to the large number of tasks that they completed, so future studies of this type may wish to include only one or two cognitive tasks in order to assess mediation effects.

The analyses did reveal that the ego depletion effect on ad-lib drinking was mediated by participants' perception of how depleted they were (i.e. self reported effort put into the emotional and cognitive suppression tasks). This finding is consistent with the strength model of self control (Baumeister et al., 1998). The implication is that participants who felt that they put more effort into controlling emotional responses to the film clip and suppressing thoughts related to the clip, subsequently consumed

more beer because they perceived their self-control resources to be depleted. Other studies have highlighted the importance of perceived self-control resources as important mediators of ego depletion effects (Clarkson et al., 2010; Job et al., 2010), and demonstrated that automatic processes such as priming of persistence can overcome ego depletion effects (Alberts et al., 2007). When combined with the present results, these studies suggest that the perception of having depleted self control resources, but not actual fatigue in behavioural control processes, is the mechanism that determines the consequences of ego depletion, including the effects on drinking behaviour. The clinical implication is that interventions which aim to challenge perceptions of depletion could be a simple and efficacious method for reducing heavy drinking and other unhealthy behaviours. Alternatively increased beer consumption may have been the direct result of exerting cognitive effort in the ego depletion tasks, rather than the perception of having depleted self control resources. In future measures of perceived cognitive demand, for example the NASA-task load index (NASA-TLX; Hart & Staveland 1988), could be utilised to get an accurate measure of multiple aspects of cognitive demand. This measure of actual cognitive effort exerted could then be compared to perception of effort exerted. It is however notable that the associations between real word cognitive effort, stress and alcohol consumption tend to be weak (e.g. Kjeerheim et al., 1997).

Previous studies that used the SRC task (e.g. study four, Field et al., 2008; Field et al., 2011) demonstrated that heavy drinkers, but not light drinkers, were faster to categorise alcohol-related pictures when required to do so by making a symbolic approach response rather than a symbolic avoidance response. The current results go one step further, as they demonstrate that individual differences in performance on the SRC task are associated with ad-lib drinking when assessed soon after completing the task. As such, these results are consistent with recent findings that demonstrate that strong automatic approach tendencies have a causal influence on drinking behaviour (Wiers et al., 2011; Wiers et al., 2010), although the data within the current thesis does not enable inference of a causal relationship. With regard to attentional bias, there was no association between either measure of attentional bias derived from the visual probe task and ad-lib drinking, which casts doubt on the role of attentional bias as an automatic cognitive processes which drives drinking behaviour (see Field et al.,

2007b; Field & Eastwood, 2005; Schoenmakers et al., 2010). One possible explanation is that the association between attentional bias and the motivation to drink is relatively weak, and very large sample sizes may be required to detect an association between attentional bias and ad-lib drinking in the laboratory (Field et al., 2009).

7.6. Chapter summary

The current results offer support for the strength model of self-control in relation to heavy drinking, as participants consumed more beer, despite a financial incentive to refrain from heavy drinking, after an ego depletion manipulation. These effects were mediated by the degree of self-reported effort expended during ego depletion, rather than by any change in executive function or impulsivity. This has implications for our understanding of the psychological mechanisms that underpin ego depletion effects in general, and on drinking behaviour more specifically. Finally, results were not generally consistent with dual-process models of addictive behaviour: although individual differences in automatic approach responses elicited by alcohol cues were associated with ad-lib drinking, these associations were not more apparent following the ego depletion manipulation, as would be predicted by those models.

8. General discussion

The current thesis aimed to explore the associations between hazardous drinking, automatic processing of alcohol-related cues and behavioural impulsivity. Firstly, the direct association between the automatic processing of alcohol-related cues and hazardous drinking was investigated. Automatic cognitive processing was assessed using measures of attentional bias and automatic approach responses towards alcohol-related cues, as both these aspects of automatic cognition have been shown to have a causal relationship with alcohol consumption (Field & Eastwood 2005; Wiers et al., 2010). Secondly, the direct associations between behavioural measures of impulsivity and hazardous drinking were investigated. As behavioural impulsivity is not a unitary construct measures of the two forms of behavioural impulsivity, impulsive decision making and inhibitory control, were utilised to investigate this construct. In addition to assessing the impact of these processes in isolation, the specific predictions of dual process models of addiction, that the association between automatic cognitive processes and hazardous drinking would be moderated by behavioural impulsivity, were also tested. These general research questions were investigated cross sectionally in young adult student populations (studies one and two), adolescents (study three), and older adults (study four). The aim of study five was to investigate the effect of a priming dose of alcohol on these processes and their association with alcohol-seeking behaviour. Finally, study six was an investigation into the mediators and moderators of the effects of ego depletion on drinking behaviour from the perspective of dual process models of addiction.

8.1. Summary of main findings

Studies one and two (described in chapter three) investigated the associations between attentional bias and automatic approach responses towards alcohol-related cues,

behavioural impulsivity and hazardous drinking within a sample of young adults (students recruited from the University campus). In study one there was no association between attentional bias and hazardous drinking, however, automatic approach tendencies and AUDIT scores were correlated, with increased automatic approach responses being associated with higher AUDIT scores. This experiment found no association between multiple measures of impulsive decision making and hazardous drinking. There was also no evidence that the associations between automatic approach responses or attentional bias and hazardous drinking were moderated by any measure of impulsive decision making. In study two there was a correlation between attentional bias during the visual probe task and hazardous drinking. Furthermore, the regression model revealed that automatic approach responses predicted some variance in AUDIT scores beyond that explained by age and self report impulsivity, although this did not reach statistical significance in the simultaneous regression model ($p < .1$). Study two assessed the association between multiple measures of inhibitory control (and a general measure of executive cognitive functioning; phonemic fluency) and hazardous drinking. Complementing study one, there was no direct association between any of these measures and hazardous drinking and none of the measures of inhibitory control moderated the associations between attentional bias or automatic approach responses and AUDIT scores.

Study three investigated these processes in a sample of adolescent secondary school children. As in study two, attentional bias towards alcohol-related cues in the visual probe task was associated with adolescent drinking behaviour. There was no association between automatic approach responses towards alcohol-related stimuli and any measure of alcohol use or hazardous drinking. Again, there was no direct association between impulsive decision making or inhibitory control and hazardous drinking (or indeed the alcohol use index). Significantly, support for dual process models of addiction was found in this study as the relationship between attentional bias and hazardous drinking was moderated by delay discounting, as this relationship was stronger in more impulsive individuals.

The final cross sectional study tested the predictions of dual process models in a sample of older adults. Unlike previous studies there was no association between attentional bias and hazardous drinking. There was however a significant direct

association between automatic alcohol-approach tendencies and AUDIT scores. There was evidence that both inhibitory control and impulsive decision making were associated with hazardous drinking in this sample, although phonemic fluency was not. Despite the evidence for direct associations between automatic approach responses, impulsive decision making, inhibitory control and hazardous drinking, none of the measures of behavioural impulsivity moderated the association between automatic approach responses and hazardous drinking.

Study five investigated the effects of a 0.65 g/kg alcohol prime (and a placebo) on attentional bias, automatic approach responses, behavioural impulsivity, phonemic fluency and alcohol seeking. In addition to looking at the main effects of the alcohol prime on these measures it was hypothesised that the effects of alcohol on ad-lib drinking (the 'alcohol priming effect') would be mediated by impairments in executive function and increases in attentional bias and automatic approach tendencies. Finally the hypothesis that alcohol induced increases in attentional and approach bias would be mediated by alcohol-induced increases in impulsivity/impairments in phonemic fluency was also tested. Firstly the alcohol prime did result in increased alcohol consumption in the taste test. There was however, no effect of either the 0.65 g/kg priming dose of alcohol, or the expectation of receiving alcohol on attentional bias, although automatic approach responses were elevated following the placebo compared to the control drink, with there being no difference between alcohol and placebo conditions. There was no evidence that the pharmacological, or the anticipated effects of alcohol, resulted in impairments in inhibitory control or increases in impulsive decision making, although the alcohol prime did impair phonemic fluency. In addition, only impairments in phonemic fluency were found to mediate the alcohol priming effect. Although automatic cognitive processing of alcohol-related cues did not mediate the alcohol priming effect, alcohol-induced impairment in phonemic fluency mediated alcohol-induced increases in automatic approach tendencies.

Study six aimed to explore the effects of ego depletion on alcohol seeking and the association between automatic cognitive processing and alcohol seeking. In addition, this study explored the possibility that ego depletion reduces the ability to control behaviour by increasing impulsivity and impairing executive cognitive functioning.

Ego depletion did result in increased alcohol seeking with participants in the ego deletion condition drinking significantly more beer in the taste test than those in the control condition. Automatic alcohol-approach responses, but not attentional bias, were significantly associated with beer consumption in a taste test, although this was not influenced by the ego depletion manipulation. This indicates that ego depletion did not strengthen the relationships between cognitive biases and ad-lib drinking. The ego depletion manipulation also had no statistically significant effect on delay discounting, inhibitory control or phonemic fluency; therefore the predictions from dual-process models were not supported. Instead, the effect of ego depletion on beer consumption in the taste test was found to be mediated by perceived effort put into the ego depletion tasks.

8.2.1 Direct association between automatic processing of alcohol-related cues and hazardous drinking

Throughout the current thesis there was consistent support for the predictions of incentive-motivational (e.g. Robinson & Berridge 2001) and cognitive models of addiction (e.g. Baker 1987). In all the cross sectional studies measures of the incentive-motivational properties of alcohol cues (attentional bias and/or automatic approach tendencies) were significantly positively correlated with hazardous drinking. In study one attentional bias was assessed with the ACT. Surprisingly, there was no association between attentional bias at either SOA and hazardous drinking. One explanation for this lack of an association is that features of the task render it insensitive. For example, the failure to find attentional bias with this task may reflect the relatively weak cueing effect the alcohol-related stimuli would have compared to the general cueing effect that presenting any visual stimuli would have. Due to these issues all subsequent studies utilised a visual probe task, (in which an alcohol-related cue and a neutral cue are presented simultaneously, so that they compete for attention) to assess attentional bias. There was a significant correlation between attentional bias and hazardous drinking in study two. This finding replicates previous research using the visual probe task that has found this task discriminates heavy and light drinkers

(Field et al., 2004b; Miller & Fillmore 2010) as well as cannabis users (Field et al., 2004a) and smokers (Mogg et al., 2003) from controls. As attentional bias was only associated with AUDIT scores when cues were presented for the long duration (2000 ms) this indicates that maintenance of attention on alcohol-related cues is an important factor in the aetiology of hazardous drinking within a non-dependent sample. Indeed, Field et al. (2004b), also found that weekly alcohol consumption was associated with the maintenance of attention on, not the initial orientation towards, alcohol-related-cues among University students. Likewise, attentional bias in the adolescent sample (study three) was associated with the alcohol use index. This finding supports previous research that has utilised different tasks in adolescent samples (e.g. an alcohol Stroop task; Field et al., 2007a). With regard to the sample of older adults in study four the finding that attentional bias was not associated with hazardous drinking is unexpected, especially since the previous studies had all found an association between attentional bias and indices of alcohol use. Indeed, previous research and the predictions of incentive-motivational models of addiction suggests that there should be a strong association between attentional bias (as measured by the visual probe task as well as other measures such as the Stroop) and alcohol use (for a review see Field & Cox 2008).

Interestingly, there was a different pattern of findings in regard to automatic approach responses towards alcohol-related cues. There was an association between automatic approach responses towards alcohol-related cues and hazardous drinking, in the young and the older adult samples (studies one, two and four). Previous research using the SRC task has found that heavy drinkers categorise alcohol-related drinks with a symbolic approach response more rapidly than an avoid response, whereas light drinkers do not show this distinction (Field et al., 2008a; Field et al., 2011). Similar findings have also been reported in cannabis users and smokers compared to controls (Field et al., 2006; Bradley et al., 2004; Bradley et al., 2008). In addition, other paradigms that assess automatic alcohol-approach responses such as the AAT (Wiers et al., 2009) and the approach-avoid IAT (e.g. Lindgren et al., 2009) have also demonstrated that heavy drinking is associated with the increased accessibility of automatic alcohol-approach responses in University student samples. Unlike the attentional bias, automatic alcohol-approach responses were not associated with

hazardous drinking or increased alcohol consumption in adolescents. This is not surprising, the only other study to date that has investigated these processes in adolescents found a negative association between alcohol-approach responses and alcohol consumption (van Hemel-Ruiter et al., 2011). Significantly, van Hemel-Ruiter et al. (2011) used the AAT rather than an SRC task so it is likely that the lack of findings with the SRC task in the adolescent sample are not task dependent but reflect a lack of predictive utility of automatic alcohol-approach responses in adolescents.

Taken together, the results from the measures of automatic cognitive processing suggest that attentional bias and automatic approach responses towards alcohol-related cues follow different developmental trajectories. Studies one and two demonstrated an association between both automatic approach responses and attentional bias and hazardous drinking as predicted by incentive-motivational and cognitive models of addiction (Robinson & Berridge 1993; 2001; Baker 1987). This suggests that alcohol-related stimuli gains incentive-motivational properties in young adult social drinkers, even though they do not have particularly long drinking histories. However, the results from the adolescent sample suggest that within this population attentional bias, but not automatic alcohol-approach responses, is associated with individual differences in drinking. One explanation for the strong association between attentional bias but not automatic approach responses and adolescent drinking is that alcohol-approach responses require a longer conditioning history to fully develop, while attentional bias may develop more rapidly after a shorter conditioning history. Indeed automatic alcohol positive associations have been shown to develop even before the onset of drinking (O'Connor et al., 2007). It is therefore possible that some aspects of cognitive bias towards alcohol-related stimuli such as attentional bias (as well as other forms of automatic cognitive processing such as alcohol-positive/arousal associations) may be to some extent socially learned (Pieters et al., 2011; Zucker et al., 1995). The lack of an association between approach response and alcohol use in adolescents suggest that a substantial conditioning history is necessary for this link to become apparent, which may not be necessary for attentional bias and alcohol use. According to incentive-motivational models of addiction automatic responses to alcohol-related cues should develop slowly as the result of a sensitisation of the mesolimbic dopamine system. It may be that attentional

processes are sensitised more rapidly than automatic approach responses, which means that while individual differences in attentional bias become evident in adolescence, automatic alcohol-approach responses would only appear later in life. Furthermore, it has been demonstrated that during adolescence there is a rapid development of brain regions associated with motivational orientation. This results in corresponding increases in appetitive responses to any rewarding stimuli in the environment (Somerville et al., 2011), this process may drive attentional bias but not approach responses. This explanation does seem plausible as the opposite pattern of results was found in older adults. This may reflect that older adults have a significant drinking history and therefore they have developed strong automatic alcohol-approach responses which the adolescent sample lacked. Although previous research has demonstrated an association between attentional bias and drinking in adult populations, these studies have largely been based on young adult samples; it is possible that as individuals age other aspects of automatic cognition drive drinking behaviour. Indeed, future research could use longitudinal designs to investigate the specific developmental trajectories of these measures over the life time and their association with alcohol use.

In summary, all the cross-sectional studies found some association between automatic cognitive processes and hazardous drinking, or regular drinking in adolescents, which in itself could be considered a hazardous behaviour. These findings support incentive-motivational (e.g. Robinson and Berridge 1993; 2001), as well as cognitive (e.g. Baker 1987) models of addiction. The inconsistency between which specific forms of automatic cognitive processing predicted hazardous alcohol use in the different samples suggest that, (1) a refinement of the tasks used to measure these processes is required, and/or (2) that more research into these processes in different samples is needed to understand the association between different measures of automatic cognitive processes and drinking in different populations.

Despite the evidence found in the cross sectional studies the two manipulation studies had mixed findings regarding the contribution of the automatic processing of alcohol-related cues to alcohol seeking in the laboratory. Firstly, in study five there was no effect of alcohol, or the anticipation of receiving alcohol, on attentional bias. Previous research utilising the visual probe task has generally found acute alcohol consumption

increases attentional bias to alcohol-related-cues (Adams et al., 2011; Duka & Townshend 2004; Schoenmakers et al., 2008). However, it is important to note that these studies utilised a lower dose of alcohol (≤0.4 g/kg) compared to study five in this thesis. Although even at these lower doses results tend to be inconsistent (e.g. no increase following 0.3 g/kg was found by Miller & Fillmore 2011). In other studies that have used priming doses of alcohol in excess of 0.6 g/kg there seems to be no subsequent increase in attentional bias (Duka & Townshend 2004; Miller & Fillmore 2011). It is likely that there is a dose dependent effect of alcohol on attentional bias towards alcohol-related cues, with lower priming doses increasing attentional bias but higher doses having no effect on baseline attentional bias or even eliminating attentional bias entirely. Although Duka and Townshend (2004) suggested the lack of an effect of moderate to high doses of alcohol on attentional bias was the result of decreased motivation to drink due to satiety, study five would suggest this is not the case as there was attentional bias exhibited in all conditions of this experiment as was increased craving and alcohol seeking. Furthermore, attentional bias did not mediate the alcohol priming effect. Likewise, there was no direct association between attentional bias and beer consumption (regardless of the presence of self control resources) in study six. These findings are surprising as attentional re-training studies have shown that attentional bias is associated with beer consumption in the laboratory (Field & Eastwood 2005). One explanation for this is that attentional bias is only associated with increased alcohol consumption in the laboratory if manipulated to be artificially increased. Attentional retraining has been found to increase craving (Field & Eastwood 2005); this increase in craving may mediate the relationship between attentional bias and beer consumption (as would be predicted by incentive-motivational models such as Franken 2003). Alternatively the association between attentional bias and alcohol consumption in the laboratory may be very weak. Indeed, there have been failures to replicate the effect of attentional retraining on beer consumption in the laboratory despite the retraining manipulation increasing craving (Field et al., 2007b).

Despite the lack of an effect of the pharmacological or anticipated effects of alcohol on attentional bias, the anticipated effects of alcohol caused an increase in automatic approach responses that were maintained following the alcohol prime. This indicates

that the expectation of receiving alcohol increases automatic alcohol-approach associations and that this increase is not altered by the pharmacological effects of alcohol. This has important implications for the future study of the effects of alcohol on automatic alcohol-approach associations. Previous research using an identical task found that a priming dose of alcohol had no affect on automatic approach responses in comparison to a placebo condition (Schoenmakers et al., 2008). Despite automatic approach responses being significantly greater than baseline following the alcohol prime this did not mediate the alcohol priming effect. Although there was no evidence for acute effects of alcohol on automatic approach responses causing the alcohol priming effect, there was some evidence for an association between these processes and beer consumption in the taste test in study six. Automatic approach responses predicted beer consumption (regardless of ego depletion condition). This, along with the findings of Wiers et al. (2010), suggests that conditioned approach responses can drive alcohol seeking behaviour in non-intoxicated individuals. These two studies suggest that although cognitive bias may be associated with drinking behaviour, cognitive bias following an alcohol preload (as measured by these tasks) is not associated with increased alcohol-seeking behaviour. Indeed the balance of evidence indicates other cognitive processes (specifically impairments in executive cognitive processes see section 8.2.2.) mediate the alcohol priming effect.

In summary, the current thesis has offered support for incentive-motivational and cognitive models of addiction as in all the cross sectional studies there was some association between the automatic processing of alcohol-related cues and hazardous drinking. However the different predictive utility of these measures in different samples indicates that different automatic processes are associated with hazardous drinking in different stages of life. This highlights the importance of understanding the development of these processes, especially if interventions are going to concentrate upon modifying them. In addition, study six revealed that automatic approach responses were associated with drinking in the laboratory (regardless of self control resources). Study five, however, found that the alcohol priming effect was not mediated by alcohol induced changes in these processes, and the effects of alcohol (and the anticipated effects of alcohol) on automatic cognitive processes were inconsistent. As with the cross sectional studies, this suggests that measures of

attentional bias and automatic approach responses assesses different underlying cognitive processes, rather than a generalised sensitisation of incentive-motivational processes.

8.2.2. Direct association between behavioural impulsivity and hazardous drinking

Unlike automatic cognitive processes, the findings regarding the associations between behavioural impulsivity and hazardous drinking were very inconsistent. Theoretically (see de Wit & Richards 2004; Olmstead 2006) behavioural impulsivity is associated with hazardous drinking, however, neither inhibitory control or impulsive decision making was directly associated with hazardous drinking in young adults or adolescents (studies one to three). Significantly, a very different pattern of findings was found in the older adult sample insofar as both measures of behavioral impulsivity were associated with hazardous drinking as predicted by de Wit and Richards (2004) and Olmstead (2006).

The lack of an association between delay discounting and hazardous drinking in the young adult and adolescent samples was surprising as previous studies have shown that increased impulsive decision making is associated with elevated alcohol consumption within young adults (e.g. Vuchinich & Simpson 1998; Murphy & Garavan, 2011) and adolescents (16-18 year olds; Field et al., 2007a). Likewise, previous research has also demonstrated that impairments in inhibitory control are associated with drinking behaviour in young adults (Colder & O'Connor 2002; Murphy & Garavan, 2011; Weafer et al., 2011), and adolescents (e.g. Wong et al., 2006). Although it was predicted that there would be a direct association between behavioural measures of impulsivity and hazardous alcohol use in young adults and adolescents, the literature regarding the associations between measures of behavioural impulsivity and drinking is very inconsistent. There have been numerous failures to demonstrate an association between delay discounting and alcohol consumption in student samples (e.g. Fernie et al., 2010; MacKillop et al., 2007). Similarly, there

have been failures to replicate the association between drinking and inhibitory control in relationship in young adult social drinkers (Yan & Li 2009; Fernie et al., 2010).

One possible explanation for the association between behavioural impulsivity and alcohol use only being found in older adults is that impulsivity may only be consistently associated with drinking in individuals who have been drinking heavily for some time. Young adults and adolescents may simply not be heavy enough drinkers (and had not been drinking heavily enough for a prolonged period of time) to show increased behavioural impulsivity. The meta-analysis of the delay discounting literature by Mackillop et al. (2011) indicates that only in samples that have been drinking over a prolonged period of time will there be a strong association between impulsive decision making and alcohol use. So, in student and adolescent samples the relatively short time spent drinking will mean that they do not suffer from increased impulsive decision making (or indeed impairments in inhibitory control). One explanation for this is that chronic alcohol use directly impairs these processes due to its effects on the prefrontal cortex (Lyvers 2000), however investigation of this would require extensive longitudinal research. Furthermore, although previous research has shown that measures of inhibitory control are associated with adolescent alcohol consumption, it is important to note that inhibitory control only contributes a significant amount of variance to the prediction of alcohol consumption in 'at risk' populations. Indeed, Nigg et al. (2006) found that inhibitory control deficits prospectively predicted a small amount of variance (1%) in adolescent alcohol consumption, whereas in adolescents specifically stated as being at risk this increased to 9%. This, along with the current results, suggest that inhibitory control does not play an important role in contributing to individual differences in alcohol consumption in adolescent samples which are not at risk, (which may also be the case for delay discounting). It is also possible that although these measures of behavioral impulsivity represent a risk factor, other psychological (e.g. individual differences in automatic processing of alcohol-related cues; see discussion in section 8.2.3), or social factors are also necessary for them to influence behavior.

An alternative explanation for the lack of an association between behavioural impulsivity and drinking behaviours in adolescents and students is that samples may have had different motivations for drinking. For example, there is no reason to assume

that student drinking is the result of an impulsive act. Indeed, studies suggest that heavy student drinking is facilitated by social norms and explicit outcome expectancies (Faulkner et al., 2006; Wicki et al., 2010). For example, a first year undergraduate student may only have a limited number of hours of University to attend to in a single week, have no work deadlines looming and the rest of his/her peer group may all be drinking heavily. For this individual heavy drinking may be a rational decision based upon their current situation, as drinking may not result in them missing work, being at a financial loss or cause relationship problems. If an older adult in full time employment with a family displayed a similar pattern of drinking to this undergraduate student then their behaviour could be seen as impulsive as they are drinking to the detriment of their work and family life. Indeed, Littlefield et al. (2009) argue that elevated impulsivity is associated with individuals who do not 'mature out' of problematic alcohol consumption patterns during their early 20s. It is pertinent at this point to come back to the general definition of impulsivity as being 'maladaptive or inappropriate behaviours' (de Wit 2009 p 23). It is easy to equate an older adult in full time employment drinking heavily to being both maladaptive and inappropriate, but less so for an undergraduate student. In the attempts to operationalise impulsivity the actual definition of what an impulsive behaviour is, rather than how it is measured, has been (to an extent) lost. This highlights the importance of not making assumptions about participants drinking motives, expectancies etc.

Finally, recent research by Friedman et al., (2008) suggested that updating working memory is the essential executive cognitive function. Indeed this study found that inhibitory control does not contribute any unique variance beyond that explained by other measures of executive cognitive functioning (updating and switching). This suggests that the most theoretically important aspect of executive cognitive function is updating. Future studies could concentrate on assessing updating rather than measures of inhibitory control.

The final two studies also had mixed findings in regard to the contribution of behavioural impulsivity to alcohol consumption. The findings regarding the acute effects of alcohol on inhibitory control are particularly surprising as there is a large evidence base showing that the measure of inhibitory control that was utilised in the priming study (the Cued Go/No-Go) is highly sensitive to the disinhibiting effects of

alcohol (e.g. Marczinski et al., 2005; Marczinski et al., 2007). The failure to replicate this may be due to the participants in study five being particularly heavy drinkers (mean weekly UK unit intake 39.00 ± 17.29; mean AUDIT scores 16.06 ± 5.32) and may therefore have shown a degree of tolerance to the disinhibiting effects of alcohol. The finding regarding delay discounting is less surprising as previous studies have reported inconsistent effects of alcohol on impulsive decision making (Ortner et al., 2003; Reynolds et al., 2006b; Richards et al., 1999).

Significantly, the alcohol prime (but not the expectation of receiving alcohol) did impair phonemic fluency, which requires inhibitory control, as well as the other core components of executive cognitive functioning, namely working memory and switching (Miyake et al., 2000). This indicates phonemic fluency tasks may be particularly sensitive to the acute effects of alcohol in heavy drinking samples. Furthermore, it was revealed that the alcohol priming effect was mediated by impairments in phonemic fluency but not by either measure of behavioural impulsivity. Previous research has found alcohol-induced impairments in inhibitory control mediate increased ad-lib alcohol consumption in the laboratory (Weafer & Fillmore 2008). This is the first study to date to show that increased alcohol seeking following an alcohol prime is mediated by impairments in phonemic fluency. There is evidence that phonemic fluency tasks measure the three core areas of executive cognitive functioning (Abwender et al., 2001; Troyer et al., 1997), it is possible that as well as targeting inhibitory control for investigation, impairments in other areas of executive cognitive functioning could be investigated as potential mediators of the priming effect. As both working memory (Balodis et al., 2007; Grattan-Miscio & Vogel-Sprott 2005) and switching (Guillot et al., 2010) have been found to be impaired by priming doses of alcohol it is possible that global impairments in executive function contribute to the priming effect rather than specifically inhibitory control. However it is also plausible that updating working memory may be the core executive cognitive function that accounts for the priming effect. If this is the case then it is possible that it is the updating aspect of the phonemic fluency task, rather than inhibitory control and set switching that is impaired. Furthermore, this raises the possibility that studies which have attributed the priming effect to impairments in

inhibitory control may have found similar results if they had assessed working memory function.

In the final study, there was no evidence that ego depletion increased impulsive decision making or impaired phonemic fluency and although there was a trend towards ego depletion impairing inhibitory control, this did not mediate increases in alcohol seeking that were found following ego depletion. One explanation for the failure to support the hypothesis is that ego depletion has its effects through expectancies rather than actually depleting a limited self control resource. Indeed, study six found that the effect of ego depletion on alcohol consumption was mediated by self reported effort put into tasks. Although this is consistent with the strength model of self control (Baumeister 2003) and the meta-analysis of Hagger et al. (2010), it also raises the possible alternative explanation that ego depletion is the result of participants expecting depleted self control rather than actual levels of self control depletion. For example, Clarkson et al. (2010), found that priming participants to believe self control resources are intact protects them from ego depletion manipulations. Similarly, Job et al. (2010), found that if participants believe self control is unlimited ego depletion manipulations had no effect on subsequent behaviour. Another possibility is that ego depletion results in a lack of behavioural control due to impairing other processes (e.g. working memory) that are as yet to be identified.

In summary, the findings regarding behavioural impulsivity were not as predicted. Only in the older adult sample was there any evidence that behavioural impulsivity is directly associated with hazardous drinking, suggesting that either behavioural impulsivity increases as samples continue to drink or that behavioural impulsivity only impacts alcohol consumption in certain samples. Like with automatic cognitive processing, this indicates that these measures have differential effects in different samples although, notably, rather than having some association with hazardous drinking in all samples, impulsivity seems to have a significant direct association with hazardous drinking in older adults only. The alcohol prime also seemed to have little effect on behavioural impulsivity; indeed there was no evidence that either measure was elevated following alcohol consumption. Despite this phonemic fluency (a general measure of executive cognitive function) was shown to be impaired by the

prime and this impairment mediated the alcohol priming effect. Furthermore, none of these measures were found to be impaired by ego depletion, and there was no evidence that they mediated increased alcohol seeking in this study. Taken together the results from the final two studies indicate that measures of behavioural impulsivity have a degree of stability, and the effects of alcohol on behavioural impulsivity do not mediate increased alcohol seeking, at least within heavy drinking student samples.

8.2.3 Evidence for dual process models of addiction

The current thesis also aimed to investigate the specific predictions of dual process models of addiction, that the association between automatic cognitive process and alcohol consumption would be moderated by individual differences in behavioural impulsivity. The evidence for the specific predictions of dual process models derived from the current thesis was very limited. There was no evidence that any of the measures of impulsive decision making or inhibitory control moderated the relationship between automatic processes of alcohol-related cues and hazardous drinking within the young or older adult samples, this is surprising as in all studies there were main effects of either cognitive bias or impulsivity. Significantly, support for dual process models of addiction was found in adolescents. In this sample, attentional bias was directly associated with scores on the alcohol use index, but not specifically with hazardous drinking. However, hazardous drinking was associated with the interaction between attentional bias and delay discounting, insofar as adolescents with greater attentional bias and steeper discounting of future rewards had higher scores on the alcohol problem index.

This pattern of findings is consistent with previous research investigating dual process models of addiction. The only cross sectional study to date that has found support for dual process models of addiction in an adult sample is Houben and Wiers (2009b) who demonstrated that inhibitory control, measured by the colour-conflict Stroop task, moderates the association between automatic alcohol-positive associations and alcohol use in a largely student sample. The failure to extend these findings to

different measures of cognitive bias or impulsivity may be because of the specific tasks used. The tasks used in the current thesis may simply be less sensitive measures of inhibitory control and automatic cognitive processes compared to those utilised by Houben and Wiers (2009b). The lack of evidence in the current thesis for dual process models of addiction in students may also be due to students simply not trying to control their behaviour. As stated earlier it is possible that students do not view their behaviour as abnormal in comparison with their peers so do not attempt to control it. Although this may be a reasonable explanation for the failure to find support for dual process models in the young adult sample, this does not explain the findings in the older adult population. In this sample although all of the behavioural substrates for dual process models of addiction were present, there was no evidence that behavioural impulsivity moderated the association between automatic approach responses and hazardous drinking.

The finding that in adolescents the association between attentional bias and hazardous drinking was moderated by individual differences in impulsive decision making is broadly consistent with dual process models of addiction, as well as previous research in adolescents (Grenard et al., 2008; Thush et al., 2008). It is notable that the alcohol use index was directly associated with attentional bias and the alcohol problem index was associated with the interaction between attentional bias and delay discounting. This suggests that increased attentional bias towards alcohol-related cues combined with steep discounting of future rewards represents a significant risk factor for hazardous patterns of drinking in adolescents. It is likely that appetitive responses towards alcohol-related stimuli alone are not sufficient to cause hazardous drinking within adolescents; increased levels of impulsivity are necessary to make the pattern of drinking hazardous. There was, however, no evidence that inhibitory control moderated the association between attentional bias and hazardous drinking as dual process models of addiction (Goldstein & Volkow 2002; Jentsch & Taylor 1999) specifically predict. This may be because improvements in inhibitory control develops earlier in adolescence than reductions in impulsive decision making, so the sample may have had relatively good inhibitory control yet relatively high levels of impulsive decision making (Prencipe et al., 2011). Significantly, only working memory capacity has previously been demonstrated to moderate the impact of automatic cognitive

processes (measured with word association tests and alcohol-positive and alcohol-arousal IAT's) on alcohol consumption in adolescents (Grenard et al., 2008; Thush et al., 2008). Impulsive decision making and working memory may therefore represent two forms of controlled processes that have a specific role in the regulation of automatic cognitive processes in relation to adolescent alcohol use.

These results have some fundamental implications for the applicability of dual process models of addiction. Despite these theories being in existence for over a decade the evidence for them within humans is limited with the majority of evidence coming from at risk adolescent samples. Indeed, study three of this thesis is, to my knowledge, the first cross sectional study to show evidence for dual process models in an adolescent sample that could not be specifically described as being 'at risk'. There is now evidence that multiple measures of automatic cognitive processes (visual probe task, IAT's, word association tests) and multiple measures of controlled processes (delay discounting, working memory) interact to predict alcohol consumption within adolescents. Despite the evidence found for dual process models of addiction in adolescents there is very little evidence for dual process models of addiction in adults. This is a significant problem for dual process models of addiction; indeed it is possible that dual process models need to be re-assessed in order to improve their predictive validity. One fundamental problem with them may be the conceptualisation of controlled behaviour within adults and especially student samples. As proposed by Wiers et al. (2007) an individual may need to build up sufficient negative experiences with alcohol before they will engage some form of controlled processes to try and control their drinking behaviour.

However, the relative lack of findings in regard to dual process models of addiction does not necessarily mean that they should be dismissed. There is certainly an argument that the assessment techniques utilised in the measurement of both automatic cognitive processes and behavioural impulsivity need some refinement, (for a detailed discussion of these limitations see section 8.3). If the ecological validity of these measures can be sufficiently increased then it is possible that the empirical support for the predictions of dual process models of addiction will be increased. Indeed, the conceptualisation of 'automatic cognitive processes' and 'behavioural impulsivity' may be overly simplistic and hinder the search for evidence for dual

process models of addiction. In addition, as the current thesis has highlighted, both automatic cognitive processes and behavioural impulsivity are dynamic processes that change throughout life, with different variables predicting hazardous drinking in different age groups. By focusing on the measurement techniques that have the greatest utility in different samples a clearer picture of the validity of dual process models could be developed.

The alcohol priming study did offer some support for dual process models of addiction. Specifically, when impairments in executive cognitive function (specifically phonemic fluency) were controlled for the main effect of the priming condition on automatic approach responses was lost. This suggests that the effect of priming on approach responses is at least partially mediated by impairments in executive cognitive function. Although impairments in executive cognitive function increased the accessibility of the automatic association between alcohol and approach, this was not a decisive factor in the priming effect. It is likely that acute alcohol consumption at the dose utilised in study five has such a large impairing effect on executive cognitive function that this accounts for increased alcohol seeking following a prime, regardless of the strength of automatic cognitive processes. Although automatic alcohol-approach responses are directly associated with alcohol seeking (as was revealed in study six), and were maintained even following a large priming dose of alcohol (study five), these relationships were not moderated by aspects of impulsivity or executive function.

Study six did not support the predictions of dual process models of addiction, as the association between automatic approach tendencies and heavy drinking was not strengthened by ego depletion. This was surprising as previous research has shown that automatic alcohol-approach associations (as measured by an IAT) predicted beer consumption in participants with depleted self control resources (Friese et al., 2008; Ostafin et al., 2008). One explanation for the failure to expand on this previous research is that it is possible that the IAT and SRC are assessing subtly different cognitive mechanisms. In addition, although the aforementioned studies found increased behavioural control by automatic processes, they did not assess for mediators of this effect so it is possible that increased behavioural control by these

processes may be the result of expectancies rather than a specific inability to control behaviour.

In summary, the current thesis has found very limited support for dual process models of addiction. The only clear evidence was found in the adolescent sample in which the impact of attentional bias on hazardous drinking was moderated by impulsive decision making as predicted. Interactions between behavioural impulsivity and automatic cognitive processing did not predict variance in hazardous drinking in any other samples. Likewise, the ego depletion study found no evidence to support dual process models of addiction, indicating that the specific mechanisms by which ego depletion affects behaviour are not, as yet, fully understood. Although there was some evidence for dual process models of addiction in study five inasmuch as impairments in phonemic fluency mediated increases in approach bias, this did not account for the alcohol priming effect. Overall, the current results suggest that the specific predictions of dual process models of addiction are most likely to be supported in adolescent samples. They also indicate that the continued refinement of measures of both behavioural impulsivity and automatic cognitive processing, in regards to their internal reliability, as well as what specific aspects of cognition they are assessing, is needed before these models can be tested further.

8.3. Limitations

There are several limitations with the current research. One fundamental issue concerns the nature of the tasks that assessed automatic responses to alcohol-related cues. Although there is a large evidence base implicating attentional bias (see Field & Cox 2008 for a review) as well as automatic approach responses (e.g. Field et al., 2008; Lindgren et al., 2009; Wiers et al., 2009) in the aetiology of hazardous drinking, the strength of the associations between these measures and actual drinking behaviour tends to be moderate (Rooke et al., 2008). This has been generally reflected in the current thesis; indeed the association between attentional bias and automatic approach responses and hazardous drinking were inconsistent across different populations.

Ataya et al. (*in press*) found that visual probe tasks have relatively poor internal reliability and suggested that these tasks may only have limited value in assessing attentional bias compared to the addiction Stroop. One explanation for this lack of internal consistency is that participants may display differing degrees of attentional bias to the different pictures used as stimuli in these tasks. For example, an individual may particularly like red wine and display attentional bias towards it. This individual may occasionally drink white wine so may show a lesser degree of attentional bias towards it, but may find beer particularly aversive, and may actually orient attention away from beer-related pictures. Therefore this individual may only have attentional bias towards specific subset of stimuli, thereby reducing the internal reliability of these measures (Field & Christiansen, *in press*). It is worth noting that the SRC task may not suffer from the same limitations as participants are responding to a relevant category feature of the stimulus (does the picture contain alcohol-related stimuli or not) so specific stimuli used in the pictures may be less important (Field et al., 2011). It is possible by taking into account drink preference the internal reliability of the visual probe task will be increased. Personalised stimuli have already been shown to be efficacious in IAT's (Houben & Wiers 2009a) as well as Stroop tasks (Cox et al., 2002). There has, other than in the aforementioned studies, been relatively little research into personalised stimuli in cognitive bias tasks particularly with regard to pictorial stimuli so these assertions are speculative.

A further issue with the assessment of automatic cognitive processes is that of cross sensitisation. It is possible that as well as regularly consuming alcohol participants also consumed dopamine agonists such as cocaine or amphetamine. Conditioned place preference studies have demonstrated that cocaine and alcohol used together results in excessive incentive salience being placed on cues associated with both drugs (Gossop et al., 2006). In addition, a significantly greater amount of dopamine being released when both drugs are administered together compared to when one of the drugs is administered in isolation (Sobel & Riley 1999). This suggests that participants who used other dopamine agonists when drinking would have exaggerated incentive motivational responses to alcohol-related cues compared with those who only drank alcohol in isolation. It is recommended that future research

should also measure other drug use to control for cross sensitisation exaggerating responses to alcohol related cues in polysubstance users.

As well as there being limitations in the assessment of automatic processing of alcohol-related cues, the measurement of behavioural impulsivity also proved problematic. Theoretically, there are two forms of behavioural impulsivity, impulsive decision making and inhibitory control (de Wit & Richards 2004; Olmstead 2006). However, the principle component analysis conducted in studies one and two indicated that tasks that ostensibly assess these general constructs may be measuring subtly different processes. Study one found that all three measures of impulsive decision making were fully independent. Although this is not surprising in the case of time estimation, the results regarding the Two Choice Delay task and delay discounting were unexpected. It is possible that the Two Choice Delay task is measuring boredom susceptibility rather than discounting of future rewards. Study two found the measures of inhibitory control were distinct. Performance on the phonemic fluency task loading on to a distinct component is expected as although it requires inhibitory control, other aspects of executive cognitive functioning are required for successful performance on this task (Abwender et al., 2001; Troyer et al., 1997). The finding that the antisaccade and the Go/No-Go task loaded on to different factors is more problematic. One explanation is that while the Go/No-Go task involves inhibiting a learnt, task specific response the antisaccade assesses the ability to inhibit a reflex. Indeed, it seems that different measures of inhibitory control do measure different processes. For example, it has been argued that Go/No-Go and Stop-Signal tasks measure different aspects of inhibitory control, action restraint and action cancellation respectively. It is, however, worth noting that within a normal population a high correlation between these two measures would be expected (Schachar et al., 2007). Furthermore, neuroimaging studies have revealed different patterns of frontal striatal activation in participants performing these tasks (Rubia et al., 2001). Finally, it has also been demonstrated that the neurotransmitters involved in performing these tasks differ with Go/No-Go task performance being associated with serotonergic functioning, while Stop-Signal tasks are associated with noradrenaline (Eagle et al., 2008). It is possible that performance on the antisaccade

task also involves different neurological substrates so it maybe assessing a slightly different component to the Go/No-Go task.

These limitations in measurement suggest that researchers investigating dual process models of addiction (or indeed either impulsivity or automatic cognitive processes in addictive behaviours) should focus on what methodology they are utilising to assess these constructs. Furthermore, attentional bias modification, the retraining of automatic approach responses, improving working memory and reducing delay discounting have all been treatment targets which have shown some success. By clearly defining these processes, as well as tailoring training programs to reflect individual differences (e.g. attentional retraining using personalised stimuli) the efficacy of these treatments could be improved making them valuable tools in the treatment of addiction or as an early intervention in hazardous drinkers.

A further consideration is that the findings regarding measures of behavioural impulsivity predicting hazardous drinking in the sample may have been the result of natural cognitive aging. Prefrontal regions of the brain have been shown to undergo marked changes in both structure and functioning as individual's age (Moscovitch & Wincur 1995). Significantly, there is evidence for an age related decline in both working memory functioning (Salthouse 1991) and inhibitory control (West 1996), for a review of age related cognitive decline see Park et al., (2001). This decline in executive cognitive functioning in the elderly is associated with reduced grey matter volume (Mirsky et al., 2011) and hemispheric asymmetry reduction (Cabeza 2002). It is possible that the natural effects of aging may exacerbate impairments in executive cognitive functioning caused by long term alcohol use. Furthermore, automatic memory processes have been found to be relatively resistant to age related decline (Hasher & Zachs 1979). This indicates that automatic cognitive processing (particularly automatic-alcohol approach responses found in experiment four) will continue to have an effect on older participants and this could result in increased drinking which could, in turn, continue to exacerbate natural decline in executive in an aging population.

A final limitation is that it is difficult to make any inferences about causality in the current research. Although attentional bias (Field & Eastwood 2005) and automatic

approach responses (Wiers et al., 2010) have both been shown to have a causal relationship with ad-lib drinking in the laboratory, there have been failures to replicate these findings (Field et al., 2007b). The current thesis is silent upon whether these processes actually cause hazardous drinking behaviour or whether they are simply epiphenomena of heavy drinking. Large scale longitudinal studies are required in order to ascertain if hazardous drinking is a direct consequence of increased cognitive bias and impulsivity, or whether drinking causes increases in these variables.

8.4. Future Research

One important outcome of the current thesis is the finding that in different age groups different measures of automatic cognition are related to hazardous drinking. It seems that in younger populations attentional bias is more strongly associated with hazardous drinking (studies two and three). Indeed, at risk adolescents have also been found to have increased attentional bias before drinking has been initiated (O'Connor et al., 2007). In the sample of older adults automatic approach responses towards alcohol-related cues, but not attentional bias, was associated with hazardous drinking. Future research could investigate further what specific aspects of cognitive bias are associated with drinking in different populations. This in turn would allow treatment programs that aim to modify cognitive bias to be improved and target different populations to increase the efficacy of such interventions.

Recently, MacKillop et al. (2011) demonstrated that impulsive decision making is more strongly related to alcohol consumption in clinical samples than in normal populations. A meta-analysis of inhibitory control studies would also be a useful tool for addiction researchers. It is possible that, like delay discounting, deficits in inhibitory control become an increasingly important factor in the aetiology of addiction as an individual moves from social drinking to alcohol abuse and onto dependence. Another important consideration for future research is that the definition of impulsivity is not lost when attempting to operationalise 'impulsivity' for experimental purposes. The assumption in much current research, and indeed in

studies one and two, is that hazardous drinking is a consequence of impulsivity. This may only be the case in certain populations; students may be drinking to a social norm and therefore not be failing in their attempts to control behaviour, as no attempt to control their drinking is ever made. This may account for the null findings in the adolescent and student samples, and the strong association between both delay discounting and inhibitory control and hazardous drinking in the sample of older adults. Future research could therefore focus on investigating populations which would be motivated to control their drinking. Alternatively, increased impulsivity may be a consequence of heavy drinking. This would also account for the lack of a relationship between behavioural impulsivity and drinking in adolescents and students (who would have relatively short drinking histories) compared to the strong associations between impulsivity and hazardous drinking in older adults, as well as being consistent with MacKillop et al. (2011). Again, longitudinal studies investigating how behavioural impulsivity changes over time, in conjunction with in depth assessment of drinking motives, socio-economic status and attempts to control drinking would be required to investigate this.

Both these (overlapping) research areas need to take into account some of the issues raised in the previous section. Firstly, the issue of cross sensitisation needs to be taken into account when investigating which aspects of automatic cognition predict hazardous drinking. It is possible that some age groups are also more likely to be using other dopaminergic drugs such as cocaine which may result in increased cognitive bias towards alcohol-related stimuli as a result of cross sensitization. This could result in increased cognitive bias in heavy drinkers taking other drugs compared to heavy drinkers not using dopaminergic drugs, as well as lighter drinkers (who take other drugs) showing cognitive bias towards alcohol-related stimuli. Indeed, the impact of cross sensitisation is one that could be explored in isolation. It would be interesting to investigate if concurrent use of dopamine agonists increased both attentional bias and automatic approach responses to the same extent within social drinkers.

Recent accounts of cognitive aging should also be taken into account in future research. Indeed a fundamental challenge for researchers may be to disentangle the effects of chronic alcohol use from natural cognitive aging and to see if chronic

alcohol use accelerates the natural deterioration of executive cognitive function. If alcohol use does exacerbate the decline in executive cognitive functioning and automatic cognitive functioning remains intact (Hasher & Zachs 1979), this implies that the predictions of dual process models of cognition may be supported in elderly samples with a long history of chronic alcohol use.

Although the current thesis did not find much support for dual process models of addiction future research can develop these models. As previously suggested when discussing behavioural impulsivity, some populations may show behaviour more consistent with the predictions of dual process models. It seems that adolescent samples display behaviour most consistent with the specific predictions of these models. It would therefore be interesting to utilise state manipulations (such as ego depletion) in these populations, to further investigate dual process models of addiction.

Finally, recent research by Miyake and colleagues has suggested that working memory/updating is the key aspect of executive cognitive functioning, and other aspects such as inhibitory control contributes no unique variance to executive cognitive functioning. Much of the current research investigating dual process models of cognition and especially the alcohol priming effect has concentrated upon inhibitory control. In light of the work of Miyake and colleagues it seems pertinent that future studies investigating the impact of executive cognitive function on alcohol use (alone and from the perspective of dual process models) should use a measure of working memory as well as inhibitory control as it would be expected that inhibitory control would not predict any unique variance in alcohol use beyond that explained by updating. This would be most important in priming studies which have almost exclusively concentrated on inhibitory control (with some notable exceptions e.g. Balodis et al., 2007; Guillot et al., 2010). It is possible that studies which have attributed the priming effect to impaired inhibitory control (Weafer & Fillmore 2008) may have found comparable results if they had used a measure of working memory. However it is also possible that inhibitory control may be affected differently by an alcohol prime than working memory and in these circumstances inhibitory control would predict unique variance in alcohol seeking behaviours.

8.5. Clinical implications

The finding regarding different aspects of attentional bias and their association with hazardous drinking has potentially important clinical implications. The current thesis found that maintenance of attention on alcohol related cues was associated with hazardous drinking in non-dependent samples. Other research (Vollstädt-Klein et al., 2009; Townshend & Duka 2007) has found that initial orientation of attention towards, but not maintenance of attention on, alcohol-related cues is associated with drinking in alcohol-dependent samples. This suggests that attentional retraining interventions (e.g. Schoenmakers et al., 2010) that focus on reducing drinking in dependent samples may be increasingly efficacious if they concentrate upon retraining the initial orientation of attention away from alcohol-related stimuli, while interventions in hazardous drinkers should concentrate on disengaging attention. Furthermore, the current thesis highlights the importance of which aspects of cognitive bias should be modified. The retraining of automatic approach responses (e.g. Wiers et al., 2011) may be a more efficacious intervention in older drinkers whereas retraining attentional bias may be a more successful intervention in younger drinkers. Indeed, large scale randomised controlled treatment trials are required for the efficacy of such interventions to be fully ascertained.

The findings regarding dual process models of addiction in adolescents may be of particular importance. It has been argued that adolescence represents a critical period for the development of hazardous drinking patterns (Dayan et al., 2010; Gladwin et al., 2011). The current results (as well as those of Grenard et al., 2008; Thush et al., 2008) suggest that targeting adolescents with increased cognitive bias (specifically attentional bias or alcohol-positive/arousal associations) towards alcohol-related cues and conducting interventions aimed at improving working memory and reducing delay discounting may be efficacious in reducing dangerous patterns of drinking. Indeed, recent research by Bickel et al. (2011) suggests that working memory training

reduces delay discounting rates in stimulant users suggesting that both these aspects of controlled processes could be targeted in one intervention. There is already some evidence for the efficacy of such training programs. Houben et al. (2011) found that working memory training reduced problem drinking, and that the specific mechanism for this improvement was a reduction in the impact of automatic processes as would be predicted by dual process models of addiction.

8.6. Concluding comments

The current thesis aimed to explore the contribution of automatic cognitive processes and behavioural impulsivity to hazardous drinking from the perspective of dual process models of addiction. The findings suggest that there is relatively limited support for the predictions of dual process models, that impulsivity will moderate the impact of automatic cognitive processes on hazardous drinking, except for in the adolescent sample. Indeed, even after priming doses of alcohol and the depletion of self control resources there was no evidence for increased behavioural control by automatic cognitive processes as would be predicted by these models. There was however consistent support for incentive-motivational and cognitive models of addiction as all the cross sectional studies showed some evidence for a direct association between automatic cognitive processing of alcohol-related cues and hazardous drinking suggesting that these processes play a role in problem drinking. Behavioural impulsivity was independently associated with hazardous drinking in older adults suggesting that these cognitive processes have different impacts on drinking behaviour dependent upon age and possibly social factors. Overall, the experiments in this thesis suggest that continued refinement of measurement tools and targeting specific groups of participants, specifically moving away from student samples, is required before the contribution of dual process models to our understanding of addiction can be fully evaluated.

Table 8.1: Summary of tasks used throughout the thesis

Measure	Construct measured	Dependent variable	Key feature(s)
Attentional cueing task	Attentional bias	Reaction time	Attentional bias to single cue, variable SOA
Stimulus response compatibility	Association strength	Reaction time	Symbolic approach avoid responses, relevant feature categorization
Visual probe	Attentional bias	Reaction time/gaze dwell time	Attentional bias to competing cues, variable SOA
Go/No-Go (passive avoidance)	Inhibitory control	Errors (respond to No-Go cue)	Learn correct numbers (4/8), respond rapidly to them
Antisaccade	Inhibitory control	Errors (looking at antisaccade cues)	Inhibit ocular responses to peripherally appearing cues
Cued Go/No-Go	Inhibitory control	Errors (respond to No-Go cue)	Respond to Go stimuli only ; cue indicating a 80% probability of a Go or No-Go trial appears before the stimuli.
Stop Signal task	Inhibitory control	Stop-Signal reaction time (SSRT)	Respond to stimuli rapidly; adjusting tone indicates when not to respond
Delay discounting (questionnaire)	Decision making	Area under the curve (AUC)	Choice between fixed delayed amount or a series of smaller immediate rewards, delay to fixed reward changes in each block.

Table 8.1 (continued)

Measure	Construct measured	Dependent variable	Key feature(s)
Delay discounting (computerised)	Decision making	Area under the curve (AUC)	Choice between fixed delayed amount or a series of smaller immediate rewards, immediate reward adjusts according to previous choice, delay to fixed reward changes in each block .
Two Choice decision task	Decision making	Mean delay to reward	Experience delays for points fixed numbers of points. Delays adjust according to participants preference for long delays (more points) or short delays (fewer points).
COWAT	Multiple aspects of ECF	Switches between word groups	Participants state as many words as they can beginning with a selected letter (3 letters used in total)
Time estimation task	Time estimation	Mean estimate	Estimate the passage of a period of time e.g. 1 minute 5 times

COWAT = controlled oral word association test

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Appendix 1 (AUDIT)

AUDIT

1) How often do you have a drink containing alcohol?

Never Less than monthly 2-4 times a month 2-3 times per week 4+per week

2) How many drinks containing alcohol do you have on a typical day when you're drinking?

1-2 3-4 5-6 7-9 10+

3) How often do you have 6 or more drinks on one occasion?

Never Less than monthly Monthly Weekly Daily or almost daily

4) How often during the last year have you found that you were not able to stop drinking once you had started?

Never Less than monthly Monthly Weekly Daily or almost daily

5) How often during the last year have you failed to do what was normally expected from you because of drinking?

Never Less than monthly Monthly Weekly Daily or almost daily

6) How often during the last year have you needed a drink first thing in the morning to get yourself going after a heavy drinking session?

Never Less than monthly Monthly Weekly Daily or almost daily

7) How often during the last year have you had a feeling of guilt or remorse after drinking?

Never Less than monthly Monthly Weekly Daily or almost daily

8) How often during the last year have you been unable to remember what happened the night before because you had been drinking?

Never Less than monthly Monthly Weekly Daily or almost daily

9) Have you or someone else been injured because of your drinking?

No Yes, but not in the last year Yes, during the last year

10) Has a relative, friend, doctor or other health worker been concerned about your drinking or suggested you cut down?

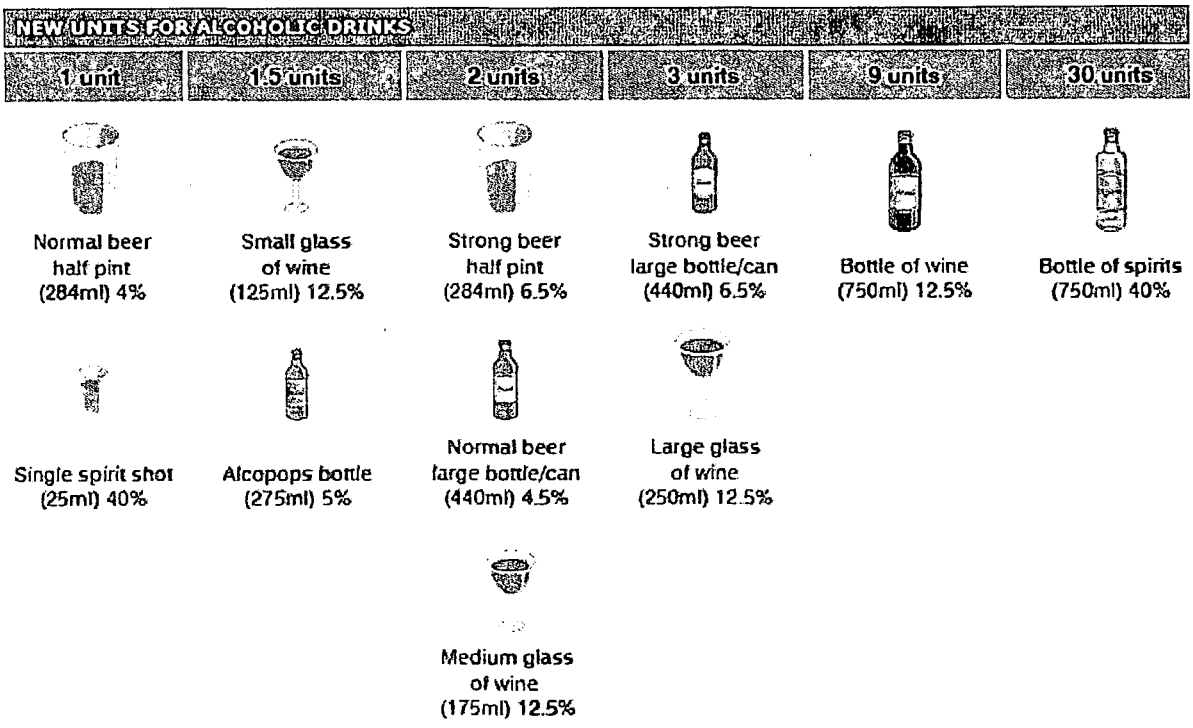
No Yes, but not in the last year Yes, during the last year

Appendix 2 (TLFB-1 week)

Timeline Followback

To help me evaluate your drinking I need to get an idea of your alcohol consumption in the past seven days. Please fill out the table with the number of units of alcohol consumed on each day, being as accurate as possible. Please use the information given below to work out how many units you consumed on each day in the past week and fill in the number of units in the table. On days when you did not drink please write 0 (zero). I realise it isn't easy to recall things with 100% accuracy, but if you are not sure how many units you drank on a certain day please try to give it your best guess.

What is a unit of alcohol?



SOURCE: Office for National Statistics

Please now fill in the following table stating the total number of alcohol units you consumed for each day. Please start from whichever day it was yesterday and work backwards. For example if today is Monday start from Sunday and work backwards, with Monday being Monday a week ago. Once you have completed this please answer the statements below the table. Please double check that you have filled in the number of units for all seven days.

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

Weekly total: ____ units

Was this 'typical' of your normal weekly alcohol consumption? YES / NO

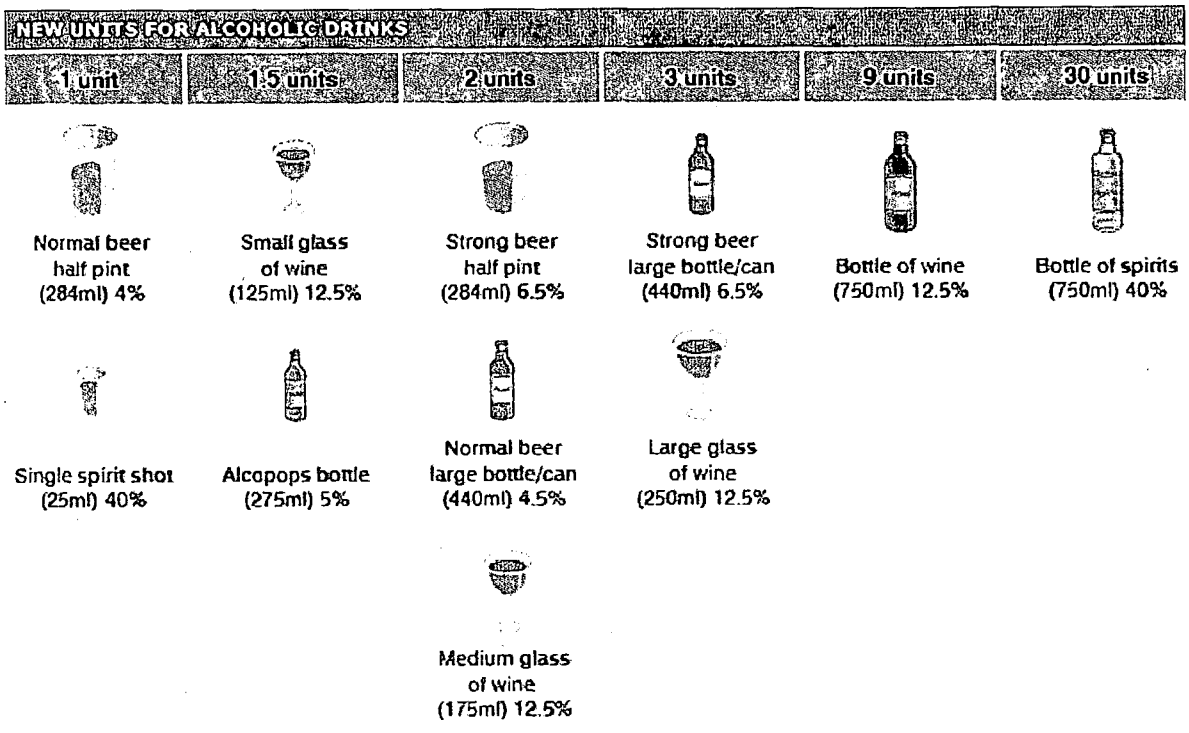
If no, how many units do you normally drink per week? ____ units

Appendix 3 (TLFB-2 week)

Timeline Followback

To help me evaluate your drinking I need to get an idea of your alcohol consumption in the past seven days. Please fill out the table with the number of units of alcohol consumed on each day, being as accurate as possible. Please use the information given below to work out how many units you consumed on each day in the past week and fill in the number of units in the table. On days when you did not drink please write 0 (zero). I realise it isn't easy to recall things with 100% accuracy, but if you are not sure how many units you drank on a certain day please try to give it your best guess.

What is a unit of alcohol?



Please now fill in the following table stating the total number of alcohol units you consumed for each day. Please start from whichever day it was yesterday and work backwards. For example if today is Monday start from Sunday and work backwards, with Monday being Monday a week ago. Once you have completed this please answer the statements below the table. Please double check that you have filled in the number of units for all seven days.

Last week:

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

Appendix 3 (TLFB-2 week)

Previous week:

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

Weekly total: ____ units

Was this 'typical' of your normal weekly alcohol consumption? YES / NO

If no, how many units do you normally drink per week? ____ units

Appendix 4 (BIS-11)

Directions: People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement and place a check in the appropriate box on the right side of the page. Do not spend too much time on any statement. Answer quickly and honestly.		**Rarely/Never**	**Occasionally**	**Often**	**Almost always/ Always**
1.	I plan tasks carefully				
2.	I do things without thinking				
3.	I am happy-go-lucky				
4.	I have "racing" thoughts				
5.	I plan trips well ahead of time				
6.	I am self-controlled				
7.	I concentrate easily				
8.	I save regularly				
9.	I find it hard to sit still for long periods of time				
10.	I am a careful thinker				
11.	I plan for job security				
12.	I say things without thinking				
13.	I like to think about complex problems				
14.	I change jobs				
15.	I act "on impulse"				
16.	I get easily bored when solving thought problems				
17.	I have regular medical/dental checkups				
18.	I act on the spur of the moment				
19.	I am a steady thinker				
20.	I change where I live				
21.	I buy things on impulse				
22.	I finish what I start				
23.	I walk and move fast				
24.	I solve problems by trial-and-error				
25.	I spend or charge more than I earn				
26.	I talk fast				
27.	I have outside thoughts when thinking				
28.	I am more interested in the present than the future				
29.	I am restless at lectures or talks				
30.	I plan for the future				

Appendix 5 (adolescent problem index)

Have any of the events in the list below happened to you after you have been drinking alcohol?:

1. I got into an argument. ☐
2. I got into a fight. ☐
3. I had to be taken to hospital. ☐
4. I damaged my clothes or other items. ☐
5. I lost items or other items. ☐
6. I got into trouble with the police. ☐

10. If you drink alcohol ***regularly***, like every couple of weeks, then we would like to know how much alcohol you normally drink. Remember, we will not tell anyone what you write and we will not judge your answers.

To help us measure how much you drink we need to get an idea of what you drank in the ***past two weeks***. Think about the last time you drank alcohol. What did you drink? How much did you drink? Write down what you drank in the space below. If you remember what brand you drank (like Stella lager or White Lightning cider) or the percentage alcohol it contained then write that down too. For example, if you shared a bottle of cider with friends then write down the size of the bottle and how much of it you drank. If you don't remember all these details write down as much as you do remember. Then think about the time before that when you drank alcohol and fill out the details. Try to do this for the last two weeks. The examples shown below are half a bottle of cider shared with friends on Friday, and a can of lager drunk on Thursday. If you need any help then raise your hand.

Which day?	Type of alcohol	Brand	Alcohol %	Bottle or can size	How much did you drink?
Examples: Friday	**Cider**	**White lightning**	**7.5**	**One litre**	**Half a bottle**
Thursday	**Lager**	**Stella**	**?**	**?**	**1 can**

If you answered YES, please answer the questions below

When was the last time you drank alcohol? (tick ONE box only):

- During the last week ☐
- One week to four weeks ago ☐
- One month to six months ago ☐
- More than six months ago ☐

How often do you drink alcohol? For example you might have tried it only once, or you may drink a few times a year, or more often (tick ONE box only):

- Almost every day ☐
- About twice a week ☐
- About once a week ☐
- About once a fortnight ☐
- About once a month ☐
- A few times a year ☐
- Other (please explain below):

_____

How many times have you been drunk since January?

_____

How old were you when you had your *first* alcoholic drink (a drink larger than a sip?:

_____

Appendix 7 (Family affluence scale)

1. How old are you?

2. Are you male or female? (circle one)

Male Female

3. Does your family have a car or a van? (circle one)

No One Two or more

4. Do you have your own bedroom? (circle one)

No Yes

5. During the past year, how many times did you travel away on holiday with your family? (circle one)

Not at all Once Twice More than twice

Temptation and Restraint Inventory

Instructions: Please read each of the following questions carefully. Circle the number that represents your answer to each question. BE SURE TO CIRCLE ONLY ONE NUMBER FOR EACH QUESTION.

Remember that your honest response -- the one that makes the most sense to you personally is the response we want. Don't worry about how other people would answer, we want your views. Please work as quickly as you can, while giving the most honest and accurate answer you can to each question. In general, your first impressions are the best.

1. When you feel anxious, are you more likely to drink?

1	2	3	4	5	6	7	8	9
Never								Always

2. When you feel lonely, are you more likely to drink?

1	2	3	4	5	6	7	8	9
Not at all								Extremely

3. How often do you attempt to cut down the amount you drink?

1	2	3	4	5	6	7	8	9
Never								Always

4. At times, do you find yourself unable to stop thinking about drinking?

1	2	3	4	5	6	7	8	9
Never								Always

5. Does seeing other people drink remind you of your efforts to control your alcohol consumption?

1	2	3	4	5	6	7	8	9
Never								Always

6. Do you ever feel so nervous that you really need a drink?

1	2	3	4	5	6	7	8	9
Never								Always

7. Do thoughts about drinking intrude into your daily activities?

1	2	3	4	5	6	7	8	9
Never								Always

8. Does seeing alcohol-related commercials, magazine ads., and/or signs for liquor stores

stimulate concerns about the need to limit your drinking?

1	2	3	4	5	6	7	8	9
Never								Always

9. Do you find that once you start drinking it is difficult for you to stop?

1	2	3	4	5	6	7	8	9
Never								Always

10. Do feelings of guilt about drinking too much help you to control your alcohol intake?

1	2	3	4	5	6	7	8	9
Never								Always

11. Is it hard to distract yourself from thinking about drinking?

1	2	3	4	5	6	7	8	9
Never								Always

12. Does the sight and smell of alcohol make you think about limiting your drinking?

1	2	3	4	5	6	7	8	9
Never								Always

13. How much difficulty do you have controlling your drinking?

1	2	3	4	5	6	7	8	9
None								A Great Deal

14. Do you ever cut back on your drinking in an attempt to change your drinking habits?

1	2	3	4	5	6	7	8	9
Never								Always

15. How much effort does it take for you to keep your drinking under control?

1	2	3	4	5	6	7	8	9
None								A Great Deal

Appendix 9 (DAQ)

Please indicate how much you agree or disagree with each of the following statements by placing a single mark along each line. Please complete every item. We are interested in how you are thinking or feeling right now as you fill out the questionnaire.

RIGHT NOW

1. I would accept a drink now if it was offered to me

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

2. I would feel as if all the bad things in my life had disappeared if I drank now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

3. I could easily limit how much I would drink if I drank now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

4. My desire to drink now seems overwhelming

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

5. Even major problems in my life would not bother me if I drank now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

6. Drinking now would make me feel less tense

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

7. Drinking would be satisfying now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

8. I would do almost anything to have a drink now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

9. I would consider having a drink now

   STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

10. I want a drink so much I can almost taste it

    STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

11. Drinking would be pleasant now

    STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

12. I would feel less worried about my daily problems if I drank now

    STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

13. I am going to drink as soon as I possibly can

    STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

14. If I started drinking now I would be able to stop

    STRONGLY DISAGREE ___:___:___:___:___:___:___ STRONGLY AGREE

Appendix 10 (SIS)

Subjective intoxication scales

This questionnaire is concerned with how you feel *right now*.
Please place a mark on each line to indicate how you feel on each dimension.

Light headed

Not at all Slightly Moderately Quite a lot Extremely

Irritable

Not at all Slightly Moderately Quite a lot Extremely

Stimulated

Not at all Slightly Moderately Quite a lot Extremely

Alert

Not at all Slightly Moderately Quite a lot Extremely

Relaxed

Not at all Slightly Moderately Quite a lot Extremely

Contented

Not at all Slightly Moderately Quite a lot Extremely

Appendix 11 (taste test 1)

Drink taste test

We would like you to taste each of the drinks and then rate them based on the criteria below by placing a mark on the lines. You may drink as much or as little of each drink as you please in order to make accurate ratings.

Orange juice

Unpleasant _____ Pleasant

Tasteless _____ Strong tasting

Bitter _____ Sweet

Flat _____ Gassy

Lager

Unpleasant _____ Pleasant

Tasteless _____ Strong tasting

Bitter _____ Sweet

Flat _____ Gassy

Appendix 12 (BMIS)

INSTRUCTIONS: Circle the response on the scale below that indicates how well each adjective or phrase describes your **present mood.**

1 = definitely do not feel / 2 = do not feel / 3 = slightly feel / 4 = definitely feel

1. Lively	1	2	3	4
2. Drowsy	1	2	3	4
3. Happy	1	2	3	4
4. Grouchy	1	2	3	4
5. Sad	1	2	3	4
6. Peppy	1	2	3	4
7. Tired	1	2	3	4
8. Nervous	1	2	3	4
9. Caring	1	2	3	4
10. Calm	1	2	3	4
11. Content	1	2	3	4
12. Loving	1	2	3	4
13. Gloomy	1	2	3	4
14. Fed up	1	2	3	4
15. Jittery	1	2	3	4
16. Active	1	2	3	4

Overall, my mood is:

Very Unpleasant Very Pleasant

-10 –9 –8 –7 –6 –5 –4 –3 –2 –1 0 1 2 3 4 5 6 7 8 9 10

Appendix 13 (Ego depletion manipulation check 1+2)

S1
Number _____

Please answer the questions below by circling one of the numbers.

1. How much effort did you exert suppressing negative emotions?

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

None at all All of my effort

2. How difficult did you find suppressing negative emotions?

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely difficult

3. How emotional did you find the clip?

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely

Please state how much you agree with the following statements by circling one of the numbers:

4. After watching the clip I felt emotionally drained

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely

5. After watching the clip I felt tired

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely

Appendix 13 (Ego depletion manipulation check 1+2)

S1
Number _____

Please answer the questions below by circling one of the numbers.

6. How much effort did you exert suppressing thoughts associated with the clip?

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

None at all All of my effort

7. How difficult did you find suppressing thoughts associated with the clip?

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely difficult

Please state how much you agree with the following statements by circling one of the numbers:

1. After completing the thought listing task I felt emotionally drained

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely

2. After completing the thought listing task I felt tired

1__2__3__4__5__6__7__8__9__10__11__12__13__14__15__16__17__18__19__20__21__22__23__24__25

Not at all Extremely

Appendix 14 (Ego depletion Taste test)

Ppt Num

Please consume as much or as little of each drink as you like in order to give your valid assessment for the questions used below. Circle your response for each item. You can drink all of the beer if you wish although you will not be given any additional beer if you finish the glass. You can sample a beer and complete all of the questions for that beer before moving on to the next beer, or you can sample each beer in succession for each question.

You have 30 minutes to complete this task. If you finish before the 30 minutes feel free to read the magazines provided.

How smooth was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How sweet was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How refreshing was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How bitter was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

Appendix 14 (Ego depletion Taste test)

How strong tasting was each drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How gassy was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How bland was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

How light was the drink?

0	1	2	3	4	5	6	7	8	9	10	Beer1
0	1	2	3	4	5	6	7	8	9	10	Beer2
0	1	2	3	4	5	6	7	8	9	10	Beer3

0= Not at all 10= Extremely.

Appendix 14 (Ego depletion Taste test)

Please rank each of the beers in order of preference (with 'a' being your favourite and 'c' being your least favourite):

(a) Beer___

(b) Beer___

(c) Beer___

Please rank each of the beers in order of alcohol content (with 'a' being the beer you believe contains the most alcohol and 'c' the least alcohol):

(a) Beer___

(b) Beer___

(c) Beer___

If you think you know what brand any of the beers are please indicate below:

Beer 1 _____

Beer 2 _____

Beer 3 _____

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ORIGINAL INVESTIGATION

Components of behavioural impulsivity and automatic cue approach predict unique variance in hazardous drinking

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Abstract
Rationale Hazardous drinking is associated with both increased impulsivity and automatic approach tendencies elicited by alcohol-related cues. However, impulsivity is a multi-factorial construct, and it is currently unclear if all components of impulsivity are associated with heavy drinking. Furthermore, emerging evidence suggests that the relationships between hazardous drinking and automatic alcohol cognitions may be moderated by individual differences in impulsivity.
Objectives The aim of this study was to investigate the independence of measures of impulsivity and their association with hazardous drinking, and to examine if the relationship between hazardous drinking and automatic alcohol approach tendencies would be moderated by individual differences in impulsivity.
Methods Ninety-seven social drinkers (65 female) completed questionnaire measures of trait impulsivity, alcohol consumption and hazardous drinking. Participants also completed computerised measures of automatic alcohol approach tendencies (stimulus–response compatibility (SRC) task), and two behavioural measures of impulsivity (Go/No-go and delay discounting tasks).
Results Principal component analysis revealed that the two measures of behavioural impulsivity were distinct from each other and from self-reported trait impulsivity, although self-reported non-planning impulsivity loaded on to two factors (trait impulsivity and delay discounting). Furthermore, all measures of impulsivity predicted unique variance in hazardous drinking as did automatic alcohol approach tendencies, although the latter relationship was not moderated by impulsivity.
Conclusions These results indicate that multiple components of impulsivity and automatic alcohol approach tendencies explain unique variance in hazardous drinking.

Keywords Alcohol · Cognitive bias · Delay discounting · Impulsivity · Inhibitory control

Introduction

Contemporary theories of addiction propose that both increased impulsivity and heightened salience of alcohol-related cues play a central role in alcohol use disorders. For example, elevated impulsivity has been closely linked with alcohol use disorders, and it may play a causal role in loss of control over drinking (de Wit 2009). Likewise, it has been shown that alcohol-related cues acquire conditioned incentive motivational properties ('incentive salience'), causing those cues to capture attention and initiate approach behaviours automatically, ultimately leading to alcohol consumption (Robinson and Berridge 2001). Recent theoretical models (Goldstein and Volkow 2002; Jentsch and Taylor 1999; Wiers et al. 2007) make more detailed predictions about how impulsivity and incentive salience might interact during the development of alcohol use disorders. For example, Wiers et al. (2007) suggested that approach behaviour automatically elicited by alcohol cues should result in increased alcohol consumption, but this effect should be moderated by individual differences in impulsivity, with highly impulsive individuals more sensitive to the incentive properties of alcohol cues. In support

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of this model, recent experimental studies have demonstrated that measures of impulsivity and executive (dys) function moderate the association between automatic processing of alcohol-related cues and individual differences in drinking behaviour (for a review, see Stacy and Wiers 2010).

Although impulsivity has commonly been associated with alcohol and other drug use disorders, there is no consensus on how best to conceptualise and measure the construct. For example, questionnaire measures assume that impulsivity is a relatively stable trait, whereas behavioural measures allow for the assessment of individual differences in impulsivity that may also fluctuate within individuals (e.g. Gauggel et al. 2010; Jones et al. 2011). Both de Wit and Richards (2004) and Olmstead (2006) have argued for two distinct components of impulsivity, which can be directly measured with behavioural tasks rather than relying on self-report questionnaires. The first component is impulsive decision making, in which individuals are oversensitive to immediate rewards but insensitive to delayed rewards or negative consequences. This is commonly measured using the delay discounting procedure (e.g. Madden et al. 1997). In this task, participants are given a series of choices between small sums of money which are available immediately, versus larger sums of money which are available after a delay. There is some evidence that heavy social drinkers and alcoholics show an increased rate of delay discounting (i.e. preference for smaller immediate rewards) compared to light drinker controls (Field et al. 2007; Petry 2001; Vuchinich and Simpson 1998). However, several studies have revealed no association between delay discounting rate and individual differences in alcohol consumption or alcohol-related problems (Fernie et al. 2010; Kirby and Petry 2004; MacKillop et al. 2007). In a recent meta-analysis, MacKillop et al. (2011) found that although there is evidence for elevated discounting of future rewards in clinical samples of alcohol abusers, it is much less pronounced in non-clinical samples. One explanation for this is that non-clinical samples may not contain enough participants with a significant drinking history to be exhibiting increased discounting of future rewards. Furthermore, many negative findings are in experiments conducted on samples of largely undergraduate drinkers (e.g. Fernie et al. 2010; MacKillop et al. 2007). Undergraduates have been shown to be motivated to drink by social factors (e.g. Faulkner et al. 2006; Wicki et al. 2010); therefore, undergraduate alcohol consumption behaviour may not be associated with individual differences in impulsivity, but a decision facilitated by conformity to a social norm.

The second, independent component of impulsivity defined by de Wit and Richards (2004) and Olmstead (2006) is deficient inhibitory control. Inhibitory control refers to the ability to control or suppress prepotent responses. This construct has been assessed with behavioural tasks including Go/No-Go (Newman and Kosson 1986) and Stop signal (Logan et al. 1984) tasks, both of which involve the suppression of prepotent motor responses. Some recent studies suggest that heavy drinking and alcoholism are associated with failures of inhibitory control on these tasks (Go/No-Go task, Colder and O'Connor 2002; Stop signal task, Goudriaan et al. 2006). Recently, Jones et al. (2011) found that induction of a disinhibited state resulted in increased alcohol consumption, relative to a control manipulation, which suggests a causal effect of disinhibition on alcohol-seeking. However, as with the delay discounting data, the findings are not consistent across studies. For example, Kamarajan et al. (2005) did not detect a selective impairment in response inhibition among alcoholics compared to controls in a study that utilised a Go/No-Go task, and Fernie et al. (2010) found no association between alcohol consumption and performance on a stop-signal task among young adult social drinkers.

In addition to behavioural measures of impulsivity, self-report measures have also been developed; these treat impulsivity as a stable personality trait that can be assessed with questionnaires. These measures, such as the Barratt Impulsivity Scales (BIS; Patton et al. 1995) require individuals to indicate their degree of endorsement of statements such as 'I do things without thinking'. Furthermore, these measures are split into subscales which assess different aspects of trait impulsivity, e.g. motor, attentional and non-planning impulsivity. Cross-sectional and longitudinal studies suggest some correspondence between elevated trait impulsivity and increased alcohol consumption or alcohol problems, in both adolescents and adults (Fernie et al. 2010; Gunnarsson et al. 2008; McAdams and Donnellan 2009; Von Diemen et al. 2008; Von Knorring et al. 1987). Indeed, evidence for the association between trait impulsivity and alcohol use has been more consistently demonstrated than for either measure of behavioural impulsivity.

There is a growing body of evidence demonstrating a clear dissociation between measures of impulsive decision making and inhibitory control. Reynolds et al. (2006) used principal component analysis to investigate the independence of measures of behavioural impulsivity and found Stop and Go/No-go tasks loaded on to a separate factor (impulsive disinhibition) from delay discounting and risk-taking tasks (impulsive decision making). Likewise, Swann et al. (2002) found two distinct components of behavioural impulsivity: 'rapid response' and 'reward-delay impulsivity'. The relationship between these two behavioural measures of impulsivity and trait measures is less clear. White et al. (1994) found trait impulsivity to be a separate construct from behavioural impulsivity, and Reynolds et al. (2006) also found that behavioural and trait measures were distinct. Swann et al. (2002) reported significant correlations between trait impulsivity and both

reward-delay sensitivity and rapid response impulsivity, although the latter was more strongly associated with trait measures. Furthermore, de Wit et al. (2007) reported a significant correlation between the non-planning subscale of the BIS and impulsive decision making and suggested that both these measures reflect insensitivity to delayed rewards. It is currently unclear how much unique variance each of the discussed types of impulsivity contributes towards hazardous drinking in non-dependent adult drinkers.

Although the literature regarding associations between impulsivity and heavy drinking is inconsistent, the literature concerning incentive motivational properties of alcohol cues is much clearer. The incentive salience of alcohol-related cues has been measured with a variety of cognitive tasks, which have been adapted from those used in mainstream experimental psychology. For example, studies of selective attention for alcohol-related cues ('attentional bias') suggest that such cues tend to 'grab the attention' among alcoholics and heavy social drinkers (Stetter et al. 1995; Townshend and Duka 2001; for a recent review, see Field and Cox 2008). Individual differences in attentional bias for alcohol-related cues prospectively predict alcohol use among heavy drinking university students (Fadardi and Cox 2008), and the degree of attentional bias predicts relapse or treatment dropout among treatment-seeking alcoholics (Cox et al. 2002). With regard to overt behavioural approach elicited by alcohol cues, it has been demonstrated that animals will direct approach behaviours towards cues that have been paired with the availability of alcohol (Krank et al. 2008), and several investigators have developed experimental paradigms for studying cue-elicited approach in humans. For example, we have adapted the stimulus response compatibility (SRC) task (De Houwer et al. 2001) to study the speed at which heavy and light social drinkers' direct approach and avoidance responses towards alcohol-related pictorial cues. In the SRC task, alcohol-related or alcohol-unrelated (control) pictures are presented on a computer screen alongside a manikin. In one phase of the task (the 'approach alcohol' block), participants are required to rapidly move the manikin towards alcohol-related pictures and away from control pictures; in another phase of the task (the 'avoid alcohol' block), participants are required to move the manikin towards the control pictures and away from the alcohol-related pictures. Heavy, but not light drinkers, respond more rapidly during the 'approach alcohol' block compared to the 'avoid alcohol' block, which suggests that alcohol-related cues elicit an automatic approach tendency among such heavy drinkers (Field et al. 2008; Field et al. in press). Palfai and Ostafin (2003) and Wiers et al. (2009) obtained comparable findings using similar tasks. In a recent manipulation study, Wiers et al. (2010) found that these automatic approach tendencies had a causal influence on the motivation to drink alcohol in the laboratory. In addition, Wiers et al. (2011) found that retraining alcoholics to avoid alcohol related cues led to improved treatment outcomes. This suggests that automatic approach bias, like attentional bias, can drive alcohol consumption and is not merely an epiphenomenon of heavy drinking. Finally, as predicted by dual process models of addiction (e.g. Wiers et al. 2007), associations between other measures of automatic alcohol cognitions and drinking behaviour are moderated by individual differences in inhibitory control (Houben and Wiers 2009), as well as other aspects of executive cognitive function, such as working memory (Thush et al. 2008). These studies revealed that drinking behaviour of individuals with better executive functioning is weakly associated with, or unrelated to, automatic processing of alcohol-related cues, whereas these relationships are much stronger in individuals with poor executive functioning.

In the present study, we used principal component analysis to investigate the independence of behavioural (delay discounting and a Go/No-Go task) and trait measures of impulsivity (the BIS). We predicted that this analysis would identify two distinct components of behavioural impulsivity, with trait impulsivity emerging as an additional distinct factor. Following this, we investigated the relative contributions of these measures as well as automatic approach responses elicited by alcohol-related cues (as assessed with the SRC task) as predictors of individual differences in hazardous drinking in a community sample. By utilising a community sample, we hoped to recruit participants with longer drinking histories and reduce the likelihood of participant drinking being facilitated by a social norm. Our primary hypothesis was that alcohol approach tendencies and the measures of impulsivity would explain unique variance in hazardous drinking. We also explored an additional prediction derived from Wiers et al. (2007), which was that the behavioural impulsivity measures would moderate the association between approach tendencies and hazardous drinking.

Method

Participants

Ninety-seven participants (65 female) aged between 18 and 59 years (mean 28.95±11.57) participated in the experiment. Inclusion criteria included regular consumption of alcohol (as confirmed by AUDIT scores >1 in all participants). Potential participants were excluded if they self-reported a current or past alcohol use disorder, or if their vision was not normal or corrected-to-normal. Participants consumed a mean of 23.31 (±20.36; range 0–93) UK units of alcohol

in the week prior to taking part in the study (1 UK unit=8 g alcohol). Participants were recruited from the staff and student population of the University of Liverpool as well as workplaces within the Liverpool area. Employers were approached by the experimenter, and consent to recruit on the premises was sought from a senior member of staff. All participants provided informed consent, and the study was approved by the University of Liverpool Ethics Committee.

Materials

Questionnaires

Time Line Follow Back (TLFB; Sobell and Sobell 1990). The TLFB is a self-report retrospective diary in which participants recall their alcohol consumption over the previous 7 days.

The Alcohol Use Disorders Identification Test (AUDIT; Saunders et al. 1993). The AUDIT questionnaire consists of ten items regarding alcohol consumption and consequences of drinking. Scores on the AUDIT range between 0 and 40, with scores of 8 or above, indicating hazardous alcohol use.

Barratt Impulsiveness Scale (BIS-11; Patton et al. 1995). This scale is a multidimensional measure of impulsivity which yields scores on three dimensions—attentional, motor and non-planning impulsiveness. The BIS consists of 30 fixed response questions scored from 0–4 with high scores being indicative of increased impulsivity.

Temptation and Restraint inventory (TRI; Collins and Lapp 1992). This scale measures preoccupation with and attempts to limit drinking, and consists of two factors 'Cognitive and Emotional Preoccupation' (CEP) and 'Cognitive and Behavioural Control' (CBC). The TRI consists of 15 questions scored from 1 to 9 on a Likert scale.

Pictorial stimuli

A picture set containing 14 alcohol-related pictures and 14 (matched) alcohol-unrelated pictures were used during the SRC task. Alcohol-related pictures consisted of alcohol-related scenes (such as a bottle and a glass of wine presented on a table), the alcohol unrelated pictures were matched to the alcohol pictures on perceptual characteristics but did not contain any alcohol-related cues (e.g. a bottle and a glass of water presented on a table). All the pictures were 100-mm high × 125-mm wide. The picture set was identical to that used by Field and Eastwood (2005) and Field et al. (2008).

Behavioural tasks

Stimulus Response Compatibility (SRC) task (Field et al. 2008) The SRC task was programmed in Inquisit version 1.33 (Millisecond software, 2002). All task parameters were identical to those used by Field et al. (2008). Each trial of the task commenced with the presentation of either an alcohol-related picture or an alcohol-unrelated (control) picture in the centre of the screen along with a small manikin above or below the picture. Participants were instructed to move the manikin either toward or away from the picture by pressing up or down on a two-button response box according to the block instructions. In the 'approach alcohol' block, participants were instructed to move the manikin towards alcohol-related pictures, and away from alcohol-unrelated pictures. These instructions were reversed in the 'avoid alcohol' block. Block order was counterbalanced across participants. Reaction time (the time taken to initiate movement of the manikin) was measured on each trial.

Go/No-Go task (GNG task; Newman and Kosson 1986) This 'passive avoidance' version of the GNG task was programmed in Inquisit version 1.33 (Millisecond software, 2002). The task requires participants to learn through trial and error which numerical stimuli are 'correct' (go cues) and which are 'incorrect' (no-go cues). Participants were instructed to withhold responses to the incorrect stimuli (no-go cues), but respond quickly to correct stimuli (go cues) by pressing the spacebar on the keyboard. On each trial of the task, one of eight two-digit numbers was presented. Four numbers (34, 42, 51, 93) were go cues and four (18, 29, 63, 85) were no-go cues. Participants initially completed eight practice trials, in which each number was presented once, followed by three blocks of experimental trials. Each experimental block consisted of 24 trials in which each of the eight numbers was presented three times each. After completion of each block, participants received feedback on the percentage of correct responses to both go and no-go cues.

Each trial began with the presentation of a white fixation cross in the centre of the screen for 1,000 ms, before a go or no-go cue was presented. Cues remained on the screen until a response or a 3-s timeout period had elapsed. Correct responses to go cues resulted in the text 'Correct!' appearing on the screen in green font for 300 ms. Inappropriate responses to no-go cues resulted in the text 'Wrong!' appearing on the screen in red font for 300 ms. If no response was made, no feedback was given. The primary dependent measure from this task was the number of commission errors, i.e. inappropriate responses to no-go cues, with a high rate of commission errors indicative of impaired inhibitory control.

Delay discounting (DD; Du et al. 2002) A computerised DD task (programmed in Visual Basic 6.0) was used to assess impulsive decision making in response to monetary rewards. The DD methodology was identical to the one

used by Fernie et al. (2010). Participants were presented with the hypothetical choice of receiving £100 at a future date or receiving a smaller amount immediately. The size of the immediate reward was adjusted by either adding 50% of the last adjustment (if the delayed reward was selected) or subtracting 50% of the last adjustment (if the immediate reward was selected). This decreasing adjustment logarithm was used by Du et al. (2002). Participants made six choices for each delay period. Monetary choices were made for delays of 1 day, 1 week, 2 weeks, 1 month and 6 months. Indifference points for each of the seven delays were analysed by computing area under the curve (AUC) values (Myerson et al. 2001). Lower values of AUC indicate steeper delay discounting, or increased impulsive decision making. For consistency with the other behavioural measures, the AUC values were reversed so that higher values represent steeper discounting.

Procedure

All participants were tested in a quiet testing cubicle at the University of Liverpool, or a quiet room within their workplace. Upon arrival, participants provided informed consent before providing a breath sample (all participants provided a zero breath alcohol reading). Participants then completed a battery of cognitive tasks (including SRC, GNG and DD tasks); task order was counterbalanced across participants. Upon completion of the cognitive tasks, participants completed the questionnaire battery (including TLFB, AUDIT, BIS and TRI). Once participants had completed the questionnaire battery, they were thoroughly debriefed. Participants recruited and tested in workplaces received £10 sterling as compensation for their time; participants recruited from the university received either £10 or course credit.

Data reduction and statistical analysis

Before analysis of reaction times from the SRC task, outliers were removed according to criteria used in previous reports (e.g. see Field et al. 2008): reaction times less than 200 ms, greater than 2,000 ms and then those reaction times that were more than 3 standard deviations above the individual mean, were discarded. Reaction times from error trials were also discarded; all data was excluded from two participants due to a high percentage of errors. We then computed mean reaction times for each of the two blocks of the task: the 'approach alcohol' block and the 'avoid alcohol' block. An SRC 'bias score' was calculated by subtracting mean response latency during the 'approach alcohol' block from mean response latency during the 'avoid alcohol' block. Higher SRC bias scores are indicative of an automatic approach tendency elicited by alcohol-related cues.

In order to identify independent dimensions of impulsivity principal component analysis (PCA) was performed. Based upon the recommendations of Jolliffe (1972, 1986), we maintained components which had eigenvalues ≥ 0.7; in addition, a scree plot was also used to confirm the maintenance of components. The ≥ 0.7 cutoff which was selected as Kaiser's rule of maintaining eigenvalues ≥ 1.0 is deemed too conservative in circumstances such as this where we expect independence of behavioural and trait measures (see White et al. 1994) and independence of the two behavioural measures (see Reynolds et al. 2006 and Swann et al. 2002). Furthermore, Kaiser's rule is more useful when applied to factor analysis rather than PCA. Sampling adequacy was established using the Kaiser–Meyer–Olkin measure (KMO), with values between 0.5 and 0.7 being acceptable, 0.7+being good to excellent (Hutcheson and Sofroniou 1999). In order to ensure sufficient correlations between items to conduct the PCA, Bartlett's test of sphericity was also performed. Finally, factor loadings greater than 0.512 were deemed significant based upon our sample size of 97 (see Stevens 2002).

Hierarchical regression analyses for moderation (Baron and Kenny 1986) were used to investigate the contribution of demographic variables (age and gender), SRC bias, self-report impulsivity, delay discounting and inhibitory control to hazardous drinking (AUDIT scores). In order to investigate interaction effects between SRC bias and delay discounting, and SRC bias and inhibitory control, interaction variables were computed by normalising variables (by subtracting the mean) before multiplying them together. Age and gender were entered in the first step of the regression; in the second step, we entered the BIS total scores; SRC bias was entered as the third step; delay discounting (AUC values) and inhibitory control (commission errors in the Go/No-Go Task) were entered in the fourth step. Finally, interactions between SRC bias and delay discounting and SRC bias and inhibitory control were entered as the final step.

Results

Descriptive statistics and inter-correlations between variables

Table 1 shows descriptive statistics and inter-correlations between age, weekly alcohol consumption, scores on the AUDIT, TRI (CEP and CBC subscales), BIS (attentional, motor and non-planning impulsiveness subscales), SRC bias, inhibitory control and delay discounting.

Principal component analysis for dimensions of impulsivity

The PCA was conducted on the behavioural measures of impulsivity and BIS subscales with oblique rotation

Table 1 Descriptive statistics and inter-correlations for age, weekly alcohol consumption, questionnaire measures and experimental tasks

	Mean (±SD)	2	3	4	5	6	7	8	9	10	11
1. Age	28.39 (11.20)	−0.14	−0.32*	−0.02	−0.13	−0.26*	−0.30*	−0.23**	0.07	0.16	−0.21*
2. Weekly alcohol consumption	23.64 (20.83)	–	0.68*	0.55*	0.23**	0.33*	0.14	0.24**	0.10	0.09	0.33*
3. AUDIT	11.87 (6.71)		–	0.61*	0.38***	0.45*	0.26*	0.39*	0.24**	0.04	0.38*
4. TRI CEP	23.55 (12.33)			–	0.44*	0.36*	0.21**	0.24**	0.14	0.02	0.17
5. TRI CBC	17.62 (9.98)				–	0.26*	0.17	0.19**	−0.01	−0.05	0.25**
6. BIS non-planning	25.17 (5.44)					–	0.41*	0.49*	0.10	−0.16	0.43*
7. BIS motor	23.33 (4.80)						–	0.56*	0.05	−0.05	0.11
8. BIS attentional	18.12 (3.18)							–	0.14	−0.04	0.11
9. SRC bias (ms)	35.40 (99.30)								–	−0.14	0.04
10. Inhibitory control (No-Go errors)	11.36 (6.40)									–	−0.20**
11. Delay discounting (AUC)	0.24 (0.23)										–

Weekly alcohol consumption in UK units, 1 unit=8 g alcohol; *AUDIT* alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). Alcohol *TRI* temptation and restraint inventory, range of TRI subscale scores (minimum to maximum); *CEP* cognitive and emotional preoccupation 9 to 81, *CBC* cognitive and behavioural control 6 to 54. *BIS* Barratt Impulsivity Scales, range of BIS subscale scores are (minimum to maximum); non-planning 12 to 48, motor 10 to 40, attentional 8 to 32. SRC bias (ms)=Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. Inhibitory control (No-Go errors)=number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting

*$p<0.01$; **$p<0.05$

(oblimin). The sampling adequacy was deemed to be acceptable (KMO=0.62), and Bartlett's test of sphericity demonstrated that correlations between items were large enough for PCA ($\chi^2(10)=87.57$, $p<0.001$). The PCA revealed three components that explained 82.64% of variance. Table 2 shows the factor loadings following rotation, which suggests that cluster one represents trait impulsivity, cluster two represents impulsive decision making and cluster three represents inhibitory control. Interestingly, the non-planning subscale of the BIS loaded onto both the trait impulsivity and the impulsive decision-making factors.

Multiple regression analyses: predicting variance in AUDIT scores

The full regression model predicted 30% of the variance in AUDIT scores (R^2 adjusted=0.30, $F(8,86)=5.87$, $p<0.001$; Table 2). In this model, age was a significant predictor of AUDIT scores ($\beta=-0.21$, $p=0.036$), with older drinkers having lower scores, although gender was not ($\beta=0.11$, $p=0.24$). Total scores on the BIS were a significant predictor of AUDIT scores ($\beta=0.28$, $p=0.005$), insofar as those with higher BIS total scores had significantly higher AUDIT scores. SRC bias was also a significant predictor of AUDIT scores ($\beta=0.23$, $p=0.018$), as stronger automatic approach tendencies elicited by alcohol cues was associated with higher AUDIT scores. Both inhibitory control ($\beta=0.20$, $p=0.039$) and delay discounting ($\beta=0.27$, $p=0.007$) predicted a significant amount of variance in AUDIT scores. Poor inhibitory control (a higher rate of commission errors in the Go\No-Go task), and a higher rate of delay discounting, were both associated with higher scores on the AUDIT. Finally, the interactions between SRC bias and inhibitory control ($\beta=0.09$, $p=0.37$) and SRC bias and delay

Table 2 Principal component analysis for behavioural measures of impulsivity and BIS subscales ($N=97$)

	Rotated components		
	1	2	3
Eigenvalues	2.13	1.15	0.85
Variance (%)	42.60	23.02	17.02
Go/No-go	0.01	0.00	**0.99**
Delay discounting	−0.13	**0.96**	−0.02
BIS attentional	**0.88**	0.02	−0.02
BIS motor	**0.87**	0.08	−0.03
BIS non-planning	**0.52**	**0.58**	0.02

Factors highlighted load above 0.512 and are considered significant (Stevens 2002)

discounting, (β=0.10, p=0.29), did not explain additional variance in AUDIT scores (Table 3).

Discussion

In the current study, we investigated the independence of behavioural and trait measures of impulsivity. We also assessed the relative contribution of these measures as well as automatic approach tendencies elicited by alcohol-related cues, and interactions between the two, as predictors of hazardous drinking. We hypothesised that the PCA would reveal impulsive decision making, inhibitory control and trait measures to be independent from each other. We also hypothesised that the different measures of impulsivity would explain unique variance in hazardous drinking as would automatic approach tendencies elicited by alcohol related cues. Finally, we tested the specific hypotheses of Wiers et al. (2007) who proposed that the association between hazardous drinking and measures of automatic alcohol cognitions would be moderated by behavioural impulsivity. Results were generally supportive of our hypotheses. The PCA revealed three components of impulsivity (inhibitory control, delay discounting and trait impulsivity) as predicted, although the non-planning subscale of the BIS loaded onto both trait impulsivity and impulsive decision making. Furthermore, delay discounting, inhibitory control, trait impulsivity and automatic approach bias all explained unique variance in hazardous drinking, although the interactions between approach bias and the measures of behavioural impulsivity did not explain additional variance.

Table 3 Regression analysis showing trait and behavioural impulsivity measures, and SRC task approach bias, as predictors of AUDIT scores

Variable	R^2	F change	β
Step 1			
Gender	0.15	$F(2,84)=7.48$*	0.20**
Age			−0.28**
Step 2			
Gender	0.25	$F(1,83)=11.62$*	0.18**
Age			−0.18**
BIS total			0.35*
Step 3			
Gender	0.29	$F(1,82)=3.91$**	0.16
Age			−0.21**
BIS total			0.32*
SRC bias			0.19**
Step 4			
Gender	0.36	$F(2,80)=4.75$**	0.10
Age			−0.21**
BIS total			0.28**
SRC bias			0.21**
No-go errors			0.18**
AUC			0.26**
Step 5			
Gender	0.37	$F(2,78)=0.73$	0.11
Age			−0.21**
BIS total			0.28**
SRC bias			0.23**
No-go errors			0.20**
AUC			0.27**
SRC bias×No-go errors			0.09
SRC Bias×AUC			0.10

BIS Total Barrett Impulsivity Scales total scores, possible range of values 30 to 120 (minimum to maximum); SRC bias (ms)=Mean reaction time on avoid alcohol block minus mean reaction time on approach alcohol block, higher values indicate increased approach bias. Inhibitory control (No-Go errors)=Number of responses to No-Go cues, higher values indicate more inhibitory control failures. Delay Discounting (AUC), values range from 0 to 1, with higher values indicating a steeper rate of delay discounting. SRC bias×No-go errors and SRC bias×AUC=product of normalised variables

*p<0.001; **p<0.05

The finding that the two measures of behavioural impulsivity were distinct from each other is consistent with arguments made by de Wit and Richards (2004) and Olmstead (2006) inasmuch that we have found two separate components of behavioural impulsivity, deficient inhibitory control and impulsive decision making. The findings of the PCA also serve to replicate and expand upon those of Reynolds et al. (2006), who also found that a Go/No-task (and a stop-signal task) measured an aspect of impulsivity ('impulsive disinhibition') that was distinct from a second factor including delay discounting ('impulsive decision making'). In addition, we have shown that trait measures of impulsivity are also independent from behavioural impulsivity, with all three subscales of the BIS loading onto a third factor (trait impulsivity). Although these results are largely consistent with Reynolds et al. (2006), we did find that non-planning impulsivity also loaded onto the impulsive decision-making factor obtained from the PCA. One explanation for this is that participants have some insight into their impulsive decision making, which influences their response to questions which are concerned with a lack of planning for future events (possibly reflecting their decreased sensitivity to future rewards, de Wit et al. 2007). This relationship is in contrast to that observed by Swann et al. (2002) who found that although trait impulsivity (particularly the non-planning of subscale of the BIS) was related to impulsive decision making, a stronger relationship was found between trait impulsivity (again, specifically non-planning) and measures of disinhibition. This, along with the current findings, suggests that

more research is required to investigate how trait measures of impulsivity are associated with behavioural measures.

Both components of behavioural impulsivity–inhibitory control and delay discounting, as well as trait impulsivity, predicted unique variance in hazardous drinking. The finding that trait impulsivity is associated with alcohol use is consistent with a large body of previous research (e.g. McAdams and Donnellan 2009; Von Diemen et al. 2008). Associations between heavy drinking and both components of behavioural impulsivity have also been reported in previous studies (e.g. delay discounting, see Vuchinich and Simpson 1998 and Petry 2001; poor inhibitory control, see Colder and O'Connor 2002 and Goudriaan et al. 2006). However, there have been numerous failures to replicate these associations, particularly among non-dependent 'social' drinkers (e.g. Fernie et al. 2010; MacKillop et al. 2007). The recent meta-analysis by MacKillop et al. (2011), along with the current study, suggest that these findings may be at least partly attributable to the samples investigated. MacKillop et al. (2011) reported that although delay discounting is elevated in clinical samples, the effect size (referring to the relationship between delay discounting and drinking habits) is smaller in non-dependent populations. If non-clinical studies recruited young adult social drinkers, particularly university students, who had no reason to attempt to control their drinking behaviour, then one would expect an inconsistent pattern of associations between impulsivity and heavy drinking. To our knowledge, the size and consistency of the relationship between inhibitory control and drinking habits in non-dependent drinkers has not been the subject of a meta-analysis, so we would encourage researchers to conduct such a meta-analysis in order to address this issue. Another explanation for these inconsistencies in the literature is suggested by studies which demonstrate that both response inhibition (e.g. Gauggel et al. 2010; Jones et al. 2011) and delay discounting (e.g. Field et al. 2006; Giordano et al. 2002) are subject to state fluctuations in groups of substance abusers. Because trait impulsivity is relatively stable, whereas behavioural measures of impulsivity show within-subject fluctuations, one would expect a more consistent association between drinking habits and trait rather than behavioural measures of impulsivity. Nonetheless, the present results provide support for theoretical models which posit that heavy drinking should be associated with elevated behavioural impulsivity. One clinical implication is that novel interventions which aim to reduce impulsive decision making, or improve inhibitory control, may be suitable targets for the treatment of alcohol use disorders. Recent research suggests that training working memory reduces rates of delay discounting (see Bickel et al. 2011); this, along with the current research, suggests that treatments targeting reducing delay discounting may reduce hazardous drinking.

We also found that 'approach bias'—the tendency to categorise alcohol-related pictures more quickly when required to make a symbolic approach movement towards the pictures rather than a symbolic avoidance movement away from the pictures—was associated with increased alcohol consumption. This replicates earlier findings from our laboratory (Field et al. 2008; in press), and it is also consistent with similar findings obtained from related tasks, all of which measure the strength of associations between the concepts of 'alcohol' and 'approach' (Ostafin et al. 2008; Ostafin and Palfai 2006; Ostafin et al. 2003; Wiers et al. 2009). Recently, Wiers et al. (2010) demonstrated that behavioural training which aimed to reduce the strength of automatic approach tendencies elicited by alcohol-related cues led to reductions in alcohol consumption which suggests that strong automatic alcohol-approach associations may be a fundamental driving force behind alcohol use and a suitable target for treatment (see Wiers et al. 2011). Overall, these results suggest that alcohol-related cues possess incentive motivational properties among heavy drinkers, and as such, they provide support for incentive motivational theories of addiction and alcohol abuse (e.g. Robinson and Berridge 2001).

We also investigated a specific prediction made by Wiers et al. (2007), that the association between automatic approach tendencies elicited by alcohol cues and drinking behaviour would be moderated by measures of behavioural impulsivity. However, our results did not provide any support for this prediction. One explanation for this finding is that we assessed automatic alcohol cognitions using an SRC task rather than an Implicit Association Test as used by Houben and Wiers (2009) and Thush et al. (2008). Furthermore, Houben and Wiers (2009) assessed inhibitory control using a Stroop task, and Thush et al. (2008) investigated other aspects of executive cognitive function (specifically working memory). It is possible that these tasks may have differing sensitivity to impairments in executive functioning/automatic alcohol cognitions, which may account for our findings.

Overall, our results suggest that behavioural impulsivity consists of two distinct components with trait impulsivity being an additional component. Interestingly, we found that non-planning impulsivity loaded onto both the trait and decision-making components of impulsivity. Furthermore, we found that increased impulsivity and automatic approach responses elicited by alcohol-related cues are associated with individual differences in hazardous drinking. We did not see evidence of moderation of the association between hazardous drinking and approach responses by behavioural impulsivity as predicted by Wiers et al. (2007).

In summary, the present results support the proposed two-factor model of behavioural impulsivity, as well as demonstrating that trait measures are an additional component of

impulsivity. Furthermore, we replicated previous reports of associations between heavy drinking and elevated impulsivity (see de Wit 2009) and measures of the incentive motivational properties of alcohol-related cues (see Wiers et al. 2007).

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Ego Depletion Increases Ad-Lib Alcohol Consumption: Investigating Cognitive Mediators and Moderators

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When self-control resources are depleted ("ego depletion"), alcohol-seeking behavior becomes closely associated with automatic alcohol-related processing biases (e.g., Ostafin, Marlatt, & Greenwald, 2008). The current study aimed to replicate and extend these findings, and also to investigate whether the effects of ego depletion on drinking behavior would be mediated by temporary impairments in executive function or increases in impulsivity. Eighty heavy social drinkers (46 female) initially completed measures of automatic approach tendencies (stimulus response compatibility [SRC] task) and attentional bias (visual probe task) elicited by alcohol-related cues. Participants were then exposed to either an ego depletion manipulation or a control manipulation, before completing a bogus taste test in order to assess ad-lib alcohol consumption. In a subsequent testing session, we examined effects of the ego depletion manipulation (vs. control manipulation) on 3 aspects of executive function (inhibitory control, phonemic fluency, and delay discounting). Results indicated that the ego depletion manipulation increased ad-lib drinking, relative to the control manipulation. Automatic approach tendencies, but not attentional bias, predicted ad-lib drinking, although this effect was not moderated by ego depletion. Ego depletion had inconsistent effects on measures of executive function and impulsivity, and none of these measures mediated the effect of ego depletion on ad-lib drinking. However, the effect of ego depletion on ad-lib drinking was mediated by self-reported effort in suppressing emotion and thoughts during the manipulation. Implications for the effects of self-control strength on drinking behavior, and cognitive mediators of these effects, are discussed.

Keywords: alcohol, ego depletion, self-control, cognitive bias, executive function

Humans display a remarkable capacity to inhibit unhealthy urges. For example, despite temptation to eat chocolate and respond to feelings of hunger a dieter may choose not to eat in order to achieve a long-term goal of losing weight and improving health. Likewise, a newly abstinent smoker may have a strong desire for nicotine, but may be able to resist these urges to maintain the long-term goal of abstinence. In a variety of domains, humans are able to direct behavior toward future goals at the expense of satisfying immediate urges and desires.

Despite this capacity for self-regulation, there are numerous examples in which self-control lapses. Risky sexual behavior, overeating, drug and alcohol abuse, and aggression are all consequences of a failure to regulate behavior (Baumeister, Heatherton, & Tice, 1993; Quinn & Fromme, 2010; Stinson, Becker, & Sales, 2008). Indeed, a large number of social and health problems are likely to have failures in self-regulation at their core (Baumeister & Heatherton, 1996). Recent research into self-regulation has focused on the "strength" model (Baumeister, Bratslavsky, Muraven, & Tice, 1998). This model proposes that self-control resources are finite; if demands on self-control are minimal then an individual will have the resources available to successfully regulate their behavior. If demands on self-control are very high and/or demands have been maintained over a prolonged period, then self-control resources will be diminished and subsequent self-control will be impaired. This state of depleted self-control resources has been termed "ego depletion" (Baumeister et al., 1998; Muraven, Tice, & Baumeister, 1997). Recent investigations have shown that exerting self-control reduces the ability to regulate behavior in subsequent self-control tasks, even when the domains of the self-control tasks are different (for a recent review see Hagger, Wood, Stiff, & Chatzisarantis, 2009). With regard to alcohol consumption Muraven, Collins, and Nienhaus (2002) found that ego depletion resulted in increased beer consumption in a bogus taste test, and this effect was seen despite a financial incentive to limit consumption. In addition Muraven, Collins, Shiffman, and Paty (2005) investigated ego depletion in naturalistic settings, by assessing participants' daily self-control demands and alcohol consumption over a three-week period using ecological momentary assessment. The primary finding was that individuals were more likely to drink in excess of their self-imposed limits on days when they had experienced a higher than average level of self-control demands.

Despite this emerging evidence, the specific mechanisms that underlie effects of ego depletion are less clear. A recent meta-analysis found that ego depletion effects were associated with the degree of self-reported effort that was exerted during self-control

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tasks, as predicted by the strength model of self-control (Hagger, Wood, Stiff, & Chatzisarantis, 2010). This suggests that participants have some awareness of the amount of effort expended during depleting tasks, with increased effort resulting in reduced self-control resources. However, recent studies suggest that the *perception* of being in a state of depleted self-control, rather than actual resource depletion, accounts for the ego depletion effect. Indeed, Ackerman, Goldstein, Shapiro, and Bargh (2009) found that simulating self-control results in self-control depletion, while observing self-control in others actually increases self-control in the observer. Manipulating beliefs about the availability of self-control resources can also protect against ego depletion effects. For example, if individuals are primed to believe that self-control resources are available (Clarkson, Hirt, Jia, & Alexander, 2010), or that self-control is unlimited (Job, Dweck, & Walton, 2010), they are impervious to ego depletion manipulations. Furthermore, other factors, such as the unconscious priming of persistence (Alberts, Martijn, Greb, Merckelbach, De Vries, 2007), can mitigate against the effects of ego depletion.

Another possibility is that the depletion of self-control resources is mediated by fatigue in specific brain areas involved in behavioral regulation, most likely the prefrontal cortex (Gailliot & Baumeister, 2007). The taxing of self-control resources may temporarily impair aspects of executive cognitive function involved in behavioral regulation, such as inhibitory control, set-shifting, and working memory (Miyake et al., 2000). Related to this effect, demands on self-control resources may temporarily increase components of behavioral impulsivity, including disinhibition and myopic decision-making (see de Wit, 2009). There is some limited evidence to suggest that ego depletion does have a detrimental effect on executive function, for example, decreases in verbal and figure fluency (Schmeichel, Demaree, Robinson, & Pu, 2006), although the effects of ego depletion on behavioral measures of impulsivity have not yet been studied.

Dual-process models, such as Deutsch and Strack (2006) and Wiers et al. (2007), provide a theoretical framework for understanding the effects of ego depletion on alcohol consumption. These models argue that alcohol consumption is determined by the interplay between automatic alcohol-related processing biases (hereafter referred to as cognitive biases), on the one hand, and controlled processes, such as executive cognitive functioning, on the other. The strength model (Baumeister, Bratslavsky, Muraven, & Tice, 1998) states that after self-control resources have been depleted the ability to control subsequent behavior is diminished; ego depletion should therefore lead to a state in which behavior is strongly influenced by cognitive biases. Theoretically, cognitive biases develop because alcohol-related stimuli gain incentive-motivational properties after chronic heavy drinking (Robinson & Berridge, 2001), which results in increased attentional allocation and initiation of approach responses in response to alcohol-related cues (Franken, 2003). For example, alcohol cues capture and hold the attention of alcoholics and heavy social drinkers (Field, Christiansen, Cole, & Goudie, 2007; Stetter, Ackermann, Bizer, Straube, & Mann, 1995; Townshend & Duka, 2001; for a recent review, see Field & Cox, 2008). Heavy drinkers also direct automatic approach responses toward alcohol cues as revealed by the stimulus response compatibility (SRC) task, (Christiansen et al., 2011; Field, Caren, Fernie, & De Houwer, 2011; Field, Kiernan, Eastwood, & Child, 2008). Importantly, both alcohol attentional bias (Field & Eastwood, 2005; Schoenmakers et al., 2010) and automatic alcohol approach tendencies (Wiers, Eberl, Rinck, Becker, & Lindenmeyer, 2011; Wiers, Rinck, Kordts, Houben, & Strack, 2010) may have causal effects on subsequent drinking behavior. As predicted by dual-process models, the association between cognitive biases and drinking behavior is stronger in participants with poor executive cognitive functioning, specifically working memory (Thush et al., 2008) and inhibitory control (Houben & Wiers, 2009). If ego depletion affects behavior because it fatigues areas of the brain associated with behavioral control (as argued by Gailliot & Baumeister, 2007), then we would expect ego depletion to increase the association between cognitive biases and alcohol consumption. Indeed, recent studies have demonstrated that cognitive bias, assessed with an implicit association test, predicts ad-lib drinking after ego depletion but not after a control manipulation (Friese, Hofmann, & Wänke, 2008; Ostafin et al., 2008). This suggests that ego depletion facilitates the ability of automatic processes to control behavior.

The Present Study

Our primary aim was to extend the findings reported by Friese, Hofmann and Wänke (2008) and Ostafin et al., (2008), who demonstrated that the association between cognitive biases and alcohol consumption was moderated by ego depletion: Following ego depletion, the association between cognitive biases and ad-lib drinking became stronger. We investigated if these findings would generalize to other aspects of cognitive bias that have been demonstrated to have a causal influence on drinking behavior, that is automatic approach tendencies and attentional bias (Schoenmakers et al., 2010; Wiers et al., 2010; Wiers et al., 2011). Our secondary aim was to investigate the specific mediators of the ego depletion effect. Gailliot and Baumeister (2007) argue that ego depletion affects behavior because it produces temporary impairments in executive cognitive functioning. Previous investigations of the effects of ego depletion on ad-lib drinking did not investigate whether these effects were mediated by changes in executive cognitive functioning or impulsivity. Furthermore, other studies (e.g., Clarkson et al., 2010) suggest that ego depletion effects are driven by the perception of self-control, so this may also be partially responsible for changes in ad-lib drinking following ego depletion. In the present study, we investigated executive cognitive functioning, impulsivity, and perceived self-control resources as possible mediators of the effects of ego depletion on ad-lib drinking.

Our study consisted of two different experimental sessions. In the first session we assessed cognitive biases using measures of attentional bias (visual probe task) and automatic approach tendencies (SRC task). Following this, participants were exposed to either an ego depletion manipulation (involving both emotional and cognitive suppression, as recommended by Ostafin et al., 2008), or a nondepleting control manipulation. Finally, participants completed a bogus taste test in order to assess their ad-lib drinking. In a second session, participants were again exposed to the ego depletion manipulation or the control manipulation, before completing a short battery of executive function and impulsivity tests comprising measures of inhibitory control, delay discounting, and phonemic fluency. These tasks were administered in a separate session in the light of evidence suggesting that completion of

executive function tasks can itself be ego-depleting (Govorun & Payne, 2006; Webb & Sheeran, 2003); if so, this would have compromised the anticipated effect of the ego depletion manipulation on ad-lib drinking in the first session.

Hypotheses were as follows. First, we hypothesized that participants in the ego depletion condition would consume more beer than participants in the control condition, which would serve to replicate previous articles (e.g., Muraven et al., 2002). Our second hypothesis was that the associations between attentional bias for alcohol-related cues, automatic approach tendencies elicited by those cues, and ad-lib beer consumption would be moderated by the experimental manipulation, with the strongest associations seen in the ego depletion group. Our third hypothesis was that the ego depletion manipulation would impair all three measures of executive function/impulsivity in the second session, and individual differences in the extent of this impairment, together with effort expended during ego depletion would be associated with, and would mediate, the effects of ego depletion on ad-lib drinking.

Method

Participants

Eighty participants (46 female) aged between 18 and 40 years ($M = 22.08 \pm 4.53$) were recruited via word of mouth and intranet advertising from the University of Liverpool. Inclusion criteria were fluency in English, and normal or corrected-to-normal vision. Participants were invited to take part if they self-reported consuming at least 15 units of alcohol (Females) or 22 units (Males) each week, which is in excess of the U.K. government guidelines for safe drinking (Edwards, 1996). Furthermore, it was made clear in advertisements and the participant information sheet that all participants must regularly drink beer, as tasting beers was a part of the procedure. Exclusion criteria included current or past self-reported alcohol use disorder, blood injury phobia, current or recent illness which may increase sensitivity to alcohol (e.g., colds and flu), and taking medication that is contraindicated for alcohol use (e.g., antidepressants, anxiolytics). Additional exclusion criteria for female participants included current breastfeeding or pregnancy; the latter was confirmed with a pregnancy test at the beginning of the first session. All participants provided informed consent before taking part in the study, which was approved by the University of Liverpool Research Ethics Committee. Participants received either course credits or £20 as compensation for their travel expenses and time.

Procedure

Testing sessions took place between 12 p.m. and 6 p.m. in laboratories at the University of Liverpool. Each participant attended the laboratory for two separate sessions, with a gap of at least two days between sessions. The first session lasted approximately 75 minutes; the second session lasted approximately 25 minutes. Diagrams illustrating key features of both experimental sessions are shown in Figure 1. During the first session, participants provided informed consent before being breathalyzed using a Lion Alcometer 500 (Lion laboratories, Barry U.K.) to confirm a baseline breath alcohol content (BAC) of zero (no participants provided a positive BAC before either session). They then completed a short questionnaire battery (Time Line Follow Back [TLFB], Alcohol Use Disorders Identification Test [AUDIT], Temptation and Restraint Inventory [TRI], Barratt Impulsivity Scales [BIS-11], and Desires for Alcohol Questionnaire–brief version [DAQ]). Participants were then seated 1m away from the computer monitor before completing the SRC and visual probe tasks, in a counterbalanced order.

Participants were randomly assigned to either the ego depletion or the control condition. Both groups of participants were told that they were to watch a film clip. Participants in the ego depletion condition were informed that they should try not to respond to the clip in any way (no facial expressions or turning away), and that they should suppress any thoughts, feelings or emotions that they may experience while watching the clip. Participants in the control condition were given no instructions before watching the clip. The experimenter remained in the laboratory throughout the experiment in order to observe participants' emotional expressions and to remind them of the task instructions if necessary. Once the clip finished, participants were asked to complete a manipulation check questionnaire. Participants were asked to rate perceived effort put into suppression, perceived difficulty of suppression, emotionality of the task, and feelings of being emotionally drained and tiredness on 25-point scales. Participants then completed a cognitive suppression task; participants in the ego depletion group were told to write down any thoughts that came to mind over the next five minutes but not to think about anything that they had just seen in the clip. Participants in the control condition were told to write down any thoughts that they had over the next five minutes, but no reference was made to the film clip. Upon completion of the cognitive suppression task participants completed a second manipulation check as well as the Brief Mood Introspection Scale (BMIS) and DAQ.

Participants were then informed that they would be tasting and rating different beers for 30 minutes, before they would be asked to complete an additional reaction time (RT) task. Participants did not actually complete this final task but were informed that it was a "short but difficult RT task on which good performance would be rewarded with a £10 bonus payment, although performance on this task is very likely to be negatively affected by alcohol consumption." This was to encourage participants to limit their drinking and similar instructions were utilized by Muraven et al. (2002) and Ostafin et al. (2008). The experimenter left the participant for 30 minutes to complete the taste test. The experimenter then returned, breathalyzed the participant, and informed them that there would be no RT test. Participants were then discharged and reminded to return for the second session.

In the second session, participants were again breathalyzed before completing the experimental manipulation (ego depletion or control) in the same manner as they did in the first session (including all manipulation checks except for the DAQ). All participants viewed a different clip from that seen in the first session. Upon completion of the manipulation, participants completed the Go-No/Go, Controlled Oral Word Association Test (COWAT), and delay discounting tasks in a counterbalanced order. An informal debrief revealed that participants were unaware of the links between the cognitive tasks, experimental manipulations, and beer consumption in the taste test. At the end of the study, participants were fully debriefed on the aims and hypotheses of the study before being released.

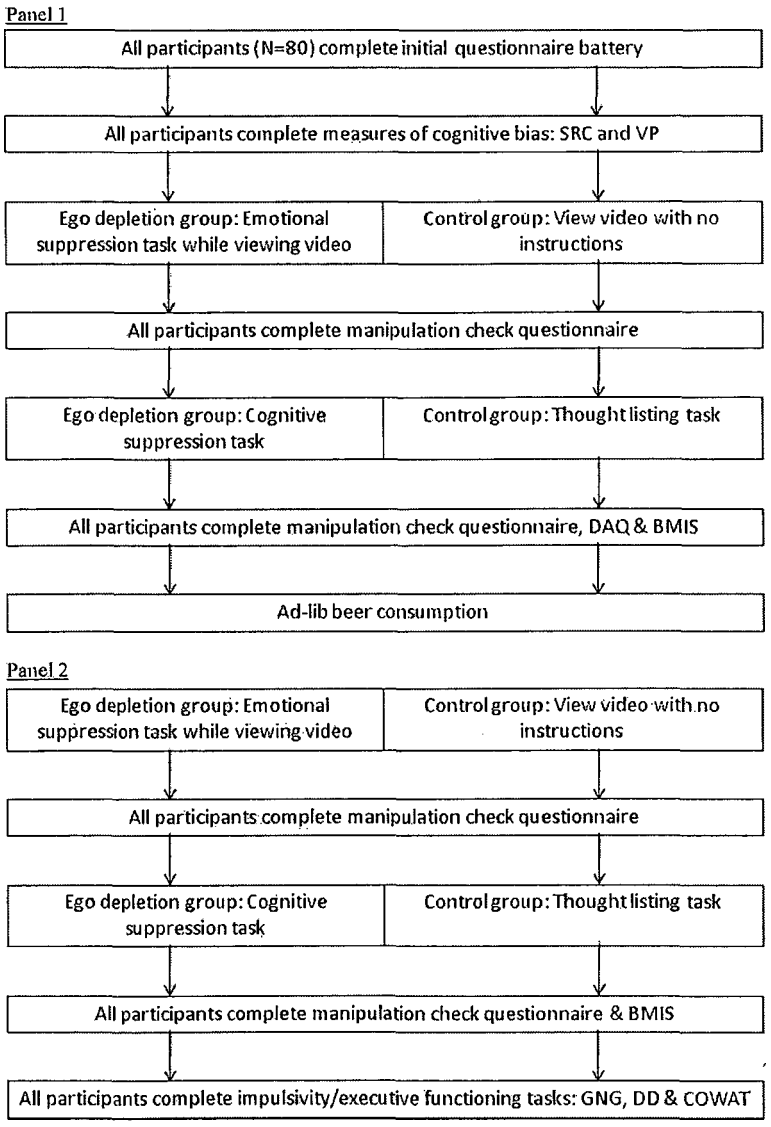

Figure 1. Schematic overviews of experimental procedures in Session 1 (panel 1) and Session 2 (panel 2).

Materials

Questionnaires.

Time Line Follow Back (Sobell & Sobell, 1990). The TLFB is a retrospective diary used to assess alcohol consumption. Participants estimated the number of alcohol units consumed over the preceding 14 days.

The Alcohol Use Disorders Identification Test (Saunders, Aasland, Babor, De la Fuente, & Grant, 1993). The AUDIT consists of 10 fixed response questions regarding alcohol consumption and consequences of drinking. Scores on the AUDIT range between 0 and 40 with scores of 8 or above indicating hazardous or harmful alcohol use.

Temptation and Restraint Inventory (Collins & Lapp, 1992). This scale measures preoccupation with and attempts to limit drinking, and yields scores on two subscales termed "Cognitive and Emotional Preoccupation" (CEP) and "Cognitive and Behavioral Control" (CBC). The TRI consists of 15 questions scored from 1 to 9 on a Likert scale.

Barratt Impulsivity Scales (Patton, Stanford, & Barratt, 1995). This scale is a multidimensional measure of impulsivity with three subscales—Attentional, Motor, and Nonplanning impulsiveness. The BIS-11 consists of 30 fixed response questions scored from 0–4 with high scores indicative of increased impulsivity.

Desires for Alcohol Questionnaire–brief version (Love, James, & Willner, 1998). The DAQ is a measure of alcohol craving that contains four different subscales—Positive and Negative Reinforcement, Strong Desires and Intentions, Mild Desires and Intentions, and Perceived Control over drinking. The DAQ consists of 14 statements that are responded to on seven point Likert scales ranging from "strongly disagree" (1) to "strongly agree" (7).

Brief Mood Introspection Scale (Mayer & Gaschke, 1988). The BMIS is a self-report measure of mood and arousal. The BMIS consists of 16 adjectives which are responded to on 4-point Likert scale ranging from "definitely do not feel" (1) to "definitely feel" (4). Four underlying mood factors are derived: Pleasant-Unpleasant, Arousal-Calm, Positive-Tired, and Negative-Relaxed. Factor scores are derived by adding or subtracting scores from relevant items. For example, the Pleasant-Unpleasant factor is computed by subtracting values for unpleasant adjectives (e.g., Grouchy, Sad) from scores derived from pleasant adjectives (e.g., Content, Happy).

Video stimuli for ego depletion manipulation. We selected an 18-minute section from the film Audition (2002) to use for the ego depletion manipulation. The entire 18-minute section contained numerous flashbacks which were cut, so the final edited clip was 10 minutes long. The clip contained graphic depictions of torture (e.g., a character has his feet amputated with wire). We used this clip as disgust is more easily and ethically manipulated than emotions such as happiness or sadness (Gross & Levenson, 1993). As the experiment consisted of two sessions, we split the scene into two 5 minute segments, the presentation of which was counterbalanced across sessions.

Pictorial stimuli Field & Eastwood, (2005) and Field et al. (2008). The SRC and visual probe tasks used a picture set containing 14 alcohol-related pictures and 14 (matched) alcohol-unrelated pictures. Alcohol pictures consisted of alcohol-related scenes (such as a bottle and a glass of wine on a table), the alcohol-unrelated pictures were matched to the alcohol pictures on perceptual characteristics but did not contain any alcohol-related cues (e.g., a bottle and a glass of water on a table). All pictures were 100-mm high × 125-mm wide.

Bogus taste test (based on Weafer & Fillmore, 2008). Participants were provided with three numbered glasses each containing 255 ml of beer. They were instructed to taste the beers and rate each one according to nine different dimensions, for example "how sweet tasting is the drink?" by marking 15-point Likert scales with anchors "not at all" and "very much." Participants were also asked to rank the beers in order of preference and order of alcohol content, and attempt to identify the beer brands. Participants were informed that they could drink as much of each beer as they wished in order to make accurate assessments, and they were given 30 minutes to do so. The beers provided were Hoegaarden wheat beer (4.9% Alcohol by volume, ABV), Carlsberg lager (4.2% ABV), and Golden Champion summers ale (5% ABV). We selected beers with distinctly different tastes to encourage participants to think more about their responses. Responses to the taste ratings, perceived alcohol content and beer preference questions were not analyzed. The dependent variable was the total volume of beer consumed.

Cognitive Tasks

Cognitive bias measures (Session 1 only).

Automatic approach tendencies. The SRC task (Field et al., 2008) was used to assess automatic approach tendencies elicited by alcohol-related cues. Each trial of the task commenced with the presentation of either an alcohol-related picture or an alcohol-unrelated (control) picture in the center of the screen along with a small manikin above or below the picture. Participants were instructed to move the manikin either toward or away from the picture by pressing up or down on a two button response box. If participants made the appropriate response, the manikin moved toward or away from the picture. If they made an inappropriate response (e.g., pushing the "up" button when a "down" response was required), a large red cross was presented in the center of the screen for 1000 ms. There was an intertrial interval of 500 ms.

There were 128 trials of the task in total, split into two blocks of 64 trials. In the "approach alcohol" block, participants were instructed to move the manikin toward alcohol-related pictures, and away from pictures that were unrelated to alcohol. These instructions were reversed in the "avoid alcohol" block. Each block began with eight practice trials in which four alcohol-related and four alcohol-unrelated pictures were presented. After the practice trials, the instructions were then reiterated before participants completed 56 experimental trials. During these trials, the 14 alcohol-related and 14 alcohol-unrelated pictures were each presented twice, once with the manikin above the picture and once with the manikin below the picture. Trials were presented in random order. The order of completion of "approach alcohol" and "avoid alcohol" blocks was counterbalanced across participants. RT (the time taken to initiate movement of the manikin) was measured on each trial. The dependent variable from the task (SRC bias) is computed by subtracting mean RT during the "approach alcohol block" from mean RT during the "avoid alcohol" block. Higher values of SRC bias indicate an automatic approach response elicited by alcohol-related cues.

Attentional bias. The visual probe task (see Schoenmakers, Wiers, & Field, 2008) was used to assess biases in selective attention for alcohol-related pictorial cues. Each trial of the task commenced with a white fixation cross presented in the center of the screen for 500 ms. Immediately after this, a pair of pictures was presented for 2000 ms, one picture on the left of the screen and the other on the right of the screen, 60 mm apart. Immediately after picture offset a probe (a white arrow, pointing up or down) appeared in one of the picture locations. Participants had to respond to the orientation of the probe by pressing up or down on a two button response box. There was an intertrial interval of 500 ms.

The task consisted of 68 trials in total. Participants first completed 10 practice trials in which neutral picture pairs were presented, following which instructions were reiterated before they completed the main block of trials. The main task consisted of 2 buffer trials (of neutral picture pairs) followed by 56 critical trials. Each of the 14 picture pairs appeared four times each, with the alcohol pictures on the left twice and on the right twice; visual probes replaced alcohol and control pictures with equal frequency. Trials were presented in random order. RT (the time taken to identify the probe) was measured on each trial, and horizontal eye movements were recorded during the 2000 ms of stimulus presentation at a sampling rate of 120 Hz (using the Eyetrace 300x system; Applied Science Laboratories, Bedford, MA). The task yields two different measures of attentional bias, one based on RTs and the other based on eye movements. The RT measure of attentional bias is calculated by subtracting mean RTs to congruent probes (those that appeared in the same location as the alcohol pictures) from mean RTs to incongruent probes (those that appeared in the same location as the control pictures), such that higher values reflect increased attentional bias. The eye movement

measure of attentional bias is calculated by subtracting total gaze dwell time on control pictures from total gaze dwell time on alcohol pictures, such that higher values reflect increased attentional bias.

Executive cognitive function/impulsivity measures (Session 2 only).

Inhibitory control. The "passive avoidance" version of the Go/No-Go task (Newman & Kosson, 1986) requires participants to learn through trial and error which numerical stimuli are "correct" (go cues) and which are "incorrect" (no-go cues). Participants were instructed to withhold responses to the incorrect stimuli (no-go cues), but respond quickly to correct stimuli (go cues) by pressing the spacebar on the keyboard. On each trial of the task, one of eight two-digit numbers was presented. Four numbers (34, 42, 51, 93) were go cues and four (18, 29, 63, 85) were no-go cues. Participants initially completed 8 practice trials, in which each number was presented once, followed by three blocks of experimental trials. Each experimental block consisted of 24 trials in which each of the eight numbers was presented three times each. After completion of each block, participants received feedback on the percentage of correct responses to both go and no-go cues.

Each trial began with the presentation of a white fixation cross in the center of the screen for 1000 ms, before a go or no-go cue was presented. Cues remained on the screen until a response or a 3-s timeout period had elapsed. Correct responses to go cues resulted in the text "Correct!" appearing on the screen in green font for 300 ms. Commission errors (inappropriate responses to no-go cues) resulted in the text "Wrong!" appearing on the screen in red font for 300 ms. If no response was made no feedback was given. The primary dependent measure from this task was the number of commission errors, with a high rate of these indicative of impaired inhibitory control.

Delay discounting (DD; Du, Green, & Myerson, 2002). A computerized DD task (programmed in Visual Basic 6.0) was used to assess impulsive decision-making in response to monetary rewards. The DD methodology was identical to the one used by Fernie, Cole, Goudie, & Field, (2010). Participants were presented with the hypothetical choice of receiving £100 at a future date or receiving a smaller amount immediately. The size of the immediate reward was adjusted by either adding 50% of the last adjustment (if the delayed reward was selected) or subtracting 50% of the last adjustment (if the immediate reward was selected). Participants made 6 choices for each delay period. Monetary choices were made for delays of one day, one week, two weeks, one month, and six months. Indifference points for each of the 5 delays were analyzed by computing area under the curve (AUC) values (Myerson, Green, & Warusawitharana, 2001). Lower values of AUC indicate steeper delay discounting, or increased impulsive decision-making.

Phonemic fluency. The COWAT (Benton, 1968) was used to assess phonemic fluency as a measure of executive functioning. In this task, participants were given a letter and instructed that they had one minute to verbally state as many words beginning with that letter as possible (excluding proper nouns and identical words with a different suffix). Participants produced words for the letters *F*, *A*, and *S*. A voice recorder was used to record responses for future analysis. The dependent measure from the COWAT was the total number of switches between word clusters (with a greater number of switches reflecting increased executive cognitive functioning). Word clusters were defined as consecutive words which begin with the same two letters, which differed only by a vowel, or were homonyms or rhyming words (Troyer, Moscovitch, & Winocur, 1997). This method for assessing switches was found to best reflect frontal functioning in phonemic fluency as well as having high test-retest reliability (Ross et al., 2007).

Data Reduction and Analysis

Before analysis of RTs from the visual probe and SRC tasks, outliers were removed according to criteria used in previous reports (e.g., see Field et al., 2008)—RTs less than 200 ms, greater than 2000 ms, and then those RTs that were more than 3 standard deviations above the individual mean, were discarded. RTs from error trials were also discarded. A simple power analysis indicates that a sample size of approximately 80 has sufficient power to detect a medium effect size (power = 0.8, $f^2 = 0.14$, $\alpha = .05$). These figures are based on previous research using the same experimental design (Ostafin et al., 2008). We assessed group differences in demographics, alcohol use indices, self-report impulsivity, and self-report craving (the latter was assessed before and after the manipulation) to ensure both groups were matched upon these variables as they are likely to influence beer consumption in the taste test.

We conducted a series of mediation analyses to investigate whether the anticipated ego depletion effect on beer consumption was a result of impaired executive cognitive function. In addition, we also assessed whether ego depletion effects were mediated by effort put into the depletion tasks. These analyses were conducted on variables that were shown to be significantly affected by the ego depletion manipulation. There has been considerable debate as to which methodologies best evaluate mediation in the social sciences. MacKinnon, Lockwood, Hoffman, West, & Sheets, (2002) investigated 14 methods for mediation analysis and found the joint significance test to be the best method due to its statistical power (in sample sizes similar to the current sample) and because it does not suffer from inflated Type I error rates. This method involves testing the statistical significance of the relationship between the IV (ego depletion) and the proposed mediator (α path), and the relationship between the proposed mediator and the DV (ad-lib beer consumption; β path). If both these relationships are statistically significant there is evidence of mediation. In addition, the PRODCLIN program (see MacKinnon, Fritz, Williams, & Lockwood, 2007) was used to generate 95% confidence intervals by using the α and β coefficients and their standard errors ($\sigma\alpha$, $\sigma\beta$) to compute the asymmetric confidence interval for the mediated effect.

Results

Group Characteristics

Table 1 shows summary statistics for the questionnaires that participants completed at the beginning of the session (weekly alcohol consumption, AUDIT, BIS-11 total score, and TRI). A multivariate analysis of variance (MANOVA) revealed no significant main effect of experimental condition, $F(6, 73) = 0.77$, $p > .10$. Therefore, ego depletion and control groups did not differ on any of these measures. There was also no significant difference in

Table 1
Group Characteristics (Mean ± SD)

Characterstics	Control (n = 40)	Ego depletion (n = 40)
Gender (M:F)	(16:24)	(18:22)
Age (years)	22.73 (5.56)	21.42 (3.62)
Past 14 day alcohol consumption (UK units)	55.35 (25.93)	60.65 (29.98)
AUDIT	13.50 (5.05)	13.33 (5.06)
TRI CEP	24.38 (11.24)	23.38 (10.23)
TRI CBC	18.38 (8.34)	16.63 (8.20)
BIS-11 Total scores	69.45 (9.83)	73.82 (12.41)

Note. Past 14 day alcohol consumption in UK units, 1 unit = 8g alcohol; AUDIT = Alcohol use disorders identification test, possible range of scores is from 0 (minimum) to 40 (maximum). TRI = Temptation and Restraint Inventory, range of TRI subscale scores (minimum to maximum); Cognitive and Emotional Preoccupation (CEP) 9 to 81, Cognitive and Behavioural Control (CBC) 6 to 54. BIS-11 = Barratt Impulsivity Scale, possible range of scores is from 30 (minimum) to 120 (maximum)

the proportion of males and females in the experimental conditions, $\chi^2(1, N = 80) = .21, p > .10$.

Effects of Ego Depletion on Subjective Alcohol Craving (Session 1 only)

A 2 × 2 mixed-model analysis of variance (ANOVA) was used to investigate the effect of experimental condition (ego depletion, control) and time (premanipulation vs. postmanipulation) on mean DAQ scores. There was a significant main effect of time, with DAQ scores higher at postmanipulation (2.85 ± 0.94) compared with premanipulation, 2.65 ± 0.86; $F(1, 78) = 10.37, p < .005$. The main effect of the experimental condition, $F(1, 78) = 2.45$, $p > .10$, and the Experimental condition × Time interaction, were not statistically significant, $F(1, 78) = 2.15, p > .10$. Therefore, the ego depletion manipulation had no significant effect on self-reported alcohol craving. Data are not shown, but are available on request.

Effects of Ego Depletion on Manipulation Check Questions and Mood (Sessions 1 and 2)

In order to assess the success of the ego depletion manipulation and whether the effects of the manipulation were consistent across Sessions 1 and 2, we analyzed the manipulation check and BMIS data using a series of 2 × 2 mixed-model ANOVAs, with a within subjects factor of session (Session 1, Session 2) and a between subjects factor of experimental condition (ego depletion, control). Participants in the ego depletion condition reported significantly more effort put into both emotional suppression, $F(1, 78) = 15.99$, $p < .001$, and cognitive suppression, $F(1, 78) = 12.28, p < .001$. Furthermore, participants in the ego depletion condition also found it significantly more difficult suppressing emotions, $F(1, 78) = 7.66, p < .001$, although there was no significant difference between conditions in self-reported difficulty in suppressing thoughts, $F(1, 78) = 1.44, p > .10$. There was also a significant Condition × Session interaction for both effort put into, $F(1, 78) = 4.10, p < .05$, and difficulty of, $F(1, 78) = 4.76, p < .05$, emotional suppression. This interaction was due to decreases in effort and difficulty ratings between Session 1 and 2 in the ego depletion condition only. There were no significant main effects of condition or session, and no significant Condition × Session interactions, for the BMIS subscales, self-reports of emotionally drained and tiredness, and the perceived emotionality of the film clip. Summary data for variables that were affected by the ego depletion manipulation are shown in Table 2.

Relationship Between Cognitive Bias Measures and Ad-Lib Drinking in Ego Depletion and Control Conditions

The primary hypothesis of this study was that the association between cognitive biases and ad-lib alcohol consumption would be moderated by the ego depletion manipulation. To test this we conducted three separate regression analyses in which we assessed the relationships between automatic approach tendencies, the RT index of attentional bias, and gaze dwell time index of attentional bias, with ad-lib beer consumption in the taste test. In the first step of the regressions we added the cognitive bias measure and experimental group (ego depletion or control). In the second step we entered the interaction between the normalized cognitive bias variable and experimental condition (with ego depletion coded as 2 and control coded as 1). First, we performed a MANOVA to test for pre-existing group differences in cognitive bias, this test revealed no significant differences in cognitive bias between the two groups, $F(3, 74) = 1.92, p > .10$.

In the first regression model, with automatic approach tendencies as the cognitive bias measure, the overall regression model was significant, as the predictors accounted for approximately 16% of the variance in beer consumption, $R^2 = .19$, R^2 *adjusted* = .16, $F(3, 74) = 5.92, p < .001$. Participants with stronger automatic

Table 2
Group and Session Comparisons of Manipulation Check Variables Significantly Affected by the Ego Depletion Manipulation (Mean ± SD)

	Session 1		Session 2	
Variable	Ego depletion	Control	Ego depletion	Control
Emotional suppression				
Effort suppressing emotions	15.68 (5.67)	9.63 (5.99)	13.65 (6.85)	9.92 (5.58)
Difficulty suppressing emotions	13.80 (6.09)	8.92 (6.09)	12.57 (.18)	10.22 (6.12)
Cognitive suppression				
Effort suppressing thoughts	12.32 (7.12)	7.65 (5.35)	11.67 (7.07)	7.27 (5.18)

approach tendencies elicited by alcohol-related cues consumed more beer during the taste test ($\beta = .31, p < .01$). Experimental condition was also a significant predictor ($\beta = .27, p < .01$), with the ego depletion group consuming more beer (429.25 ml ± 212.77) than the control group (285.03 ml ± 218.27). However, the interaction between automatic approach tendencies and experimental condition was not statistically significant ($\beta = .06, p > .10$), which indicates that the association between automatic approach tendencies and beer consumption was not moderated by the ego depletion manipulation.

Regarding attentional bias, although the regression models for both the gaze dwell time index, $R^2 = .11$, R^2 *adjusted* = .07, $F(3, 76) = 3.09, p < .05$, and the RT index, $R^2 = .10$, R^2 *adjusted* = .07, $F(3, 76) = 2.94, p < .05$, of attentional bias were significant, this was carried by the effect of ego depletion on beer consumption. Neither the gaze dwell time index ($\beta = .07, p > .10$) or the RT index ($\beta = .03, p > .10$) were significant predictors of beer consumed. Furthermore, the interactions between the gaze dwell time index of attentional bias and condition ($\beta = .03, p > .10$), and the RT index of attentional bias and condition ($\beta = .02, p > .10$), were not significant.

In order to explore gender differences, we conducted additional regression analyses by adding gender in the first step, cognitive bias and experimental condition in the second step, and the interaction between the normalized cognitive bias variable and experimental condition in the third step. Gender was a significant predictor of beer consumption, with males drinking more beer than females, ($\beta = .44$, $p < .01$). Importantly, all other results were unaffected.

Effects of Ego Depletion on Executive Function/Impulsivity

A MANOVA revealed that there was a trend toward a main effect of condition on the measures of executive cognitive function/impulsivity, $F(3, 76) = 2.39, p < .10$. Ego depletion (0.22 ± 0.03) and control groups (0.20 ± 0.03) did not differ in discounting rates (AUC values) obtained from the delay discounting task, $F(1, 78) = 0.19, p > .10$. Regarding COWAT switches, there was no significant difference between ego depletion compared with the control group, 28.32 ± 8.06 vs. 25.43 ± 8.44; $F(1, 78) = 2.47$, $p > .10$. However, there was a trend toward increased commission errors (impaired inhibitory control) on the Go/No-Go task in the ego depletion group compared with participants in the control group, 9.10 ± 5.47 vs. 6.85 ± 5.74; $F(1, 78) = 3.22, p < .10$.

Investigating Mediators of the Effect on Ego Depletion on Beer Consumption

As the joint significant test requires the proposed mediator to be affected by the IV, we only conducted the mediation analysis using proposed mediators that were affected by the ego depletion manipulation (i.e., commission errors on the Go/No-go task and perceived effort put into emotional and cognitive suppression tasks).

There was a trend toward an effect of ego depletion on commission errors (α path; $r^2 = .04, \beta = .20, p < .10$), although commission errors were not associated with beer consumption (β path; $r^2 = .01, \beta = .10, p > .10$). This analysis indicates that impairments in inhibitory control did not mediate the effect of ego depletion on alcohol consumption.

To investigate a possible mediating role for perceived effort put into the ego depletion tasks, we combined scores from the two manipulation check variables assessing how much effort participants put into the emotional and cognitive suppression tasks. The effect of the ego depletion manipulation on effort put into suppression was significant (α path; $r^2 = .25, \beta = .50, p < .001$) as was the association between effort put into suppression and beer consumption (β path; $r^2 = .05, \beta = .22, p < .05$). PRODCLIN revealed that the upper and lower 95% confidence limits (CLs) for the indirect effect of ego depletion on beer consumed were more than 1; therefore, indicating statistically significant mediation of the ego depletion effect on beer consumed by perceived effort put into the tasks 95% CL[1.82, 17.03].

Discussion

The current study investigated the effects of ego depletion on ad-lib alcohol consumption, and the role of cognitive processes in these effects. In an initial session, participants completed two tasks that assessed cognitive biases for alcohol cues (automatic approach tendencies and attentional bias) followed by either an ego depletion manipulation or a control manipulation, before they completed a taste test to measure their ad-lib alcohol consumption. In a subsequent testing session participants underwent ego depletion or the control manipulation and then completed tasks assessing executive cognitive functioning and impulsivity. We hypothesized that the ego depletion manipulation would increase ad-lib drinking (beer consumption in the taste test), and we also predicted that the associations between cognitive biases and ad-lib drinking would be moderated by the experimental manipulation, with stronger associations in the ego depletion group compared with the control group. We also predicted that the ego depletion manipulation would influence executive function and impulsivity, and that these changes, together with self-reported effort expended during ego depletion, would mediate the effects of ego depletion on ad-lib drinking. Our results provided partial support for these hypotheses. As predicted, the ego depletion manipulation resulted in increased beer consumption. Individual differences in automatic approach tendencies elicited by alcohol-related cues (as assessed with the SRC task) were associated with ad-lib drinking, although this association was not moderated by the ego depletion manipulation as we had predicted. Neither measure of attentional bias was associated with beer consumption, regardless of ego depletion. Contrary to expectations, the effects of ego depletion on measures of executive function and impulsivity were inconsistent across different measures, and none of these measures appeared to mediate the effect of ego depletion on ad-lib drinking. Finally, the effect of ego depletion on ad-lib drinking was mediated by self-reported effort expended during ego depletion.

The main effect of ego depletion on beer consumption supports the strength model of self-control and directly replicates previous studies such as Muraven et al. (2002): Relative to a control (nondepleting) manipulation, beer consumption was increased following ego depletion, even though participants were given an incentive to refrain from drinking. Therefore, this finding adds to a growing body of evidence which suggests that ego depletion leads to a reduction in self-control resources which can be detected in a variety of domains, including increased alcohol consumption, smoking, overeating, emotion regulation, and expended physical

and mental effort (Hofmann, Rauch, & Gawronski, 2007; Muraven et al., 1997; Muraven et al., 2002; Shmueli & Prochaska, 2009; Stucke & Baumeister, 2006).

We tested predictions derived from dual-process models (e.g., Deutsch & Strack, 2006; Wiers et al., 2007), namely that ad-lib drinking would be more strongly associated with cognitive biases for alcohol cues after ego depletion, due to the reduced availability of self-control resources. Our results did not support these predictions: We found no associations between attentional bias and ad-lib drinking, and although we did see an association between automatic approach tendencies and ad-lib drinking, this association was not moderated by the ego depletion manipulation. Although previous studies have found stronger associations between implicit alcohol cognitions and ad-lib drinking after ego depletion when alcohol versions of the implicit association test (IAT) were used (Friese et al., 2008; Ostafin et al., 2008) we failed to extend these findings using different measures of cognitive biases for alcohol cues. In the present study, we measured automatic approach tendencies elicited by alcohol-related cues (with the SRC task) and attentional bias for alcohol cues (with the visual probe task). Given recent experimental findings, which suggest that both attentional bias (Field & Eastwood, 2005; Schoenmakers et al., 2010) and automatic approach tendencies (Wiers et al., 2011; Wiers et al., 2010) appear to exert a causal influence on subsequent drinking behavior, one implication of our results is that the impact of these cognitive biases on drinking behavior does not seem to be further exacerbated when self-control resources are depleted. This finding is problematic for dual-process models (e.g., Wiers et al., 2007; Deutsch & Strack, 2006), which posit that individual differences in self-control resources should increase the impact of automatic alcohol cognitions on subsequent drinking behavior.

We also hypothesized that the effects of ego depletion on ad-lib drinking would be mediated by executive function and impulsivity, as was predicted by Gailliot and Baumeister, (2007). Contrary to hypotheses, ego depletion had inconsistent effects on these measures: inhibitory control was mildly impaired, but verbal fluency and delay discounting were unaffected. Furthermore, performance on these tasks after ego depletion did not mediate the effects on subsequent drinking behavior. One explanation for these findings is that we assessed effects of ego depletion on ad-lib drinking in an initial testing session, but effects of ego depletion on executive function were assessed in a subsequent testing session. We opted for this experimental design, rather than assessing effects of ego depletion on executive function, impulsivity, and ad-lib drinking in a single session, as we were concerned that completion of the executive function and impulsivity measures would serve to deplete self-control resources (e.g., see Govorun & Payne, 2006), which may have obscured effects of the ego depletion manipulation on ad-lib drinking, and prevented us from testing our primary hypothesis. Unfortunately, analysis of the manipulation check data revealed that the ego depletion manipulation appeared to be less effective in the second session compared with the first, in that participants' self-reported effort, and perceived difficulty in suppressing emotions, was lower during the second session compared with the first. This is consistent with results from a meta-analysis, which found that ego depletion effects tend to decline in magnitude after training on depleting tasks (Hagger et al., 2010). Therefore, in order to conduct an appropriate test of the hypothesis that behavioral effects of ego depletion are partially mediated by changes in executive function and impulsivity, future studies should assess these variables in a single session, in order to prevent practice effects from weakening the effectiveness of the manipulation. It is also possible that participants became fatigued due to the large number of tasks that they completed, so future studies of this type may wish to include only one or two cognitive tasks in order to assess mediation effects.

Our analyses did reveal that the ego depletion effect on ad-lib drinking was mediated by participants' perception of how depleted they were (i.e., self reported effort put into the emotional and cognitive suppression tasks). This finding is consistent with the strength model of self-control (Baumeister et al., 1998). The implication is that participants who felt that they put more effort into controlling emotional responses to the film clip and suppressing thoughts related to the clip, subsequently consumed more beer because they perceived their self-control resources to be depleted. Other studies have highlighted the importance of perceived self-control resources as important mediators of ego depletion effects (Clarkson et al., 2010; Job et al., 2010), and demonstrated that automatic processes, such as priming of persistence, can overcome ego depletion effects (Alberts et al., 2007). When combined with the present results, these studies suggest that the *perception* of having depleted self-control resources, but not actual fatigue in behavioral control processes, is the mechanism that determines the consequences of ego depletion, including the effects on drinking behavior. The clinical implication is that interventions, which aim to challenge perceptions of depletion, could be a simple and efficacious method for reducing heavy drinking and other unhealthy behaviors.

Previous studies that used the SRC task (e.g., Christiansen et al., 2011; Field et al., 2008; Field et al., 2011) demonstrated that heavy drinkers, but not light drinkers, were faster to categorize alcohol-related pictures when required to do so by making a symbolic approach response rather than a symbolic avoidance response. The current results go one step further, as they demonstrate that individual differences in performance on the SRC task are associated with ad-lib drinking when assessed soon after completing the task. As such, these results are consistent with recent findings that demonstrate that strong automatic approach tendencies have a causal influence on drinking behavior (Wiers et al., 2011; Wiers et al., 2010), although our own data do not enable us to infer a causal influence. With regard to attentional bias, we did not observe any association between attentional bias and ad-lib drinking, which casts doubt on the role of attentional bias as a cognitive bias which drives drinking behavior (see Field et al., 2007; Field & Eastwood, 2005; Schoenmakers et al., 2010). One possible explanation is that the association between attentional bias and the motivation to drink is relatively weak, and very large sample sizes may be required to detect an association between attentional bias and ad-lib drinking in the laboratory (Field, Munafo, & Franken, 2009).

In summary, our results offer support for the strength model of self-control in relation to heavy drinking, as participants consumed more beer, despite a financial incentive to refrain from heavy drinking, after an ego depletion manipulation. These effects were mediated by the degree of self-reported effort expended during ego depletion, rather than by any change in executive function or impulsivity. This has implications for our understanding of the psychological mechanisms that underpin ego depletion effects in general, and on drinking behavior more specifically. Finally, results were not generally consistent with dual-process models of addictive behavior: although

individual differences in automatic approach responses elicited by alcohol cues were associated with ad-lib drinking, these associations were not more apparent following the ego depletion manipulation, as would be predicted by those models.

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