Investigating mediators and moderators of the alcohol priming effect

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Graeme Knibb

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Contents

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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Abstract

Acute doses of alcohol can increase subsequent craving and alcohol consumption. This 'alcohol priming effect' may be an important determinant of both relapse among alcohol-dependent individuals, and binge drinking behaviours among social drinkers. This thesis aimed to investigate potential mediators and moderators of the alcohol priming effect. Current models have proposed alcohol-induced impairments of inhibitory control to underlie the alcohol priming effect (Field et al, 2010). However, there is currently inconclusive evidence for this claim. The overarching aim of this thesis was to, therefore, clarify the extent to which acute alcohol consumption indirectly affects subsequent consumption via these impairments. Each experimental study included a measure of inhibitory control (a stop-signal task), administration of alcohol and a measure of *ad libitum* alcohol consumption. This data was then synthesised in chapter 7. Overall, there was no effect of alcohol on inhibitory control and, therefore, inhibitory impairments did not mediate alcohol priming. This thesis also investigated two potential novel moderators of the alcohol priming effect; the role of beliefs about alcohol's acute effects (chapter 6) and the effect of social influences (chapters 3, 4 and 5). Belief's regarding the effects of alcohol were not found to affect *ad lib* alcohol consumption but did affect alcohol-induced impairments of inhibitory control. In addition, the alcohol priming effect had little effect on drinking when in the presence of others. Notably, alcohol consumption was increased in the presence of others relative to when alone (chapter 4) and when exposed to a heavy, relative to a light, drinking friend (chapter 5) regardless of whether a priming dose had been consumed. In addition, the association between self-reported peer and personal alcohol consumption was moderated by urgency (one facet of impulsivity) but only when drinking induced disinhibition was low (chapter 3). Overall these findings suggest the importance of the alcohol priming effect as a determinant of binge drinking is minimal. Furthermore, these findings suggest that the effect of alcohol on inhibition is exaggerated in the current literature. Previous findings may have been influenced by individual beliefs regarding the acute effect of alcohol. Other people's drinking does; however, appear to exert a consistent effect on personal alcohol consumption. Models of alcohol priming require considerable adjustment in light of these findings.

1 Chapter 1: General Introduction

1.1 Alcohol prevalence and harms

Alcohol consumption is prevalent across the globe. In 2015 adults (people aged 15+) consumed approximately 6.43 litres of alcohol per capita (LPC) with 18.4% of adults reporting at least one incidence of heavy episodic drinking within the previous 30 days (WHO, 2016). In particular, Central (11.64 LPC), Eastern (11.55 LPC) and Western Europe (11.13 LPC) reported the highest rates of LPC. An estimated 63.5 million cases of alcohol dependency were recorded worldwide in 2015, making alcohol dependency the most common form of dependency across the world, with the highest rates recorded in Eastern Europe and the lowest in North Africa and the Middle East (WHO, 2016).

The United Kingdom (UK) has particularly high levels of alcohol use, with a greater LPC and a higher prevalence of heavy drinking than the European average (WHO, 2016). Indeed, within the UK, 57% of people aged 16 or older reported consuming alcohol in 2017 (29.2 million people). Among the countries of the UK, adults in England were the most likely to have consumed alcohol in the past week (57.8%; HSCIC, 2016). Furthermore, self-report estimates suggest approximately 11.5 UK units are consumed per adult per week, although alcohol sales in the UK indicate this amount to be 20.4 units, suggesting self-report data currently underestimates alcohol consumption within the UK (Boniface, Kneale, & Shelton, 2014).

Consistent heavy drinking can result in alcohol dependency. Recently, within England, the number of individuals undergoing treatment for alcohol dependency has reduced from 85,035 in 2015-2016 to 80,454 patients in 2016-2017. However, according to estimates the vast majority of those with alcohol dependency are not currently undergoing treatment, with estimates of alcohol-dependent individuals being approximately 595,000 (Public Health England, 2017). Chronic alcohol use is a major risk factor for early mortality and disability and is a contributing factor for a variety of diseases and disorders including; liver cirrhosis, a variety of cancers, gastrointestinal diseases,

neuropsychiatric disorders, foetal alcohol syndrome, diabetes, cardiovascular diseases as well as alcohol-related injuries (e.g. Peacock et al., 2018; Rehm & Imtiaz, 2016).

In 2016 alcohol use was identified as being the seventh leading risk-factor for global disability-adjusted life years (DALY's) and mortality with 85 million of DALYs and 33 per 100,000 deaths attributable to alcohol (Peacock et al., 2018). Within the UK, alcohol has been suggested to be responsible for 10% of all DALYs, which are primarily due to; alcohol-related cirrhosis, depression, cerebrovascular disease, cancers, automobile accidents, unintentional and intentional injuries (Balakrishnan, Allender, Scarborough, Webster, & Rayner, 2009).

According to the National Health Service (NHS, 2018), in the UK, there were 1.1 million alcohol-related hospital admissions in 2016/2017, which is 1% higher than in 2015/2016. In 2016 there were 7,327 alcohol-specific deaths which is 4% higher than in 2015 (11.7 deaths per 100,000; HSCIC, 2016) and an increase of 11% since 2006. Importantly, while the majority of these outcomes may be due to chronic alcohol use, a large proportion is also due to acute alcohol intoxication. Indeed, excessive alcohol consumption or 'binge drinking' (Wechsler & Isaac, 1992) is also associated with alcohol-related injury and death. For example, acute alcohol intoxication has been suggested to be a factor in approximately 42% of alcohol-related deaths and 46% of estimated potential life years lost (Chikritzhs, Jonas, Stockwell, Heale, & Dietze, 2001).

1.1.1 *Binge drinking*

The term 'binge drinking' was first used by Wechsler and Isaac (1992) to describe excessive alcohol consumption on a single drinking occasion. Since then a variety of definitions regarding what constitutes a drinking 'binge' have been suggested. For example, the National Institute on Alcohol Abuse and Alcoholism (National Institute on Alcohol & Alcoholism, 2004) defined binge drinking as drinking occasions which results in a blood alcohol concentration that exceeds .07. Currently, binge drinking is often defined as drinking a given number of standard drinks or alcohol units on a single occasion. However, while a number of countries have developed alcohol guidelines, which define what is considered a standard measure or unit, in an attempt to aid self-monitoring of alcohol

consumption (Kalinowski & Humphreys, 2016), these guidelines often vary and have changed over time. For example, within the UK, these guidelines take the form of 'alcohol units' (Department of Health, 1995). At the time of writing, these guidelines recommend that (regardless of sex) people do not exceed 14 UK units of alcohol per week (HSCIC, 2016), with these units spread over three or more days. This limit was introduced due to economic modelling suggesting alcohol consumption over 14 units to lead to increased harm (Alan et al., 2015). Currently, these guidelines do not offer guidance on daily limits of alcohol consumption. However, previous UK guidelines suggested that males should not exceed 3 to 4 UK units per day, while females were recommended to consume no more than 2 to 3 UK units (Department of Health, 1995). According to these previous guidelines, a drinking occasion was considered to constitute a 'binge' if the amount consumed exceeded this daily dose (HM Government, 2012). Despite the changes to these recommendations, a number of organisations (e.g. HSCIC, 2016) have continued to use this as a definition of binge drinking.

In the UK, approximately a quarter (26.8%) of adults binge drink regularly and binge drinking is also prevalent among young adults (aged 16-24) with estimates suggesting that 20% had binged within the last week (2016/2017; NHS, 2018). Binge drinking is particularly prevalent among university students. A recent systematic review, which assessed research from the last 10 years, suggested that approximately two thirds of University students in the UK are hazardous drinkers; with estimates of weekly binge drinking ranging from 70-85% and the percentage of students engaging in binge drinking within the past 12 months estimated to be approximately 83% (Davoren, Demant, Shiely, & Perry, 2016).

Individuals who binge drink may be at an increased risk of alcohol-related harms compared to those who regularly drink in moderation. For example, Hingson, Zha, and White (2017) found individuals who consume three or more times the recommended daily dose of alcohol in the US to be at greater risk of drink-driving and, following alcohol consumption, physical confrontation, injury, admittance to an emergency department, being arrested or subject to other legal issues. Within the UK binge drinking is associated with over a million crimes (Chaplin, Flatley, & Smith, 2011; NHS, 2018) including interpersonal violence, sexual violence and homicide (Abbey, 2002; Brewer & Swahn,

2005; Perkins, 2002) and can also lead to a number of adverse secondary effects such as poor academic achievement at school/university, problems at work and problems maintaining relationships (Humensky, 2010; Pedersen, 2013). Furthermore, binge drinking is associated with poorer mental wellbeing with high rates of anxiety, depression and suicidal ideation reported by binge drinkers (Norberg, Olivier, Alperstein, Zvolensky, & Norton, 2011; Pedersen, 2013; Schaffer, Jeglic, & Stanley, 2008). This social cost of binge drinking is reflected economically, with costs to the police force and health services within the UK, due to binge drinking, being approximately £21 billion (HM Government, 2012).

Critically, a number of studies have demonstrated an association between binge drinking in adolescence and the subsequent development of an alcohol use disorder (e.g. Chassin, Pitts, & Prost, 2002; Viner & Taylor, 2007). However, while it is not yet clear whether this association is causal or indicative of other underlying factors, recent work has shown a number of neural impairments to be associated with binge drinking behaviour among young adults, which are also present when controlling for potential confounds such as familial alcohol misuse, comorbid psychopathologies and comorbid substance use (Courtney & Polich, 2010; Maurage et al., 2012; Townshend, Kambouropoulos, Griffin, Hunt, & Milani, 2014). Furthermore, these impairments seem to match those observed among alcohol-dependent patients although at a reduced magnitude. This supports the notion that binge drinking and alcohol use disorder represent different stages on a continuum (Courtney & Polich, 2010).

Binge drinking is, therefore, associated with a wide variety of adverse outcomes and significant economic cost. If effective interventions are to be developed, then it is important to understand this phenomenon. Indeed, a variety of mechanisms, correlates and theoretical frameworks of binge drinking have been proposed. This thesis focuses on one prominently researched mechanism; the alcohol priming effect. The following section reviews previous work which has investigated substance priming in general before focusing on alcohol priming specifically.

1.2 Substance Priming

Substance priming concerns the ability of acute doses of a substance to precipitate further substance-seeking behaviours such as craving and consumption (de Wit, 1996). Substance priming research has its roots in animal models of substance use. For example, early studies show that animals would resume self-administering a drug following an acute dose of that drug, despite this behaviour previously being extinguished (de Wit & Stewart, 1981, 1983; Stretch & Gerber, 1973). For example, priming doses reinstate drug use among animals for heroin, (Leri & Stewart, 2001), morphine (Mueller, Perdikaris, & Stewart, 2002) cocaine (Anker & Carroll, 2010; Mueller & Stewart, 2000), nicotine (Budzynska, Kruk, & Biala, 2009; Chiamulera, Borgo, Falchetto, Valerio, & Tessari, 1996; Shaham, Adamson, Grocki, & Corrigall, 1997), amphetamine (Alderson, Latimer, Blaha, Phillips, & Winn, 2004) and methamphetamine (Yu et al., 2011). This effect has also been shown in humans with acute doses of nicotine (Perkins, Grobe, & Fonte, 1997), cocaine (Dudish-Poulsen & K. Hatsukami, 1997; Jaffe, Cascella, Kumor, & Sherer, 1989; Walsh, Geter-Douglas, Strain, & Bigelow, 2001), and cannabis (Curran, Brignell, Fletcher, Middleton, & Henry, 2002) all producing craving and/or consumption of these substances. However, due to both ethical and legal issues concerning the administration of illicit substances, the majority of research has assessed the alcohol priming effect. Indeed, alcohol lends itself particularly well to the assessment of priming effects as humans often consume a number of drinks over a period of time whereas consumption of other drugs (i.e. nicotine) can lead to satiation (McMorrow & Foxx, 1983).

1.2.1 *The alcohol priming effect*

Initially, human studies of alcohol priming focused on priming effects among alcoholic patients. Indeed, the alcohol priming effect is particularly relevant for alcohol-dependent patients as priming is likely an important determinant of relapse. ‘Slip drinks’ have been anecdotally reported to precipitate relapse and research into the alcohol priming effect supports this. For example, Ludwig and Wikler (1974) administered a priming dose of alcohol and a placebo to alcohol-dependent patients. Alcohol, but not placebo, led to increased craving and the number of times participants were prepared to press a button to obtain more alcohol. Other work has found alcohol-dependent patients

to, following a priming dose of alcohol, work harder to obtain alcohol by riding on an exercise bike (Bigelow, Griffiths, & Liebson, 1977; Ludwig, Wikler, & Stark, 1974) and, severely, but not moderately, alcohol-dependent patients have been shown to consume more alcohol, drink more quickly and report greater desire to drink following a prime (Hodgson, Rankin, & Stockwell, 1979).

The majority of more recent alcohol priming research has focused on priming effects among social drinkers. Research has robustly demonstrated an alcohol priming effect on subjective measures of alcohol-seeking (i.e. craving and desire for alcohol). This effect may occur following relatively small doses, for example, Chutuape, Mitchell, and de Wit (1994) found a small dose of alcohol, 0.25 grams of alcohol per kilogram of body weight (0.25g/kg), to lead to increased desire for alcohol 30 minutes, but not 60 minutes, following alcohol consumption. The authors also found 0.50g/kg of alcohol increased desire for alcohol 30 minutes and 60 minutes later. Additionally, using a concurrent choice procedure, increased preference for alcohol was found, relative to money, following both doses of alcohol. Conversely, de Wit and Chutuape (1993) 0.25g/kg did not increase craving 30 or 60 minutes letter but did find increased craving following a 0.50g/kg dose. However, both doses of alcohol increased the likelihood participants would subsequently choose to consume additional alcohol over a monetary reward. Also using a 0.25g/kg dose, Fillmore (2001) found participants who are highly preoccupied with alcohol to report increased desire to drink following an alcohol prime. Greater doses of alcohol (0.55/0.60g/kg) have also been shown to increase craving relative to placebo (Fillmore & Rush, 2001; Rose, Jones, Clarke, & Christiansen, 2014) and increase the likelihood of choosing alcohol over low-value monetary rewards (Fillmore & Rush, 2001).

A smaller number of studies have also demonstrated acute doses of alcohol to increase alcohol consumption in the lab. These studies often employ a ‘bogus taste test’ (Jones et al., 2016) wherein participants are provided with a number of alcoholic drinks, ostensibly in order to rate their taste on a number of scales. In reality, the *ad libitum* alcohol consumption of participants is measured. Using this method, a 0.40g/kg (Fernie, Christiansen, Cole, Rose, & Field, 2012), 0.60g/kg (Baines, Field, Christiansen, & Jones, 2019) and 0.65g/kg (Christiansen, Rose, Cole, & Field, 2013) doses

have been shown to increase *ad lib* alcohol consumption. This suggests moderate to high doses, ranging from 0.40g/kg-0.65g/kg to effect subsequent alcohol consumption using this paradigm.

There have, however, been some inconsistent findings within the alcohol priming literature. For example, Adams et al (2012) found craving for alcohol to decrease, rather than increase, five minutes following a 0.40g/kg dose. Likewise, Adams et al (2017) found craving to increase over time regardless of whether a 0.40g/kg, a 0.60g/kg dose or placebo alcohol was administered but did not find craving to be greater within alcohol conditions. Both these findings may be due to the use of VAS scales rather than validated questionnaires. Other studies which have used validated measures have also failed to find an effect of alcohol (0.40g/kg and 0.75g/kg) on craving (Attwood, Penton-Voak, Goodwin, & Munafo, 2012; Evans & Bisaga, 2009); however, these studies were both underpowered to detect such an effect.

1.2.2 *The effect of dose, time and subjective response*

The alcohol priming effect is influenced by the dose of alcohol administered as well as the latency from administration to assessment of alcohol-seeking. For example, Rose and Duka (2006) found craving for alcohol, and hypothetical choice for alcohol, increased 30 minutes after a 0.60g/kg, but not following a 0.30g/kg, dose. Importantly, craving began to decrease following 30 minutes, and also significantly declined 60 and 90 minutes later. Similarly, Rose and Grunsell (2008) found an acute dose of alcohol (0.60g/kg for males, 0.50g/kg for females) increased craving for alcohol 30 minutes later, but no effect was found for a placebo. These findings suggest that the priming effect is contingent on both dose and time, with larger priming effects present following moderate to high doses of alcohol (i.e. 0.50g/kg-0.80g/kg) relative to low doses and, approximately 30 minutes following alcohol administration. The effect of time on priming may be due to blood alcohol levels (BALs) which differ across time. Importantly, these two studies (Rose and Duka, 2006; Rose and Grunsell, 2008) showed that BALs peaked concurrently with craving. Indeed, there is evidence to suggest that the subjective effects of alcohol are dependent on blood alcohol levels with stimulant and sedative effects being present at different points of the blood alcohol curve; known as the biphasic

effects of alcohol (Earleywine & Erblich, 1996; Holdstock & de Wit, 1998). Stimulant effects of alcohol are associated with the initial increase in BAL following consumption (the ascending limb of the blood alcohol curve) and sedative effects have been associated with decreasing BAL (the descending limb).

Subjective responses to alcohol, in particular, susceptibility to the stimulant effects of alcohol on the ascending limb of the blood alcohol curve and reduced responsiveness to the sedative effects on the descending limb of the blood alcohol curve may, therefore, promote alcohol priming. This is supported by King, Houle, de Wit, Holdstock, and Schuster (2002) who found alcohol increased stimulation on the ascending limb, relative to placebo, as well as sedation during the descending limb of the blood alcohol curve. Importantly, desire for alcohol was shown to increase during the ascending limb, the time at which stimulant effects are experienced. Furthermore, heavier drinkers were shown to be more susceptible to these stimulant effects and demonstrated a reduced response to alcohol's sedative effects while also reporting an increased desire for alcohol, relative to light drinkers, during the ascending limb. This effect was dose-dependent with a 0.80g/kg dose leading to greater effects than a 0.40g/kg dose. Corbin, Gearhardt, and Fromme (2008) also demonstrated 0.60g/kg of alcohol to increase both stimulation and sedation, relative to placebo and found stimulant effects following alcohol to predict subsequent *ad lib* alcohol consumption.

Initially, it was suggested that acute doses of alcohol elicit withdrawal symptoms in alcohol-dependent patients and, therefore, subsequent alcohol consumption was driven by negative reinforcement wherein patients attempt to alleviate withdrawal symptoms with additional alcohol (Ludwig et al., 1974; Rose, 2013). Despite the intuitiveness of this notion, this does not account for findings which have demonstrated priming effects among non-dependent social drinkers. Indeed, given that susceptibility to the stimulant effects of alcohol, on the ascending limb of the blood alcohol curve, is associated with the alcohol priming effect, it follows that positive reinforcement may underlie alcohol priming. This is consistent with learning theories which suggest the positive pharmacological effects of alcohol to drive further consumption (Stewart & de Wit, 1987; Stewart, de Wit, & Eikelboom, 1984).

1.2.3 *Genetic factors*

Given that the positive stimulant effects of alcohol are thought to underlie alcohol priming, a number of genetic variants which may affect the subjective effects of alcohol have been investigated as possible moderators of the alcohol priming effect. For example, healthy individuals and alcohol-dependent patients reported greater susceptibility to the positive subjective effects of alcohol if they possessed a polymorphism of the mu opioid receptor gene (OPRM1, A118G, rs1799971; Ray et al., 2013; Ray & Hutchison, 2004). Individuals with this variant also report greater craving following exposure to alcohol-related stimuli (van den Wildenberg et al., 2007) and self-administer greater amounts of alcohol (Hendershot, Claus, & Ramchandani, 2016; Hendershot, Wardell, McPhee, & Ramchandani, 2017). However, there are mixed findings as a number of genome-wide association studies and meta-analyses have found no evidence for an association between this genotype and a diagnosis of alcohol dependence (e.g. Arias, Feinn, & Kranzler, 2006; Schwantes-An et al., 2016). These contrary findings may be due to previous work employing relatively small sample sizes. A recent study aimed to resolve this by using an adequately powered study to test the association between alcohol consumption in the lab and the OPSM1 rs1799971 genotype and found no association between this genotype and subjective responses to alcohol or alcohol craving. Moreover, this study found no association between OPSM1 rs1799971 and alcohol consumption among dependent and non-dependent participants using a 90-day drinking diary (Sloan et al., 2018).

Another genetic variant which may alter subjective response to alcohol is GABRA2 (rs279858), however, there have been contradictory findings; with some suggesting GABRA2 to be associated with greater 'liking' (following an alcohol prime) for alcohol and greater positive stimulation (Haughey et al., 2008) and others suggesting these effects to be mitigated among carriers (Pierucci-Lagha et al., 2005). Research which has found alcohol response to be mitigated, however, did not assess the indirect effects of GABRA2 on consumption and used a non-dependent sample. In a recent study, Boyd, Schacht, Prisciandaro, Voronin, and Anton (2016) found non-treatment seeking alcohol dependents to report greater levels of stimulation following a priming dose of alcohol which was associated with greater *ad lib* alcohol consumption. Furthermore, there was an indirect effect of

GABRA2 on alcohol consumption, in that those the association between the genetic variant and increased alcohol consumption was mediated via stimulation.

1.2.4 *Implicit cognition*

Implicit cognitive processes have also been suggested to underlie the alcohol priming effect. Indeed, alcohol consumption and addictive behaviours more generally, have been suggested to be underwritten by these implicit cognitive processes which are automatic and occur outside of conscious awareness. Dual-process models of cognition propose two distinct systems; a 'quick' implicit impulsive association system which automatically evaluates cues in terms of their emotional or motivational properties, and a 'slow' explicit reflective system which includes processes such as deliberation, regulation and expectancies (Strack & Deutsch, 2004; Wiers et al., 2007). Through classical conditioning, this implicit system is argued to become automatically activated by substance-related cues. This leads to mere exposure to substance-related cues automatically triggering implicit associations, allocation of attention (known as 'attentional bias') and activation of approach responses (known as 'approach bias'). For a comprehensive review of implicit cognitive processes in addiction see Stacy and Wiers (2010).

Importantly, these implicit cognitive processes have all been shown to be related to alcohol-seeking. For example, a meta-analysis has shown that attentional bias towards alcohol-related stimuli, over neutral stimuli, to be positively associated with craving (Field, Munafò, & Franken, 2009). Likewise, approach biases have been shown to be related to *ad lib* alcohol consumption, hazardous drinking and relapse (Kersbergen, Woud, & Field, 2015; Martin Braunstein, Kuerbis, Ochsner, & Morgenstern, 2016) and positive implicit associations about alcohol have been shown to predict prospective hazardous drinking (Lindgren et al., 2016). Meta-analyses have shown implicit cognitions overall to be associated with substance use (Rooke, Hine, & Thorsteinsson, 2008) and explain variance over and above that explained by explicit measures (Reich, Below, & Goldman, 2010), however subsequent work suggests the amount of additional variance to be small (Blanton, Burrows, & Jaccard, 2016). Moreover, there is a multitude of inconsistent findings which suggest the clinical

relevance of implicit cognitions to be questionable (see Christiansen, Schoenmakers, & Field, 2015; Field, Marhe, & Franken, 2014; Snelleman, Schoenmakers, & van de Mheen, 2015).

While the role of these implicit cognitions for substance use is far from clear, it is important to note that the effect of alcohol on these processes has been studied. For example, as well as altering mood, feelings of sedation and stimulation, acute doses of alcohol may also increase the saliency of alcohol-related stimuli leading to an increased attentional bias towards alcohol. For example, Schoenmakers, Wiers, and Field (2008) found alcohol attentional bias to be increased following a 0.30g/kg dose of alcohol relative to placebo administration. Importantly, this suggests that only relatively low doses of alcohol are needed in order to potentiate attentional bias toward alcohol-related stimuli. Conversely, Duka and Townshend (2004) found no difference in alcohol attentional bias following a 0.30g/kg and a 0.60g/kg dose, however, as there was no placebo or control drink administered, it is possible that the 0.30g/kg dose was sufficient to increase attentional bias; with further dose increases having no additive effect. Doses lower than 0.30g/kg may, however, not increase alcohol attentional bias, for example, attentional bias has been shown to be present following 0.40g/kg of alcohol, relative to placebo, but to not be present following 0.13g/kg of alcohol (Adams, Ataya, Attwood, & Munafo, 2012). Other work has failed to demonstrate an effect of acute alcohol on attentional bias. For example, Miller and Fillmore (2011) found attentional bias towards alcohol-related stimuli to be similar following placebo alcohol, 0.32g/kg and 0.64g/kg dose of alcohol. In addition, Schoenmakers and Wiers (2010) conducted a field study in two pubs and found the amount of alcohol participants had consumed during that day to be positively correlated with craving, although, attentional bias was negatively correlated with the amount of alcohol participants had consumed further suggesting higher doses of alcohol do not significantly increase attentional bias.

It has been suggested that the effect of acute alcohol on alcohol attentional bias may, at least partially, mediate the alcohol priming effect (Field, Schoenmakers, & Wiers, 2008; Field, Wiers, Christiansen, Fillmore, & Verster, 2010). However, there is currently insufficient evidence for this claim. For example, Fernie et al. (2012) found a 0.40g/kg dose of alcohol to increase attentional bias towards alcohol-related stimuli among moderate but not heavy drinkers and found alcohol

consumption to be greater following alcohol relative to placebo for both heavy and moderate drinkers. However, the magnitude of this priming effect was not associated with the observed increase in attentional bias.

As well as attentional bias, several studies have also investigated the effect of acute alcohol consumption on other implicit measures such as implicit associations (i.e. whether alcohol consumption potentiates the association between alcohol and positively-valenced stimuli). In order to assess this, Implicit Association Tasks (IAT), which require participants to rapidly categorise stimuli as either positive or negative have been used (Greenwald, McGhee, & Schwartz, 1998). Theoretically, individuals who implicitly perceive alcohol positively should be quicker to categorise alcohol-related stimuli with positive stimuli than negative stimuli. Other studies have assessed the association between alcohol stimuli and approach or avoidance by having participants initiate a response towards or away from alcohol-related stimuli. To assess these approach biases Stimulus-Response Compatibility (SRC) and alcohol approach-avoidance (AAT) tasks are often used which assesses the latency of participants to approach or avoid alcohol-related stimuli. As with the IAT, shorter latencies to approach alcohol to suggest an implicit association between alcohol and, in this case, approach (Mogg, Bradley, Field, & De Houwer, 2003).

Surprisingly, there is a paucity of research which has investigated the effect of acute doses of alcohol on implicit associations using the IAT. Some research has; however, used the SRC task albeit with mixed results. For example, previous research which has assessed the effect of acute alcohol on approach biases has not found an effect when compared to placebo (Fernie et al., 2012; Jünger et al., 2017; Schoenmakers et al., 2008). However, a study that also included a control drink (i.e. a drink which participants know does not contain alcohol), found both alcohol and placebo to increase approach biases towards alcohol-related stimuli relative to the control (Christiansen et al., 2013).

Taken together, the relevance of implicit cognition for alcohol priming is unclear. There is currently inconsistent research regarding the effect of alcohol on attentional biases (e.g. Adams et al, 2012; Miller & Fillmore, 2011), which at present suggest attentional bias is not associated with alcohol priming (Fernie et al, 2012). There is a lack of research which has assessed the effect of

alcohol on implicit associations. However, approach biases may be affected by alcohol, albeit via the anticipated rather than the pharmacological effects of alcohol (Christiansen et al, 2013).

1.2.5 *Cross-priming*

Administration of a priming dose of a given substance may also increase the consumption, and saliency, of substances other than that which was originally administered; a process known as cross-priming. For example, the administration of an alcohol prime has been shown to increase food intake and craving (Caton, Ball, Ahern, & Hetherington, 2004; Christiansen, Rose, Randall-Smith, & Hardman, 2016; Rose, Hardman, & Christiansen, 2015; Yeomans, 2010), cocaine craving (Marks, Pike, Stoops, & Rush, 2015), and, along with d-amphetamine, increased craving for tobacco smoking and subsequent reduced latency to smoke (Cousins, Stamat, & de Wit, 2001; Kahler et al., 2014; King, McNamara, Conrad, & Cao, 2009) Administration of diazepam has been shown to increase craving for, and consumption, of alcohol (Poulos & Zack, 2004) and alcohol primes have been shown to increase the saliency of smoking and cocaine-related stimuli (Field, Mogg, Zetteler, & Bradley, 2004; Montgomery et al., 2010).

1.2.6 *Acute alcohol consumption and 'loss of control'*

One explanation for cross-priming is that the consumption of a rewarding substance primes general, rather than substance-specific, reward-seeking. Consistent with this notion, the stimulating effects of alcohol, during the ascending limb of the blood alcohol curve, should mediate the cross-priming effects of alcohol. However, this is disputed by research which has demonstrated the effect of alcohol on urge and latency to smoke to be unaffected by alcohol's stimulant and sedative effects (Kahler et al., 2014).

An alternative conception is that alcohol (and substance) priming impairs individual's capacity to self-regulate their behaviour, leading to the continued consumption of available rewarding substances, be it further alcohol or, for example, food. Consistent with this notion, Christiansen et al (2016) found a 0.6g/kg dose of alcohol, relative to placebo, to lead to increased consumption of food. Importantly, this effect was mediated by performance on a colour-conflict Stroop task (Stroop, 1935)

used to assess participants' inhibitory control; the ability to control behaviour in order to inhibit a pre-potent response. In this instance, alcohol-impaired performance on the Stroop task and this impairment was associated with increased food consumption. Furthermore, this occurred despite no increase in food craving between alcohol and placebo sessions, suggesting the increased food consumption to reflect a lack of control over eating rather than increased desire.

For many years it has been suggested that alcohol may lead to a 'loss of control' over behaviour. For example, Jellinek (1952) proposed that if alcohol-dependent patients were to consume a small dose of alcohol this may trigger a binge drinking episode. In addition, acute alcohol intoxication can lead to a variety of disinhibited behaviours e.g. verbal disinhibition, aggression and risky sexual behaviour (Cooper, 2002; Kallmen & Gustafson, 1998; Parrott & Eckhardt, 2018). Central to these findings is the notion that alcohol impairs the ability of individuals to regulate their behaviour, leading to disinhibition. Indeed, a number of models (e.g. Field et al., 2010) have suggested that the effects of alcohol on mechanisms, such as inhibitory control, may promote the use of further alcohol and other substances (the alcohol priming effect) and it is this process which the current thesis is concerned with. Therefore, the following sections outline previous research on inhibitory control and the related concept of impulsivity before discussing the association between inhibitory control and alcohol use.

1.3 Inhibitory control: measures and neural mechanisms

Inhibitory control refers to the ability to inhibit pre-potent and/or inappropriate responses (Fillmore & Weafer, 2013) and is the antithesis of disinhibition (i.e. poor inhibitory control is indicative of disinhibition). A number of measures have been devised to assess inhibitory control; the next section outlines the most prevalent.

1.3.1 *Inhibitory control measures*

1.3.1.1 Stroop task

During the colour-conflict Stroop task (Stroop, 1935) participants are provided with names of colours presented in a variety of different colours. Participants are required to read the colour that the

word is written in and to disregard the semantic content of the word. The difference in the latency to read words on trials wherein the word and colour are incongruent relative to when the word and colour are congruent is used as the main outcome measure of inhibitory control in this task. Greater latencies suggest participants had greater difficulty inhibiting the pre-potent response to read the words and are therefore considered to be indicative of poorer inhibitory control.

1.3.1.2 Anti-saccade task

While inhibitory control measures often employ manual responses, inhibitions of oculomotor movements have also been assessed (Logan & Irwin, 2000; Roberts, Fillmore, & Milich, 2011). Importantly, manual and ocular inhibitory control may be independent, utilise distinct inhibitory systems and ocular response latencies are smaller; perhaps reflecting a reflexive action rather than pre-potent responses present in manual measures of inhibition. As saccadic movements have been associated with allocation of attention (Godijn & Theeuwes, 2003a), oculomotor disinhibition may reflect, and be more closely related to attention than is manual disinhibition.

A popular measure of oculomotor disinhibition is the anti-saccade task (AS; Hallett, 1978). This task involves making a saccade away from a target stimulus which appears on a computer screen. Errors to inhibit the reflexive action to saccade towards the target stimulus and latency to saccade away are used as the main outcome measure of inhibitory control in these tasks (Logan & Irwin, 2000). Conversely, alternative versions of the task, such as the delayed ocular response task, require participants to delay rather than inhibit saccades (Godijn & Theeuwes, 2003b; Weafer, Milich, & Fillmore, 2011).

1.3.1.3 The go/no-go (GNG) task

A GNG task requires participants to respond to a stimulus presented on a computer screen (go stimuli) as rapidly as possible. On other occasions, participants are required to withhold responding to a certain stimulus (no-go stimuli). Go stimuli are most frequently presented (up to 90% of occasions) while no-go stimuli are less frequently presented (10% of trials); this produces a pre-potent propensity to respond, which participants are required to override (Gomez, Ratcliff, & Perea, 2007; Newman,

Widom, & Nathan, 1985). Commission errors (incorrectly responding to a no-go stimulus) and omission errors (incorrectly withholding a response when presented with a go stimulus) are often used as outcome measures on this task (e.g. de Wit, Enggasser, & Richards, 2002).

There are a number of variants of the GNG task. For example, the cued GNG task includes additional stimuli which are presented prior to go or no-go stimuli which indicates the likelihood that a particular stimulus is likely to be presented. This decreases response latencies on trials when a go cue is presented prior to go stimuli and increases errors for trials when a go cue is presented prior to a no-go trial (Miller, Schaffer, & Hackley, 1991). This may be due to participants preparing responses prior to the presentation of the go stimulus (Posner, 1980). Another derivative of the GNG task is the passive avoidance task or the go/no-go discrimination task (Helmers, Young, & Pihl, 1995; Newman et al., 1985). This task involves allocating random numbers as go and no-go stimulus and participants are required to complete the task in a similar manner to the standard GNG task while discriminating between numbers assigned as go-stimuli and those assigned as no-go stimuli. The mean number of commission and omission errors as well as total errors are typically used as outcome measures for this task (Yechiam et al., 2006).

1.3.1.4 Stop-signal task

The SST (Logan, 1994; Logan & Cowan, 1984) is conceptually similar to the GNG task but with some important differences. On each trial of this task, participants are presented with one of two go-stimuli (e.g. an arrow which points either left or right) and are required to press the appropriate key (e.g. the left arrow key if the stimuli points left, the right key if right). However, on a number of occasions (often 25%) a separate stop-signal is presented after these go-stimuli (e.g. an auditory tone), on these trials participants are required to inhibit responding.

Performance on the SST is often conceptualised as an 'independent horse race' (Logan & Cowan, 1984; Logan, Van Zandt, Verbruggen, & Wagenmakers, 2014; Verbruggen, Best, Bowditch, Stevens, & McLaren, 2014); between a go process and a stop process. The go process is initiated following the presentation of the go stimuli and the stop process begins upon the presentation of the

stop signal. Inhibition is successful on those occasions when the stop process ends earlier than the go process. Alternatively, on occasions when the go process ends before the stop process, inhibition is unsuccessful, and a response is incorrectly initiated.

An important aspect of the SST is the latency between the presentation of the go stimulus and the stop-signal; known as the stop-signal delay (SSD). Early work shows that participants are better able to inhibit responding when the latency of the SSD is short, while successful inhibition is unlikely with SSDs of 100ms and over (Vince, 1948). Therefore, longer SSD latencies increase the difficulty of successful inhibition. However, as a result of the SSD participants may delay responding (therefore delaying the go process) in order to ensure the probability of responding (*p*[respond|signal]) is equal over all trials (Lappin & Eriksen, 1966). SSD latencies were fixed within initial versions of the SST (Logan & Cowan, 1984), however, more recent iterations have employed tracking algorithms which progressively alter the SSD according to the participant's performance (e.g. Verbruggen, Logan, & Stevens, 2008).

A number of outcome measures can be derived from the SST including the number of errors (i.e. unsuccessful inhibitions following stop-signals) and mean reaction time to go stimuli, however, the main advantage the SST has over the GNG task is that the task is designed to enable the computation of stop-signal reaction time (SSRT). This outcome is the latency of the stop process itself which is not overtly measured. Instead, SSRT is computed using a stochastic process (Verbruggen, Best, et al., 2014). According to the independence horse race model (Logan & Cowan, 1984), the beginning of the SSD is the starting point of the stop process, while the end point of the stop process is derived from the distribution of the participants go reaction times, when no stop-signal is present, and *p*[respond|signal]) for a particular SSD. Therefore, SSRT can be calculated by deducting the SSD from this endpoint (Verbruggen & Logan, 2008).

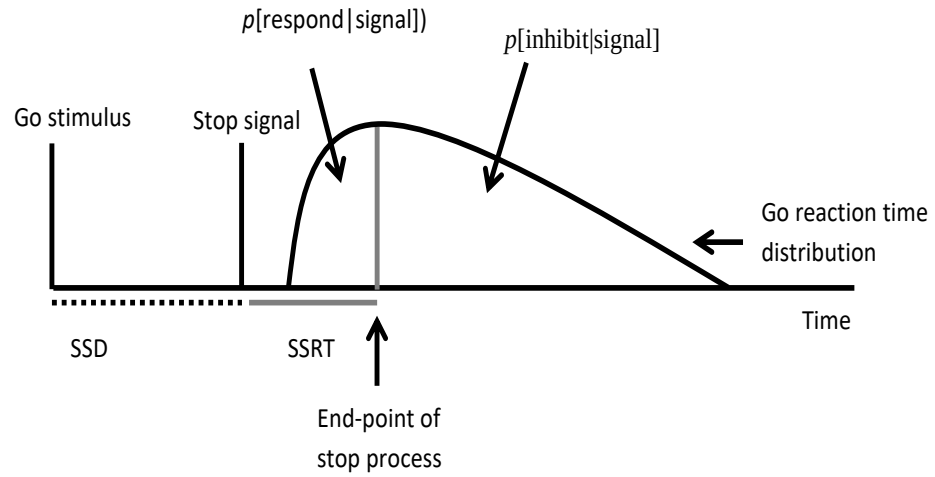

Figure 1. Schematic outline of the independent horse race model based on Verbruggen and Logan (2008).

While conceptually similar, it is important to note that the GNG task and the SST may assess different inhibitory processes (Littman & Takács, 2017). Indeed, the GNG task often includes the presentation of a no-go stimulus concurrently or as an alternative to a go stimulus. On the other hand, within the SST, stop signals are presented following the presentation of a go stimulus. Moreover, these tasks exert an effect on different neural circuitry; with the GNG task being more strongly associated with activation in the frontoparietal network, while the cingulo-opercular control network is affected to a greater magnitude by the SST (Swick, Ashley, & Turken, 2011). Distinct neurochemical mechanisms have also been implicated for both tasks; with serotonin seemingly involved with performance in the GNG task and noradrenaline involved with SST performance (Eagle, Bari, & Robbins, 2008; Eagle et al., 2009). These two tasks may, therefore, measure distinct components of inhibitory control; action restraint and action cancellation (Schachar et al., 2007; Wessel, 2017). As go and no-go stimulus are presented concurrently during the GNG task the go and stop processes are also initiated simultaneously. Therefore, the onus of the GNG task is to inhibit response in the preparation phase (action restraint). Alternatively, as the go stimulus is presented prior to the stop-signal during the SST, the go process had already begun and the emphasis in this task is on cancelling this action (action cancellation). Furthermore, action restraint is an automatic 'bottom-up' process which is assessed via the GNG task as the response and stimulus are repeatedly paired.

Alternatively, action cancellation is more effortful and 'top-down' as the stimulus and response are erratically paired and further control is required to cancel a response which has already been initiated (Verbruggen & Logan, 2008).

1.3.2 *Neural mechanisms*

A number of brain regions have been shown to be integral for successful cognitive control and inhibition. For example, individuals with frontal lesions have been shown to display disinhibited behaviour, implicating the prefrontal cortex (Holmes, 1938; Stuss & Alexander, 2007). The right inferior frontal gyrus (RIFG) may be particularly important, with damage to this area and transcranial magnetic stimulation, which can temporarily reduce activity in the target area, both being shown to result in greater SSRT's in a stop-signal task (Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Boehler, Appelbaum, Krebs, Hopf, & Woldorff, 2010; Chambers et al., 2007; Juan & Muggleton, 2012).

Medial prefrontal areas are also likely to be important for inhibition with damage to the left hemisphere, specifically the pre-supplementary motor area, and the dorsolateral prefrontal cortex being associated with poorer performance on a GNG task (Aron, Robbins, & Poldrack, 2014; Boehler et al., 2010; Picton et al., 2007). Importantly, prefrontal regions are not the only regions suggested to be involved in inhibition. The cerebellum (Rubia, Smith, Taylor, & Brammer, 2007), thalamic areas and the basal ganglia, particularly the striatum, may also be involved, with lesions of the basal ganglia inducing increases in SSRT's similar to that of those associated with frontal damage (Aron & Poldrack, 2006; Rieger, Gauggel, & Burmeister, 2003). Collectively these areas have been referred to as the inhibition-related network (Gan et al., 2014).

The neural mechanisms that underlie inhibition may differ from childhood to adulthood, with research suggesting inhibitory control improves with age (Tillman, Thorell, Brocki, & Bohlin, 2008). For example, a recent fMRI study (Rubia et al., 2013), which used a sample of 13 to 38-year-olds, found increased activation in the right inferior, dorsolateral prefrontal and temporoparietal regions, and the cerebellum with age during an SST. These increases in activation occurred alongside

decreased activation in several other brain regions including; both hemispheres of the ventrolateral orbitofrontal cortex, posterior right insula, the ventral striatum, the supplementary motor area, the cerebellum and the posterior cingulate. This age-related change in brain activation during inhibition suggests that inhibition in children involves brain regions which develop early on while adults employ regions which develop later. The authors argue that this is consistent with increased specialisation and cognitive maturation. Similar findings have been reported for other inhibitory control tasks such as the Stroop and GNG tasks (Andrews-Hanna et al., 2011; Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Rubia et al., 2006).

1.4 Inhibitory control: impulsivity and executive function

Inhibitory control has often been conceptualised as one component of the broad construct of 'impulsivity.' Impulsivity has been broadly described as a '…tendency to engage in inappropriate or maladaptive behaviours…' (de Wit, 2009 pp. 22) and impulsive acts as '…behaviours…performed with little or inadequate forethought…' (Evenden, 1999 pp. 349). It has been argued that impulsivity is an important determinant of substance use; contrariwise impulsivity may also be potentiated by both chronic and acute substance use (de Wit, 2009). However, impulsivity is a complex and multifaceted concept with no single definition with the term being applied to a range of seemingly distinct behaviours including; trait impulsivity, impulsive choice, and impulsive actions. This section briefly outlines the concept and components of impulsivity as well as the related construct of executive function.

1.4.1 *Impulsive traits*

Impulsivity has been considered a dimension of personality for many years, with numerous models of personality containing an aspect related to impulsivity. Given the range of scales which purport to assess trait impulsivity (e.g. Cloninger, Svrakic, & Przybeck, 1993; Dawe, Gullo, & Loxton, 2004; Gray, 1978; Tellegen & Waller, 2008; Zuckerman, 1994) and the variety of conceptualisations and discrepancies among them, there has been some attempt to identify core impulsive traits. Specifically, Whiteside and Lynam (2001) conducted a factor analysis on a number

of commonly used self-report scales of impulsivity and proposed four subscales; urgency, lack of premeditation, lack of perseverance and sensation seeking, ultimately developing the UPPS to assess these factors. Within the UPPS framework, urgency refers to the tendency to act impulsively due to high levels of (positive or negative) affect. This trait has since been split into two separate subscales (Cyders & Smith, 2007, 2008) called positive and negative urgency. With positive urgency referring to the tendency to behave rashly when experiencing positive affect and negative urgency the tendency to behave rashly when experiencing negative affect. However, given that positive and negative urgency are often highly positively correlated with one another, it has been suggested that a single urgency factor may be more suitable (Sharma, Markon, & Clark, 2014; Stautz, Dinc, & Cooper, 2017). Lack of premeditation refers to the propensity to act without planning, lack of perseverance refers to the inability to continue with a task and sensation seeking refers to the tendency to pursue new experiences (Zuckerman, 1994). The UPPS conceptualisation of impulsivity has been subsequently validated and is suggested to have good convergent and discriminant validity (Miller, Flory, Lynam, & Leukefeld, 2003; Smith et al., 2007; Whiteside, Lynam, Miller, & K. Reynolds, 2005).

Previous research shows there to be a relationship between alcohol/substance use and the UPPS conceptualisation of impulsivity and this relationship may differ by trait (Stamates & Lau-Barraco, 2017). A recent review found sensation-seeking to be strongly associated with alcohol use among adolescents and positive and negative urgency to be the best predictors of alcohol-related problems (Stamates & Lau-Barraco, 2017). Similarly, a meta-analysis of the relationship between UPPS traits and adolescent alcohol use (Stautz & Cooper, 2013) found all traits to be positively correlated with alcohol consumption and problematic alcohol use. However, alcohol consumption was more strongly associated with positive urgency and sensation seeking, while both positive and negative urgency was most strongly associated with problematic alcohol use. Furthermore, a subsequent meta-analysis, which assessed the relationship between UPPS and alcohol use (Coskunpinar, Dir, & Cyders, 2013), found similar associations between every impulsivity trait and drinking frequency, however, drinking quantity was associated most strongly with lack of

perseverance. Conversely, alcohol dependence was most strongly associated with lack of premeditation and negative urgency, while the magnitude of the association for problematic alcohol consumption was greatest for negative and positive urgency. Finally, binge drinking was most strongly associated with sensation seeking. However, when controlling for age, gender and total alcohol consumption sensation seeking is not associated with binge drinking, although negative urgency is (Bø, Billieux, & Landrø, 2016).

Indeed, additional research suggests urgency traits to be a particularly important determinant of substance use. For example, positive and negative urgency has been found to be associated with alcohol and cannabis use among adolescents (Stautz & Cooper, 2014b), while positive urgency has been shown to predict subsequent illicit drug, quantity of alcohol use and negative consequences of drinking among college students (Cyders, Flory, Rainer, & Smith, 2009; Zapolski, Cyders, & Smith, 2009). A recent meta-analysis found all UPPS traits, with the exception of lack of perseverance, to be related to cannabis use behaviours, particularly problematic use (VanderVeen, Hershberger, & Cyders, 2016). Certain UPPS traits may also relate to treatment outcomes, with recent work suggesting high negative urgency and lack of premeditation to be associated with poorer treatment outcomes for individuals undergoing psychotherapy for substance use (Hershberger, Um, & Cyders, 2017). Importantly, recent research has suggested positive urgency to be related to *ad lib* alcohol consumption in the lab but only among individuals manipulated into experiencing a positive mood state (Dinc & Cooper, 2015). This finding suggests that positive urgency may be related to alcohol consumption only when experiencing high levels of affect.

1.4.2 *Impulsive choice*

Another conceptualisation of impulsivity is the extent to which individuals prefer small immediate rewards over large long term rewards; known as delay discounting (DD; Ainslie, 1975; Bickel & Marsch, 2001). With individuals who often choose smaller immediate rewards considered to be more impulsive than those who more often opt for larger delayed rewards. From this perspective, impulsivity can be assessed by calculating the magnitude of discounting a reward due to a delay.

Similar concepts include probability discounting which concerns the inclination for larger less likely reward over smaller more likely rewards (Green & Myerson, 2004) and effort discounting which pertains to the tendency to discount large rewards which require greater effort relative to small rewards which require minimal effort (Prévost, Pessiglione, Météreau, Cléry-Melin, & Dreher, 2010).

An increased tendency to discount the value of rewards which are delayed has been shown to be greater among individuals with a variety of addictive behaviours; including smokers, problem gamblers, opiate users, cocaine users and individuals with alcohol dependency (Baker, Johnson, & Bickel, 2003; Coffey, Gudleski, Saladin, & Brady, 2003; Mitchell, Fields, D'Esposito, & Boettiger, 2005; Petry, 2001a, 2001b). DD is also associated with addictive behaviours over time and is predictive of response to treatment (Krishnan-Sarin et al., 2007; Stanger et al., 2012) and prospective alcohol involvement among adolescents (Fernie et al., 2013). Meta-analyses have shown DD rates to be greater among clinical samples relative to control samples and to be related to severity, quantity, and frequency of drug use (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017; MacKillop et al., 2011). DD tasks have also assessed preferences for drug-related rewards (i.e. "Would you prefer £20 now or 10 pints of beer in x" were x is a given time period) and a range of drugs have been found to be associated with steeper DD performance for that drug (Adams, Attwood & Munafo., 2017; Baker et al., 2003; Estle, Green, Myerson, & Holt, 2007; Mitchell, 2004).

1.4.3 *Factor structure of impulsivity*

Given the range of measures used to assess impulsivity, there have been a number of attempts to clarify its underlying factor structure (Caswell, Bond, Duka, & Morgan, 2015; Christiansen, Cole, Goudie, & Field, 2012; Mackillop et al., 2014; Reynolds, Ortengren, Richards, & de Wit, 2006; Sharma, Kohl, Morgan, & Clark, 2013; Sharma et al., 2014; Stahl et al., 2014), however, these findings have been mixed, with numerous factor structures being proposed.

Importantly, research has suggested that self-report measures of impulsive choice (i.e. DD tasks) and so-called impulsive action (i.e. GNG and SST) assess distinct facets of impulsivity. For example, Reynolds, Ortengren, et al. (2006) found behavioural measures to reflect two components.

The first component was deemed ‘impulsive disinhibition’ and corresponded to the GNG and SST. The second component, ‘impulsive decision-making’, included DD. In addition, trait self-report measures of impulsivity were generally unrelated to behavioural measures of impulsivity. Similarly, Christiansen et al. (2012) found DD and performance on a GNG task to fall under two separate factors. Non-planning impulsivity also loaded onto the DD factor as well as onto another factor. This other factor also included self-reported impulsivity (BIS 11).

Variations in the factor structure of impulsivity may be due to a lack of consistency in the measures used across studies and the inclusion, in some studies, of measures designed to assess other processes (i.e. memory and risk-taking). MacKillop et al. (2016) attempted to address this by using a range of commonly used assessments and avoided those which assessed factors related to, but distinct from, impulsivity. The authors found a three-factor structure of impulsivity, with ‘impulsive action’ comprised of performance on a continuous performance task, a GNG, and an SST. ‘Impulsive choice’ was the second factor and was derived from performance on a DD task and three monetary choice questionnaires. Finally, the ‘impulsive personality traits’ factor was a product of all UPPS subscales. Moreover, associations between these factors were low, with impulsive personality traits moderately related to impulsive action and impulsive choice, but with no relationship between impulsive choice and impulsive action.

1.4.4 *Impulsivity, inhibition, and executive (dys)function*

It is also important to note that impulsivity may be related to executive function. Executive function refers to a set of processes involved with regulation of thoughts and actions, consideration and planning for the future (Barkley, 2004). Work has suggested executive function to consist of three fundamental factors; the ability to alter or ‘shift’ attention between tasks or mental sets (‘set shifting’), the ability to update and manipulate working memory content (‘updating’ or simply ‘working memory’) and the ability to inhibit responding (inhibitory control; Lehto, Juujärvi, Kooistra, & Pulkkinen, 2003; Miyake et al., 2000). While these executive functions are all thought to stem from activation in the frontal-parietal network (Niendam et al., 2012) they are separable from one another,

with surprisingly low inter-correlations between them; a pattern which has been described as displaying 'unity and diversity' (Friedman & Miyake, 2017).

Rather than being separate constructs, executive function and impulsivity may be best conceptualised as being on opposite ends of a continuum (Bickel, Jarmolowicz, Mueller, Gatchalian, & McClure, 2012). With inhibition considered to be a component of executive function. Conversely, disinhibition, a failure to inhibit an action, is diametrically opposite to inhibition and has been conceptualised as a component of impulsivity. Therefore, the terms inhibition, response inhibition, inhibitory control, and disinhibition reflect the same process.

The three constructs of executive functioning have also been shown to load onto one factor deemed common executive function or the central executive (Friedman et al., 2008). Correlations between measures of inhibition have been shown to be subsumed by the common executive function factor, with inhibition, in particular, being highly correlated with the common factor (Miyake & Friedman, 2012), leading to the absence of an inhibition factor (Friedman et al., 2008; Ito et al., 2015). It has been argued that this suggests inhibition to be the primary factor of executive function (Hall & Fong, 2015) and synonymous with common executive function (Valian, 2015; Zacks & Hasher, 1994). Alternatively, it has also been suggested that inhibition may be more reliant on the common factor than set shifting and updating. This common mechanism has been proposed to involve the maintenance and management of goals while simultaneously countering interference (Friedman & Miyake, 2017; Friedman et al., 2008; Munakata et al., 2011). This may be more important for inhibition (over the other two factors) as successful performance on inhibitory control tasks requires participants to successfully counter interference (pre-potent responses) in order to perform well (Friedman & Miyake, 2017).

1.5 Inhibitory control: Deficient inhibition and substance use

Deficiencies in inhibition have been investigated both as a risk factor for and as a consequence of damaging long-term substance use (de Wit, 2009; Grant & Chamberlain, 2014; Jentsch et al., 2014; Perry & Carroll, 2008; Weafer, Mitchell, & de Wit, 2014). Research investigating

the inhibitory control of substance dependent individuals, prospective studies, which have investigated the predictive utility of inhibitory control on subsequent use, and research concerning inhibition among participants with substance dependent relatives have been conducted to assess the potential causal nature of inhibitory deficits on substance use. This section outlines research which suggests inhibitory deficits to be a consequence and risk factor for substance use. In addition, the effect of acute alcohol consumption on inhibition and how this may underlie the alcohol priming effect is discussed.

1.5.1 *Deficient inhibition among substance users*

A number of models have proposed deficient inhibition to result from substance use itself and, therefore, perpetuating further use. Previous work has suggested addictive behaviour to be the result of the positive reinforcing effects of substances which act on the mesolimbic dopamine system and promote dopamine release and, thus, salience (e.g. Robledo & Koob, 1993). This response becomes paired with the substance and the behaviour is reinforced. Further work suggested that repeated administration of a substance can lead to the substance, as well as cues related to that substance, becoming endowed with 'incentive salience' leading to increased attention and approach towards the substance in question (Robinson & Berridge, 1993). More recent neurobiological approaches to addiction, however, highlight the role of neuroadaptations that occur as a consequence of chronic substance use (Uhl, Koob, & Cable, 2019). For example, Jentsch and Taylor (1999) proposed that damage to the frontal cortex, due to chronic substance use, which has been demonstrated in both humans and animals (Calu et al., 2007; Jentsch & Pennington, 2014), may lead to impairments in inhibitory control leading to an inability to effectively inhibit substance-seeking behaviour (disinhibition). Similarly, Goldstein & Volkow (2002; 2011) propose a model of addiction known as the I-RISA syndrome of drug addiction which suggests drug use to be underwritten by incentive salience but that disinhibition, due to chronic substance use, may lead to increased relapse and bingeing as individuals are unable to inhibit the automatic approach tendencies towards substances. Thus, suggesting inhibition to exert top-down control on these automatic responses.

There is substantial evidence that substance users have greater inhibitory deficits than do non-users, with deficits in inhibitory control observed among users of; cannabis (e.g. Behan et al., 2014; Moreno et al., 2012), cocaine (e.g. Crunelle, Veltman, van Emmerik-van Oortmerssen, Booij, & van den Brink, 2013; Fillmore & Rush, 2002; Hester & Garavan, 2004; Kaufman, Ross, Stein, & Garavan, 2003; Li, Milivojevic, Kemp, Hong, & Sinha, 2006; Verdejo-Garcia, Perales, & Perez-Garcia, 2007), methamphetamine (e.g. Monterosso, Aron, Cordova, Xu, & London, 2005) opiates (e.g. Fu et al., 2008), khat, (Colzato, Ruiz, van den Wildenberg, Bajo, & Hommel, 2010) and among alcohol-dependent patients (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009; Li, Luo, Yan, Bergquist, & Sinha, 2009). There have, however, been some inconsistent findings which report no inhibitory deficits among substance and alcohol users (e.g. Colzato et al., 2010; Fernie, Cole, Goudie, & Field, 2010; Gamma, Brandeis, Brandeis, & Vollenweider, 2005; Quednow et al., 2007; Yang et al., 2009).

These inconsistencies may be due to the varying measures which have been employed and lack of statistical power. Therefore, a number of meta-analyses have been conducted in order to clarify differences in inhibition among substance users. For example, a recent meta-analysis found disinhibition, assessed using a GNG task, to be moderately impaired among individuals defined as having an addiction (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). A comparable meta-analysis also found moderate impairment in SSRT among individuals diagnosed as addicts (Lipszyc & Schachar, 2010), in particular, alcohol and cocaine dependent-patients. A recent, more comprehensive, analysis (Smith, Mattick, Jamadar, & Iredale, 2014) assessed deficits in inhibitory control among users across a variety of different substances and did not collapse the analysis across drug types. This meta-analysis found larger effect sizes when an SST was used in comparison to the GNG task; suggesting the SST to be more sensitive to deficits in inhibitory control. Overall, deficits were found in users of cocaine, MDMA, tobacco, methamphetamine, and khat. However, there were no deficits found, regardless of measure, for cannabis and opiate users and no deficits observed for tobacco smokers using the SST. Performance on all measures of inhibition was impaired among alcohol-dependent patients; while non-dependent heavy drinkers displayed deficits on the SST but no deficits on GNG tasks. Again, this suggests the SST is a sensitive measure of inhibitory deficits

among non-dependent alcohol users. Furthermore, a recent meta-analysis of imaging studies (Zilverstand, Huang, Alia-Klein & Goldstein, 2018) analysed the results of 30 studies which compared substance users to non-users while they performed inhibitory control tasks. Overall, only 13 of these studies found poorer inhibitory control among substance users. However, the authors note that many of the studies which found no difference did not contain adequate control conditions. In addition, 23 studies did find differences between the two groups in brain activity with substance users presenting decreased activity in several regions including; the anterior insula, the dorsal anterior cingulate, the inferior parietal lobule, as well as the ventro and dorso- lateral prefrontal cortex (reflecting brain regions previously outlined as underlying inhibitory control; see section 1.3.2).

As well as differentiating between alcohol-dependent patients and non-dependents, inhibitory control impairments may be on a continuum so that the severity of impairment is related to alcohol use even among non-clinical samples (Parsons, 1998). Indeed, deficits on a variety of inhibitory measures have been found to be associated with alcohol use. This includes; the SST (Nederkoorn et al, 2009), standard GNG tasks (Weafer et al, 2011b), a GNG task with embedded images (Czapla et al 2015); the passive avoidance GNG task (Colder & O'Connor, 2002), and the related GoStop task (Houston et al, 2014) but not the antisaccade task (Weafer, Milich & Fillmore, 2011).

In addition to alcohol consumption, craving may also be moderated by inhibitory control with previous work finding craving following exposure to alcohol-related cues to be greater among social drinkers and alcohol-dependent patients with poorer inhibitory control (Papachristou, Nederkoorn, Corstjens, & Jansen, 2012; Papachristou et al., 2013; Papachristou, Nederkoorn, Havermans, Van Der Horst, & Jansen, 2012).

1.5.2 *Deficient inhibition as a risk factor*

While these studies, investigating the association between substance use and inhibition, provide some insight, it is not possible to properly assess whether deficiencies in inhibitory control are indeed a consequence of substance use or if individuals with poorer inhibitory control are more likely to use substances. Poor inhibitory control could, therefore, constitute a risk factor for substance

use. Indeed, a number of prospective studies have been conducted which help clarify the role of inhibition on substance and alcohol use. For example, Fernie et al. (2013) found performance on an SST among children aged 12-13 to predict alcohol use every six months across a period of two years. Importantly, alcohol use was not found to predict SST across the six-time points, suggesting a causal relationship. A similar finding is reported by Rubio et al. (2008) who found SST performance to significantly predict the likelihood of alcohol use disorder in adults 4 years later.

Further supporting the notion that inhibitory deficits constitute a risk factor for substance use is the finding that close relatives of individuals with substance dependence perform worse on measures of inhibition than matched controls (Acheson, Richard, Mathias, & Dougherty, 2011; Ersche et al., 2012; Gierski et al., 2013). This finding is particularly important given that close relatives (particularly offspring) of individuals with substance use disorder have an increased likelihood of dependency themselves (Begleiter & Porjesz, 1999) suggesting a heritable basis for deficient inhibition and subsequent dependency (Gierski et al., 2013). Consistent with this, Nigg et al. (2006) found SST performance among adolescents to predict alcohol-related problems, illicit drug use and comorbid alcohol and illicit drug use and this occurred even when controlling for IQ, parental alcoholism, age and a number of childhood psychopathologies. Crucially, the strength of the association between SST performance and subsequent alcohol/substance use was found to be greatest for families with paternal alcoholism.

1.5.3 *Acute alcohol effects on inhibition*

Acute doses of alcohol can lead to temporary inhibitory impairments similar to those observed among chronic alcohol users. These alcohol-induced deficits may be due to the pharmacological effects of alcohol on brain regions associated with inhibition. Supporting this, Tsujii, Sakatani, Nakashima, Igarashi, and Katayama (2011) assessed brain activity during a GNG task following both a placebo and a 0.50g/kg alcohol prime. The findings suggested reduced activity in the right inferior frontal cortex (an area implicated with inhibition, see section 1.3.2), as a result of acute alcohol consumption, to lead to increased errors. Similarly, a more recent fMRI study (Gan et al,

2014) found alcohol-induced impairments of inhibitory control to be related to mitigated responses in the inhibitory-related network, particularly right frontotemporal areas. These areas have been implicated in attentional capture and updating of responses as well as error monitoring, the attenuation of which may lead to impaired responses on inhibitory measures.

A considerable amount of research has been conducted assessing alcohol-induced impairments of inhibitory control. Importantly, early work found alcohol to impair inhibition while reaction times remained unaffected (Mulvihill, Skilling, & Vogel-Sprott, 1997). This and other work suggests inhibitory control to be affected by alcohol at relatively low doses and without the presence of other general motor impairments (e.g. Holloway, 1995). A variety of measures have been used to assess the effect of alcohol on inhibition including; Stroop tasks, SST's, GNG tasks, and anti-saccade tasks.

Regarding the Stroop task, there are inconsistent findings regarding the effect of alcohol on inhibition. With some studies finding an effect at 0.60g/kg (Christiansen, Rose, et al., 2016; Rose & Duka, 2007, 2008) and at an amount designed to achieve a BAC of 0.08% (Curtin & Fairchild, 2003). However, other work has found Stroop performance to not differ following alcohol, relative to placebo (Bombeke, Schouppe, Duthoo, & Notebaert, 2013; Gustafson & Kallmen, 1990).

Regarding the SST, there appears to be a dose-response relationship between alcohol consumption and associated inhibitory impairments. For example, de Wit, Crean, and Richards (2000) did not find a 0.20g/kg dose to increase SSRTs but did find a 0.40g/kg dose to impair inhibition relative to the 0.20g/kg dose and no alcohol. Other research has suggested SSRT's to be greater, relative to placebo, for doses ranging from 0.40g/kg to 0.80g/kg (Caswell, Morgan, & Duka, 2013; Gan et al., 2014; Mulvihill et al., 1997; Reynolds, Richards, & de Wit, 2006).

However, there are a number of inconsistent findings. For example, Caswell et al (2013) found 0.8g/kg of alcohol to increase SSRT's relative to placebo but this was not shown for a 0.4g/kg dose. Furthermore, Loeber and Duka (2009) did not find an effect of alcohol on SSRT's following a 0.80g/kg dose, although this finding was reported as marginal (p=.06). A recent study (Baines et al.,

2019) did find a 0.60g/kg dose of alcohol to impair performance on an SST relative to placebo but found no difference in performance between an alcohol dose and a control drink. This may reflect compensatory effects wherein individuals perform better following a placebo as they are not subject to the impairing effects of alcohol but are attempting to overcome expected impairments (see section 1.5.5).

Furthermore, the variant of SST used may be important with studies employing a GoStop task finding no effect of acute alcohol consumption (Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008; Guillot, Fanning, Bullock, McCloskey, & Berman, 2010; Reed, Levin, & Evans, 2012). Moreover, a study using fixed stop-signals (contrary to an SST with a tracking algorithm to adjust signal presentation) failed to show an effect of alcohol on SSRT's but did find alcohol to lead to increased inhibition errors (Guillot et al., 2010). Saccadic SST's have also been demonstrated to be unimpaired by alcohol while manual SST's are (Campbell, Chambers, Allen, Hedge, & Sumner, 2017), suggesting the effect of alcohol on inhibition to be modality specific. Indeed, there are inconsistent findings regarding the effect of alcohol on the related anti-saccade task with some studies reporting alcohol-induced impairment, others demonstrating improvement, and others suggesting no effect (e.g. Khan, Ford, Timney, & Everling, 2003; Marinkovic, Rickenbacher, Azma, Artsy, & Lee, 2013; Vorstius, Radach, Lang, & Riccardi, 2008).

Furthermore, the effects of alcohol on standard GNG tasks are inconsistent, with some studies suggesting alcohol-induced impairment (Claus & Hendershot, 2015; Rose & Duka, 2008; Tsujii et al., 2011) and others suggesting no impairment (Ortner, MacDonald, & Olmstead, 2003; Reynolds, Richards, et al., 2006). However, the cued GNG task seems consistently affected by alcohol consumption in a dose-response manner. For example, Marczinski and Fillmore (2003) found increased alcohol-induced impairment following a 0.45g/kg dose of alcohol relative to placebo, and following a 0.65g/kg dose relative to 0.45g/kg. Impairment on the cued GNG, relative to placebo, has been found for doses ranging from 0.45g/kg to 0.65g/kg (Fillmore & Rush, 2001; Fillmore, Ostling, Martin, & Kelly, 2009; Marczinski, Abroms, Van Selst, & Fillmore, 2005; Marczinski, Combs, & Fillmore, 2007; Weafer & Fillmore, 2008; Weafer & Fillmore, 2012; Weafer, Fillmore, & Milich,

2009). The effect of alcohol on cued GNG performance appears to be robust with, to my knowledge, only one published study (Christiansen et al, 2013) failing to find an effect of alcohol (0.65g/kg) on cued GNG task performance, relative to both placebo and a control drink. However, this study involved a large battery of cognitive tasks and it is, therefore, possible that inhibitory-impairments may have dissipated. A further issue is that inhibitory control is not directly assessed via cued GNG tasks and is merely inferred from inhibition errors. SST's, therefore, offers a more direct measure of inhibition (SSRT).

A recent paper (Bartholow et al., 2018) highlights a number of important issues with research investigating the acute effects of alcohol on inhibition which may have led to inconsistent results. In particular, the authors highlight issues with studies using only one task, the prevalence of small sample sizes in the literature, the lack of control conditions and a lack of consideration for the biphasic effects of alcohol and how that may affect inhibition. To address this, the authors used a large sample size (N=216) who completed a Stroop, anti-saccade and an SST. Participants completed these measures at baseline and subsequently following alcohol, placebo or a control drink and either during the descending limb of the blood alcohol curve or during both the ascending and descending limbs. The authors report alcohol-induced inhibitory impairments; in the anti-saccade under both ascending and descending limbs, during the descending limb only for the SST and impairment in response accuracy (but not reaction times) on both the ascending and descending limbs on the Stroop.

1.5.4 *Alcohol-induced impairments and the alcohol priming effect*

A number of models (Goldstein & Volkow, 2002, 2011; Jentsch & Taylor, 1999) suggest impaired inhibition, as the result of chronic alcohol use, to lead to increased consumption as there is a reduced ability to inhibit the effect of automatic conditioned responses Given that acute doses of alcohol may lead to transient deficits of inhibition, similar to long-term deficits observed among chronic users, Field et al (2010) suggest that acute alcohol-induced inhibitory impairments lead to a loss of control over drinking (an alcohol priming effect). The authors argue that these impairments and automatic appetitive responses (i.e. attention and approach biases) interact to increase alcohol-

seeking. According to this suggestion, inhibitory control has a top-down effect, enabling individuals to resist automatic responses, however, when inhibition is impaired this may lead to a reduced ability to regulate automatic responses, potentiating alcohol-seeking (see figure 2).

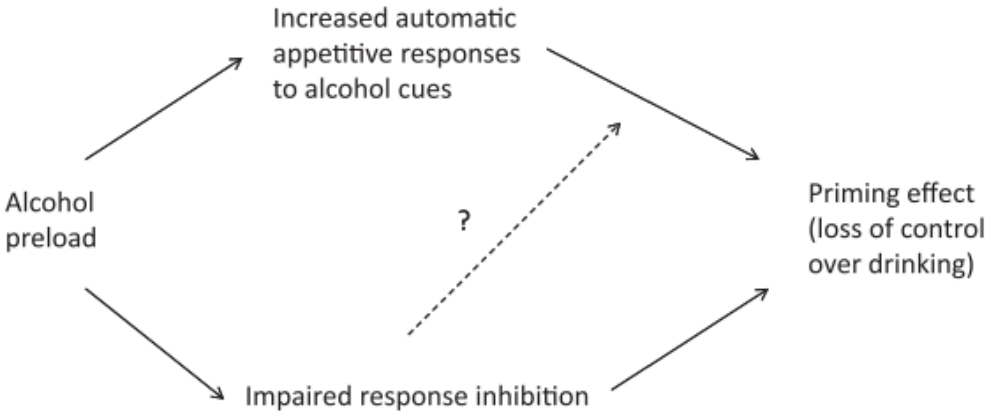

Figure 2. Model of alcohol priming as proposed by Field et al (2010).

Despite this model being widely cited, there is surprisingly little evidence that alcohol-impaired inhibition underlies the alcohol priming effect and to my knowledge, only one study has shown an association between impaired inhibition and alcohol consumption. In this study, Weafer and Fillmore (2008) had participants complete a cued GNG task prior to and following a priming dose of alcohol (0.65g/kg) and, in a separate session, a placebo. Participants returned to the lab for a third session and completed a bogus taste test which assessed their *ad lib* alcohol consumption. The main finding was that the magnitude of the alcohol-induced impairment on the cued GNG task was associated with the amount of alcohol consumed during the bogus taste test, with the impairment accounting for 20% of the variance in *ad lib* alcohol consumption. However, while this study demonstrates an association between alcohol-induced impairments of inhibitory control it does not offer evidence that such impairment mediates the alcohol priming effect *per se*. This is because *ad lib* alcohol consumption was assessed during a separate session from the alcohol prime and cued GNG task. Furthermore, a number of studies which have included priming doses, inhibitory and alcohol-seeking measures within the same session have not found these impairments to mediate alcohol-

seeking or have demonstrated alcohol priming effects in the absence of an effect on inhibitory control (e.g. Christiansen et al., 2013).

There is currently inconclusive research regarding the proposed interaction (Field et al, 2010) between these impairments and automatic processes. Two studies which have used inhibitory control tasks with embedded alcohol images have not found alcohol-induced impairments (at 0.00g/kg, 0.40g/kg and 0.60g/kg doses) of inhibitory control to be affected by the presence of alcohol-related cues (Rose & Duka, 2008; Adams et al, 2013). However, a recent study which used an attentional-bias behavioural activation (ABBA) task which similarly assesses inhibition in the presence of alcohol-related cues (Weafer & Fillmore, 2015) reported greater inhibitory impairments following alcohol (0.65g/kg) in the presence of alcohol-related cues. Moreover, the magnitude of this impairment predicted self-reported number of drinking days but did not predict binge drinking days or the total amount of drinks consumed.

1.5.5 *Anticipated effects, beliefs & motivation*

While much research has focused on the pharmacological effects of alcohol on inhibitory control the anticipated (placebo) effects of alcohol may also be important. Indeed, there is a lack of data which allows investigation of placebo effects, as the majority of studies which have investigated the acute effects of alcohol on inhibition often compare inhibition, following alcohol, to a placebo condition. While this isolates the pharmacological effects of alcohol, it does not allow for analysis of the anticipated effects of alcohol. In order to assess the anticipated effects of alcohol, a placebo condition should be compared to a control condition, wherein participants are provided with a drink which they are aware contains no alcohol. Indeed, the effects of alcohol in the 'real-world' are the result of both the pharmacological and anticipated effects; therefore, comparisons between alcohol conditions and control conditions reflect the most ecologically valid comparison. Ideally, a further condition wherein participants consume alcohol but are led to believe that it is non-alcoholic would also be used (as in Marlatt, Demming, & Reid, 1973) however, this is often impractical given the

unmistakable taste and subjective effects of alcohol; particularly at doses typically administered during priming studies.

Indeed, there is some evidence to suggest that placebo alcohol can lead to a priming effect. This placebo priming effect was first demonstrated by Marlatt et al. (1973) using a fully-balanced placebo-controlled design. In this study, the authors administered a priming dose of alcohol or tonic to alcohol-dependent patients and social drinkers and manipulated whether participants were told they had consumed alcohol or told that they had been given a non-alcoholic drink. It was found that the contents of the drink alone did not affect subsequent *ad lib* drinking but that those who had been told they had consumed alcohol as the priming dose did consume significantly more.

More recent work has also demonstrated this effect. For example, Christiansen et al. (2013) administered a 0.65g/kg dose of alcohol, a placebo drink and a control drink to participants over three sessions. Craving for alcohol was found to be increased following alcohol, relative to both placebo and control conditions, and placebo, relative to the control condition. However, while *ad lib* alcohol consumption was increased following the alcohol prime, the amount of alcohol consumed between placebo and control conditions did not differ. In addition, Rose, Hobbs, and Drummond (2013) found evidence of a placebo priming effect. In this case, craving for alcohol increased over time while an acute dose of alcohol increased craving more rapidly before decreasing. Moreover, placebo doses have been shown to increase both craving and *ad lib* alcohol consumption relative to a control drink (Christiansen et al, 2013; Christiansen, Jennings, & Rose, 2016; Christiansen, Townsend, Knibb, & Field, 2017). Similarly, Leeman, Corbin, and Fromme (2009) found craving following placebo, but not alcohol, administration to predict subsequent *ad lib* drinking. However, this study did not include a control condition so it is not known whether this association is due to the anticipated effects of alcohol or is simply reflective of the association between craving and *ad lib* consumption while participants are not intoxicated.

There is also some evidence to suggest that the anticipated effects of alcohol can lead to disinhibition. Concerning social inhibition, a meta-analysis, which assessed the effect of placebo alcohol on social and behavioural consequences, found placebo alcohol to increase so-called ‘deviant

social behaviours' (Hull & Bond, 1986). Subsequent studies have assessed the effect of placebo alcohol on behavioural measures of inhibition using experimental tasks with mixed results. For example, while Christiansen et al (2013) did not find placebo to impair performance on a cued GNG task, Christiansen et al (2016) found performance on a passive avoidance GNG task to be impaired following placebo alcohol relative to a control drink. Importantly, in this study, the magnitude of these impairments correlated with positive and negative outcome expectancies suggesting beliefs about the acute effects of alcohol to be associated with placebo-induced disinhibition.

Indeed, beliefs about the effects of acute alcohol consumption may be an important determinant of subsequent performance on a number of tasks. In the first study to assess the effect of belief's on task performance, Fillmore, Mulvihill, and Vogel-Sprott (1994) had participants consume either placebo alcohol or placebo caffeine and manipulated the expected effect of these substances, with participants being told that they should expect improved or impaired performance in a pursuit rotor task. Those who had consumed caffeine performed better when they expected improvement relative to those who expected impairment. Interestingly, the opposite was found for the alcohol groups, with participants who expected improved performance fairing worse than those who expected impairment. The authors suggest this finding to be due to a compensatory effect with those who believed they had consumed alcohol performing better due to an attempt to overcome expected impairing effects of alcohol. A similar study from the same research group (Fillmore & Vogel-Sprott, 1994) administered a 0.56g/kg dose of alcohol or a placebo to participants before completing a pursuit rotor task. Overall, alcohol was found to impair performance with those who expected the most impairment performing the worst. Beliefs regarding the acute effects of alcohol have also, importantly, been found to affect performance on an SST. In this study (Fillmore & Blackburn, 2002), participants led to believe that alcohol would impair their reactions on the task subsequently had shorter response latencies and made fewer inhibitory errors (following either alcohol or placebo) than those not led to expect an impairment. However, no effect of belief was found within a no-alcohol condition. Again, these findings are suggestive of a compensatory effect; wherein improved performance is the result of an increased attempt to override the believed impairing effects of alcohol.

Despite these early findings regarding its malleability, inhibitory control has often been treated as an immutable trait. However, in addition to work investigating the effect of alcohol and alcohol-related cues on inhibitory control, recent research has begun to investigate state changes of inhibition due to environmental and motivational factors (review; Jones, Christiansen, Nederkoorn, Houben, & Field, 2013). For example, concerning environmental factors, one study found increased disinhibition in a group of participants who completed an SST which also contained alcohol-related cues while simultaneously being exposed to olfactory alcohol-related cues within a semi-neutral bar laboratory, relative to a control condition (Field & Jones, 2017). Regarding motivational factors, a number of studies have shown performance on inhibitory measures to be affected by instructions provided by researchers. In these studies, participants were either provided with instructions which emphasised the importance of responding to go signals quickly or instructions emphasising the importance of inhibiting during stop trials. Subsequently, those instructed to respond quicker made more inhibitory errors than the other group (Guerrieri, Nederkoorn, Schrooten, Martijn, & Jansen, 2009; Jones, Cole, Goudie, & Field, 2011; Jones, Field, Christiansen, & Stancak, 2013; Jones, Guerrieri, et al., 2011). The authors argue that these instructions may, therefore, induce a disinhibited mindset which may transfer to subsequent behaviour. This is supported by the finding that participants, for whom quicker responding was emphasised, consumed more food and alcohol in subsequent *ad lib* sessions. Given that previous work has found beliefs about the acute effects of alcohol to influence performance on measures of inhibitory control (Fillmore et al, 2001) and motivational factors can affect inhibition and subsequent alcohol consumption (Jones, Cole, et al., 2011; Jones, Guerrieri, et al., 2011) it, therefore, follows that beliefs regarding the acute effects of alcohol may influence the alcohol priming effect.

1.6 Norms and modelling

While the alcohol priming effect may be an important determinant of binge drinking (Field et al, 2010) there are other factors which may also exert an effect on binge drinking behaviour. Indeed, the majority of drinking occasions, and therefore binge drinking episodes, take place in the presence of others (Ally, Lovatt, Meier, Brennan, & Holmes, 2016) and there is a substantial amount of

research suggesting that peer drinking has an effect on individuals drinking behaviour. Having peers who consume alcohol, frequent contact with peers, and consuming alcohol within a social group have all been shown to be associated with elevated alcohol consumption and binge drinking (Creemers, Spanakis, Delforterie, & Huizink, 2017; Eisenberg, Golberstein, & Whitlock, 2014; Elisaus et al., 2018; Kelly et al., 2016; Kuntsche, Otten, & Labhart, 2015; Scholly, Katz, & Kehl, 2014). According to Borsari and Carey (2001), peers may influence drinking directly i.e. through offering drinks, and also indirectly through social modelling and perceived norms. Two types of norms are generally cited as influencing alcohol consumption, descriptive and injunctive norms (Cialdini, Reno, & Kallgren, 1990). In relation to alcohol use, injunctive norms concern perceptions regarding peer acceptance and approval of drinking, while descriptive norms refer to perceptions about the quantity and frequency of alcohol use among peers (Borsari & Carey, 2003).

1.6.1 *Injunctive norms*

Overall, results are mixed regarding the effect of injunctive norms on alcohol consumption. For example, a cross-sectional study found social approval of alcohol to be positively associated with intentions to drink alcohol (Rimal & Real, 2005), importantly this relationship was not moderated by descriptive norms, suggesting injunctive norms exert a distinct effect on intentions to drink. Similarly, Pedersen et al. (2017) found, both descriptive and injunctive norms to be associated with past year and monthly alcohol frequency and peak amount of drinks. However, after controlling for descriptive norms, only injunctive norms were associated with the quantity of alcohol consumed. Conversely, other work has found injunctive norms moderate the association between descriptive norms and drinking behaviour, with the association between descriptive norms and alcohol consumption being greater for those who perceive the peers as being more accepting of their alcohol consumption (Lee, Geisner, Lewis, Neighbors, & Larimer, 2007). The relationship between injunctive norms and alcohol consumption has also been found to be greater for more proximal peers (e.g. friends, family) in comparison to more distal peers (e.g. other university students; LaBrie, Hummer, Neighbors, & Larimer, 2010).

A number of studies have investigated whether the manipulation of injunctive norms can affect the intention to consume alcohol, negative consequences of alcohol use, as well as frequency and volume of alcohol consumption. One recent study (Prince, Maisto, Rice, & Carey, 2015) included the delivery of a brief motivational intervention alongside an (a) injunctive norms intervention, (b) a descriptive norms intervention, (c) a combined injunctive and descriptive norms intervention. A control group which received no intervention was also included and alcohol use and consequences were assessed 4-6 weeks later. Overall, the greatest reduction in alcohol use and negative consequences were found among those who had completed the injunctive and combined descriptive and injunctive interventions. However, other work which has attempted to manipulate injunctive norms has been less successful. For example, (Robinson, Jones, Christiansen, & Field, 2014) used a web-based platform to expose participants to norm messages and assessed the effectiveness of these messages on intentions to consume alcohol. Participants were either exposed to an injunctive norm, a descriptive norm, both injunctive and descriptive norm messages, a health message, or a control message; their intentions to drink responsibly were assessed following this exposure. However, intentions to drink responsibly did not increase following exposure to any of these messages.

1.6.2 *Descriptive norms*

Descriptive norms have been suggested to affect behaviour via a process of informational social influence wherein individuals base their own drinking behaviour on the drinking behaviour they believe their peers engage in (Deutsch & Gerard, 1955). Findings concerning descriptive norms and alcohol consumption are much more consistent (relative to injunctive norms) with an abundance of cross-sectional research reporting a positive relationship between perceived peer alcohol use, personal alcohol use as well as binge drinking (e.g. Borsari & Carey, 2001; Borsari & Carey, 2003; Jones-Webb et al., 1997; Larimer, Turner, Mallett, & Geisner, 2004; Lee et al., 2012; Robinson et al., 2014; Robinson, Jones, Christiansen, & Field, 2015). However, due to the cross-sectional nature of this research, it is not possible to determine if the effect of perceived peer alcohol consumption is causal. It is, therefore, possible that this research reflects selection effects, i.e. individuals select their peers based on their own preferences. Additionally, individuals may base their perceptions of their friend's

alcohol consumption on their own (the false consensus effect; Marks & Miller, 1987). There have been some attempts to clarify this relationship, with recent work demonstrating an association between peer and personal alcohol use even when controlling for previous alcohol use (Leung, Toumbourou, & Hemphill, 2014). However, Guo et al, (2015) found the peer effect to be present only for individuals (college students) who had previously consumed alcohol which would suggest that the peer effect may, at least partly, be explained by selection effects. Indeed, it is likely that both selection and peer alcohol use both contribute to this association (Urberg, Luo, Pilgrim, & Degirmencioglu, 2003). In addition, individuals tend to overestimate their peer's alcohol use (although some claim this tendency has been exaggerated; see Pape, 2012) and greater levels of alcohol use are associated with the magnitude of this overestimation (Haug, Ulbricht, Hanke, Meyer, & John, 2011) suggesting that descriptive norms need not be accurate in order to affect alcohol use.

Several studies have also investigated whether the effect of descriptive norms on drinking is moderated by other factors. For example, the peer effect has been found to be stronger when the drinking of more proximal peers are tested (e.g. using questions pertaining to close friends rather than 'other students'; Cox & Bates, 2011) and among adolescents from dysfunctional families and those who have poor familial relationships (Leung et al., 2014). In addition, the effect of descriptive norms has been shown to be present for individuals low in sensation-seeking and aggression (Grazioli et al., 2018). Similarly, trait measures of self-control have also been found to moderate this relationship, with individuals reporting lower levels of self-control displaying a stronger association between peer and personal alcohol use (Robinson et al., 2015; Wills, Pokhrel, Morehouse, & Fenster, 2011). An additional study, however, did not find a moderating effect of self-control (Visser et al, 2013). However, in this study a sample of Dutch adolescents aged 16+ (which was the legal drinking age at the time) was used which differs from other work (Robinson et al, 2015; Wills et al, 2011) which used a sample of young adults. Finally, Stautz and Cooper (2014b) found urgency (see section 1.4.1), moderated the relationship between peer and personal alcohol (as well as cannabis) use. Indeed, there is evidence to suggest that elevated levels of urgency are associated with increased susceptibility to peer influences (Stautz & Cooper, 2014a). Importantly, for the current thesis, as urgency has been

found to predict alcohol consumption in the lab only when high levels of affect are experienced (Dinc & Cooper, 2015) it is possible that the positive effects (Leeman, Toll, & Volpicelli, 2007) of acute alcohol consumption may further potentiate this effect.

1.6.3 *Modelling*

Peers may also influence alcohol consumption directly via modelling or imitation. Central to this notion is the seminal work of Bandura (1971) who proposed social learning theory (SLT). According to SLT human behaviour is influenced by the behaviour of others from a young age, with individual's observing and modelling others behaviour in an attempt to integrate and become socially adept.

Recent ecologic momentary assessment (EMA) research has directly assessed the effect of peer groups on drinking behaviours within drinking environments. For example, Thrul and Kuntsche (2015) assessed participants alcohol consumption at four time-points throughout the evening and the number of friends present, every Thursday, Friday and Saturday over five weekends using smartphones. Overall, the authors found a positive association between the number of friends present and alcohol consumption (with this effect being greater for males than females). Using a similar method, Labhart, Anderson, and Kuntsche (2017) found that as the number of friends present increased so did the likelihood that participants would drink more than originally intended. Other EMA research suggests that the effect of peer group on drinking may be affected by the group's gender composition. Women consume less alcohol when with men only relative to a mixed-gender group; while men consume more alcohol when in mixed groups, but fewer drinks when in all-female groups (Thrul, Labhart, & Kuntsche, 2017). However, while this research demonstrates associations between peer group compositions and alcohol use, only laboratory research has thus far been able to demonstrate causal effects of social modelling on alcohol consumption.

Experimental laboratory research investigating modelling of alcohol consumption often employs confederates who consume a specified amount of alcohol according to experimental condition. For example, the earliest study to use this method (Caudill & Marlatt, 1975) exposed

participants to a confederate during a ‘wine tasting task’. During the task, the confederate would either drink heavily, lightly, or the confederate would be absent. Subsequently, it was found that participants exposed to the heavy-drinking confederate consumed significantly more alcohol, took more sips and consumed a greater volume of alcohol per sip than those within the light-drinking confederate condition and no confederate condition.

According to a meta-analysis of 13 experiments (Quigley & Collins, 1999), participants consumed significantly more alcohol, produced significantly higher blood alcohol levels, consumed significantly more alcohol per sip and sipped more when exposed to a heavy-drinking confederate, relative to a control condition. The effect size of confederate drinking appears to be large, with greater effects reported when experiments were conducted in natural settings. In addition, the behaviour of the confederate also seemed to influence modelling, with unfriendly confederates having no effect on alcohol consumption.

More recent work has shown that modelling of alcohol consumption occurs regardless of the sex of the confederate. For example, Larsen et al (2010) assessed participants alcohol consumption when exposed to an opposite sex and same-sex confederate who were instructed to drink alcoholic or non-alcohol drinks. Overall, participants were more likely to consume alcohol when the confederate also did, although this was not moderated by the sex of the confederate. Other work has shown modelling of alcohol use in a real bar (Larsen, Overbeek, Granic, & Engels, 2012), regardless of engagement with the confederate (Larsen, Lichtwarck-Aschoff, Kuntsche, Granic, & Engels, 2013), and to occur to the same extent when participants are experiencing stress relative to when they are not (Larsen, Engels, Granic, & Huizink, 2013). Men have also been found to imitate sips more so than women, even when controlling for increased alcohol consumption, and participants imitate sips more when both themselves and the confederate consume alcohol (as opposed to a soft-drink), which may occur due to an increased tendency to monitor others alcohol consumption in an attempt to ingratiate themselves with the confederate or to avoid appearing to be drinking excessively (Larsen, Engels, Souren, Granic, & Overbeek, 2010). Modelling of alcohol consumption may also be affected by ingratiation motives (Robinson et al., 2016) with imitation being more pronounced when participants

believe the confederate will be judging them later and when participants are unsure whether the confederate has accepted them. Therefore, lab research may exaggerate the effect of modelling as participants are often exposed to a confederate who they do not know and with whom they may wish to ingratiate themselves. Importantly, it has been shown that participants also model their friend's alcohol use in the lab (Dallas et al., 2014). In this study, pairs of friends were tested and one member of this pair was covertly provided with instructions to select alcohol or soft-drinks when offered by the experimenter during a task. Participants were found to select and consume significantly more alcoholic drinks when their friend chose to consume alcohol than when their friend chose soft-drinks.

In addition, social contexts have been shown to exert an effect on responses to alcohol. For example, greater liking of alcohol has been reported in social situations vs. isolation, and participants have been shown to be more likely to choose to drink alcohol in social contexts relative to when alone (Doty & de Wit, 1995). Conversely, acute alcohol consumption has been shown to affect response to social situations by potentiating social interaction, bonding, and reducing social anxiety (de Wit & Sayette, 2018; Sayette, 2017; Sayette, Creswell, Dimoff, Fairbairn, Cohn, Heckman et al, 2012). However, there is a lack of research which has accounted for alcohol's acute effects on imitation of alcohol consumption. For example, while considerable research has investigated the effect of alcohol on social processes and the effect of modelling on alcohol consumption, whether modelling occurs to a lesser or greater extent when intoxicated has not yet been investigated. This is important as the majority of binge drinking occurs following alcohol consumption and, therefore, when subject to alcohol's acute effects.

1.7 The current thesis

The current thesis aimed to investigate moderators of the alcohol priming effect. The first aim of the current thesis was to assess the role of two novel moderators of the alcohol priming effect, specifically, whether the alcohol priming effect is moderated by other people's drinking and beliefs about alcohol's acute effects. The second aim of the thesis was to further investigate and clarify the role of alcohol-induced inhibitory-control impairments in the alcohol priming effect.

1.7.1 *Other people's drinking*

The first three experimental chapters focus on whether other people's drinking behaviour moderates the alcohol priming effect. As the majority of binge drinking takes place in the presence of others (Ally et al, 2016), and other people's drinking has been shown to exert a strong effect on personal alcohol consumption (Quigley & Collins, 1999), it is surprising that no study has, to my knowledge, assessed this effect following acute alcohol consumption. This is important to clarify, as while the alcohol priming effect has been suggested to underlie binge drinking, the relative importance of this in the presence of others remains unknown. In study 1 (chapter 3), a cross-sectional design was employed to assess whether descriptive norms regarding alcohol consumption was moderated by self-reported drinking-induced disinhibition. As urgency has been shown to moderate the relationship between peer and personal drinking behaviour (Stautz & Cooper, 2014b), this was also assessed. Furthermore, given that urgency may only affect drinking when high levels of affect are experienced (Dinc & Cooper, 2015), which may be induced following alcohol consumption (Leeman et al, 2009), the interaction between drinking-induced disinhibition and urgency on the peer effect was also assessed.

Study's 2 and 3 (chapters 4 and 5) assessed the effect of peer drinking on the alcohol priming more directly by employing participant's friends as confederates (see Dallas et al, 2014) and instructing them to consume predetermined amounts of alcohol while in the presence of the participant. Specifically, study 2 aimed to assess whether the alcohol-priming effect was mitigated when exposed to a light-drinking confederate. Participants were tested either following alcohol or a placebo and were either tested alone or in the presence of the confederate. Study 3 tested the effects of both light and heavy-drinking confederates on alcohol consumption over two sessions, in one session participants consumed a priming dose of alcohol and consumed a control drink in the other. As trait facets of impulsivity (as assessed in study 1) may be an important moderator of the relationship between peer and personal drinking behaviour, behavioural measures may also be important. Therefore, across these two studies, I also aimed to assess whether the mediating role of alcohol-

induced impairments of inhibitory control on alcohol priming differed (using an SST) due to the presence of a confederate.

1.7.2 *Beliefs about the acute effects of alcohol*

An additional novel potential moderator of the alcohol priming effect is beliefs regarding the effect of acute alcohol consumption. Previous findings have shown inhibitory control to be affected by beliefs regarding alcohol's acute effects (Fillmore et al, 2001). In addition, experimentally induced changes in inhibition (Jones et al, 2011) have been shown to affect alcohol consumption. Therefore, it follows that beliefs regarding alcohol's acute effects may underlie the alcohol priming effect. This was assessed in Study's 4 and 5 (chapter 6; Knibb, Roberts, Robinson, Rose, & Christiansen, 2018). In both studies, beliefs were manipulated by providing false information to participants regarding their ability to regulate their behaviour following an acute dose of alcohol. Participants attended two sessions, receiving an acute dose of alcohol in one session and a placebo in the other. Subsequently, their alcohol consumption was assessed using a bogus taste test.

1.7.3 *Inhibitory control*

The second aim of this thesis was to assess and clarify the indirect effect of alcohol-induced inhibitory impairments on the alcohol priming effect. To do this, an SST was used across four studies (study's 2-5) and then the extent to which alcohol-induced impairments in inhibitory control resulted in increased alcohol consumption was explored. Finally, the data from study's 2-5 were combined to give a more statistically powerful exploration of the indirect effect of an acute dose of alcohol on alcohol consumption via alcohol-induced impairments of inhibitory control. Taken together, this provides a comprehensive test of models that argue impaired inhibition to underpin the alcohol priming effect.

2 Chapter Two: General Methods

Several methods were used repeatedly across the thesis. Those which were employed over more than one study are outlined in detail here, along with information pertaining to their psychometric properties and the rationale for their use.

2.1 Questionnaire measures

2.1.1 *Alcohol use disorders identification test (Appendix 1)*

The Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, Delafuente, & Grant, 1993) was used in each study to assess the hazardous drinking behaviour of participants. Specifically, the measure was developed to identify hazardous drinkers (individuals who may be at risk of experiencing alcohol-related adverse consequences) and harmful drinkers (individuals currently undergoing such consequences; Reinert & Allen, 2002).

The AUDIT consists of 10 fixed-response items which concern alcohol use and consequences of alcohol consumption. The first eight items are four-point scales scored from 0-4 while the final two items are three-point scales scores as 0, 2 or 4. There are a number of cut-offs which have been used to classify individuals according to the extent that their alcohol consumption is problematic (Meneses-Gaya, Zuardi, Loureiro, & Crippa, 2009). However, according to WHO, scores equal to or greater than 8 suggest hazardous or harmful drinking while those scoring 20 or above may require further evaluation for alcohol dependence (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001).

The AUDIT has demonstrated consistently high test-retest reliability and internal consistency (Meneses-Gaya et al., 2009; Reinert & Allen, 2002, 2007). The sensitivity and specificity of the measure have also been shown to be acceptable and similar to other screening tools (Allen, Reinert, & Volk, 2001). Importantly, for the current thesis, the psychometric properties of the AUDIT have also been validated among college students (Kokotailo et al., 2004).

Computerized versions of the AUDIT have similar effectiveness in identifying harmful drinking behaviour as the standard pencil and paper iteration (Butler, Chiauzzi, Bromberg, Budman,

& Buono, 2003). Shortened versions of the AUDIT have also been developed. In study 3, the AUDIT-C is used, (Bush, Kivlahan, McDonell, Fihn, & Bradley, 1998) which consists of the first three items of the full AUDIT which specifically measures consumption rather than alcohol-related problems. As with the full AUDIT, the AUDIT-C has demonstrated effectiveness as a screening tool, high levels of internal consistency, and test-retest reliability (Reinert & Allen, 2007). As with the full AUDIT, the psychometric properties of the AUDIT-C have been validated among college students (Barry, Chaney, Stellefson, & Dodd, 2015).

2.1.2 *Time Line Follow Back (Appendix 2)*

The timeline follow back (TLFB; Sobell & Sobell, 1992a) was completed at baseline for each study to assess alcohol consumption over the previous two weeks, in this thesis UK units were assessed (1 UK unit= 8g of alcohol). The TLFB is a widely used alcohol use measure designed to record retrospective estimations of alcohol use by having individuals recall previous days alcohol consumption and has been used to assess consumption from between one week to twelve months (Buu et al., 2014; Sobell & Sobell, 1992a). However, TLFB assessments over longer intervals may be less accurate (Hoeppner, Stout, Jackson, & Barnett, 2010). Therefore, each study in the current thesis used a TLFB to record retrospective alcohol consumption for the preceding two weeks.

The test-retest reliability of the TLFB has been demonstrated to be high when used with alcohol-dependent patients (Cohen & Vinson, 1995), social drinkers (Hoeppner et al., 2010; Sobell et al., 2001) and when delivered via computer (Sobell, Brown, Leo, & Sobell, 1996).

2.1.3 *Leeds dependence questionnaire (Appendix 3)*

The Leeds dependence questionnaire (LDQ: Raistrick et al., 1994) was administered at the beginning of each study to assess dependency. The LDQ is a brief 10-item clinical measure of dependency which aims to assess psychological dependence. Each of the 10-items reflects aspects of dependency outlined by the International Statistical Classification of Diseases and Related Health Problems- 10th edition (IDC-10; World Health Organization, 1992). Each item is scored 0-3 with total scores ranging from 0-30.

It is notable that the factor structure of the LDQ may be inconsistent. For example, Raistrick et al. (1994), Heather, Raistrick, Tober, Godfrey, and Parrott (2001) and Thomas and McCambridge (2008) propose the measure to consist of a single factor. However, previous research (Lennings, 1999) suggests the measure may consist of two factors ("craving" and "positive reasons for use") while among a sample low in dependency (Hartney et al., 2003) two factors ("drinking ideation" and "achieving and maintaining intoxication") were yielded. More recently, among a clinical sample of young adults, a single factor was identified (Kelly, Magill, Slaymaker, & Kahler, 2010).

Despite inconsistency concerning the factor structure of the LDQ, the psychometric properties of the measure have generally been shown to be good. Indeed, the validation paper outlining the development of the LDQ (Raistrick et al., 1994) describes adequate concurrent, discriminant and group validity. The authors also report the measure to have very high levels of internal consistency (α=.94). This paper validated the LDQ among four samples (alcohol users and opiate users recruited from the Leeds addiction unit, alcohol users recruited via GP's and student alcohol users). Subsequently, the measure has demonstrated good levels of test-retest reliability and internal consistency within a larger clinical sample (Heather et al., 2001), among a clinical sample of young adults (Kelly et al., 2010) and among a population of young social drinkers (Thomas & McCambridge, 2008).

2.1.4 *Drinking induced disinhibition scale (Appendix 4)*

The drinking induced disinhibition scale (DIDS; Leeman et al., 2007) is a nine-item measure which assesses disinhibited behaviour following alcohol. In particular, it measures the extent to which respondents expect behaviours, which may be inhibited in day-to-day life, to become disinhibited following alcohol consumption. The measure assesses three different types of disinhibition (euphoric, dysphoric and sexual) each calculated using three items. While some measures of alcohol expectancies have included subscales assessing disinhibition (e.g. Southwick, Steele, Marlatt, & Lindell, 1981; Wood, Nagoshi, & Dennis, 1992), the DIDS differs in that it is the only measure, to my knowledge, which assesses disinhibited behaviours following alcohol relative to when no alcohol has

been consumed. For example, while completing the scale, respondents are provided with a statement or behaviour (e.g. "Greater feelings of personal freedom than when not drinking") and indicate how likely or unlikely, from 1 (highly unlikely) to 6 (highly likely), they are to experience this following alcohol consumption. Respondents are informed that a score of 1 suggests that this has never occurred while drinking while a score of 6 suggests this to happen on every drinking occasion. This scale was used to assess the extent to which participants experience the disinhibiting effects of alcohol within study's 1, 2 and 3.

In the paper outlining the development of the DIDS (Leeman et al., 2007), the authors state that, across two studies, discriminant and convergent validity were established and report all subscales to have good levels of internal consistency (euphoric study 1 α = .76, study 2 α=.65; dysphoric study 1 α =.80, study 2 α =.81, sexual study 1= .70, study 2 α=.80). Similar levels of internal consistency have also been demonstrated in subsequent research (e.g. Leeman, Toll, Taylor, & Volpicelli, 2009). Moreover, DIDS scores have been found to predict cross-sectional and prospective alcohol-related problems and heavy episodic drinking (Leeman, Toll, et al., 2009).

2.1.5 *Desire for Alcohol Questionnaire (Appendix 5)*

The Desire for Alcohol Questionnaire (DAQ; Love, James, & Willner, 1998) was used to assess craving in all alcohol administration studies. DAQ's were completed on three occasions during each experimental session at baseline; following the administration of a priming drink and prior to measures of alcohol consumption. The DAQ assesses current desires/cravings for alcohol and consists of 14 items scored on 7-point Likert scales. Craving assessed using the DAQ has been shown to be reliably associated with severity of alcohol use disorder (Pasche, Garner, Baldwin, & Sinclair, 2013) and craving following alcohol consumption (Courtney et al., 2013).

The factor structure of the DAQ has been shown to be inconsistent. Originally, the measure was shown to assess four different factors of craving (' negative reinforcement', 'strong desires and intentions', 'mild intentions and positive reinforcement' and 'controllability of alcohol consumption'(Love et al., 1998). However, a number of different three-factor structures have been

identified. For example, Kramer et al. (2010) suggest a three-factor structure consisting of "strong desires and intentions", "negative reinforcement", and "positive reinforcement and ability to control drinking". Conversely, Pasche et al. (2013) proposed that the DAQ consists of "desire to drink" "the ability to control drinking" and "positive and negative reinforcement". Due to inconsistencies in the factor structure of the DAQ, the current thesis used the mean total of the DAQ as the main outcome measure of craving across all studies. The internal consistency of the mean measure has been shown range from adequate to very good (α= .70-.93; Courtney et al., 2013; Kramer et al., 2010; Pasche et al., 2013).

2.1.6 *Subjective intoxication scales (Appendix 6)*

Subjective intoxication scales (SIS; Duka, Tasker, & Stephens, 1998) were used in studies in which alcohol was administered. The SIS was completed on three occasions during each experimental session, at baseline, following administration of a priming drink and prior to measures of alcohol consumption. The SIS consists of six 100mm visual analogue scales (anchored from 'not at all' to 'extremely') developed which were designed to assess changes in subjective states following alcohol consumption. The six subjective feelings assessed include; light-headedness, irritableness, stimulation, alertness, relaxation, and contentedness. Previous research has primarily used the light-headedness scale as an indication that subjective intoxication is achieved following alcohol use (e.g. Christiansen et al., 2017) and therefore this measure is the primary measure of subjective intoxication used throughout the current thesis.

2.2 Behavioural measures

2.2.1 *Bogus taste test*

To assess *ad libitum* alcohol consumption a bogus taste test (Jones et al., 2016; Marlatt et al., 1973) was used in two studies (Study's 4 and 5). While the general procedure of the taste test remained the same the number of drinks provided, and the brands used varied.

The bogus taste test was first developed by Marlatt et al. (1973) as a method to unobtrusively measure the amount of alcohol participants consume. While there are variations, all bogus taste tests

involve presenting alcohol to participants ostensibly to assess taste perception. Participants are given a set time period in order to taste the drink(s) provided to them and complete measures of taste perception (i.e. questionnaires). During this time, they are instructed that they can consume as much or as little of the drink(s) as they wish. The amount of alcohol consumed is then measured.

Since its initial conception, a variety of bogus taste tests have been employed. For example, non-alcoholic beverages being presented alongside the alcoholic drink , in an attempt to control for thirst (e.g. Jones, Cole, Goudie, & Field, 2012) and the use of non-alcoholic alcohol to investigate alcohol consumption in the absence of any pharmacological effect (e.g. Christiansen, Rose, Cole, & Field, 2013; Monk, Qureshi, McNeill, Erskine-Shaw, & Heim, 2017). Importantly, a reanalysis of bogus taste test data from 12 independent studies (Jones et al., 2016) has found alcohol consumption during a bogus taste test to be unaffected by the time of day or the day of the week that the bogus taste test was conducted. In addition, the awareness that alcohol consumption was being monitored did not affect consumption during the test. There was also evidence of construct validity with alcohol consumed during the test being related to sex, craving, alcohol consumption and pleasantness of the drinks.

2.2.2 *Stop-signal task (Appendix 7)*

The current thesis used a version of the SST programmed using Inquisit software (Millisecond Software, 2006) which replicates the STOP-IT program developed by Verbruggen, Logan & Stevens (2008). An SST was selected over other methods of assessing inhibition as this allows the computation of SSRT which provides the most direct measure of inhibition (see section 1.3.1.4).

For every trial a white fixation cross was presented first for 500ms followed by an arrow which pointed right on 50% of occasions and left on 50% of occasions. Participants were required to respond by pressing the appropriate key on the keyboard. For 75% of trials this stimulus was uninterrupted, however, on 25% of trials a stop-signal, in this case, an auditory tone, was presented. On these trials, participants were required to inhibit responding. The task consists of 4-blocks of 64

trials. This version of the SST used a tracking procedure to adjust the delay (SSD) between the 'go' stimuli (arrows) and the stop-signal (tone). For all studies which used an SST, SSRT was used as the main outcome measure of inhibitory control. However, go reaction times (average reaction time for all go trials) and the number of inhibition errors (number of times a response was made on stop trials) is also presented. SSRT was calculated using the integrated method, which has been suggested to provide more accurate estimates (Verbruggen, Chambers, & Logan, 2013). In order to do this, the number of go reaction times was multiplied by the probability of responding at a given delay (*p*[respond|signal]) to give *n*. Go reaction times were then ranked and the *n*th RT was selected. This was repeated per task and the average was calculated.

2.3 Drink administration

All studies, with the exception of study 1, involved the administration of alcohol (based on; Christiansen, Jennings, et al., 2016; Christiansen et al., 2017) and placebo (or a control drink in the case of study 3). Within each administration study, participants were weighed and provided with a drink consistent with their body weight. On occasions when alcohol was administered participants received 0.50 grams of alcohol per kilogram of body weight (0.50g/kg). This dose was selected on the basis of previous research which has demonstrated priming effects and inhibitory impairments at this dose (e.g. de Wit & Chutuape, 1993; de Wit, Crean, & Richards, 2000; Fernie et al., 2012). Moreover, this moderate dose allows participants to consume further alcohol during *ad lib* sessions without exceeding ethical limits, imposed by the University of Liverpool, for alcohol administration. For all studies, the alcoholic drink was vodka (37.5% ABV) mixed with lemonade to a ratio of one-part vodka to three parts lemonade. Within placebo conditions, participants received lemonade of an equal volume. Similar to previous research (e.g. Christiansen, Jennings, et al., 2016; Christiansen et al., 2017) which has used placebo alcohol, an atomizer was used to spray a vodka mist around the rim of the glass and on top of the liquid. The control drink used in study 3 was merely lemonade and participants were made aware that there was no alcohol contained within the drink. Following each drink administration (regardless of whether alcohol, placebo or control was administered) there was an absorption period of ten minutes following which a breathalyzer sample was taken.

3 Chapter 3: Study 1 Peer and personal drinking: Investigating the moderating effects of urgency, self-control, and (affective) drinking-induced disinhibition.

The first study of this thesis aimed to investigate the association between peer and personal alcohol consumption and whether this association was moderated by trait self-control and urgency. In addition, this study investigated the whether these moderations were in turn dependent on the extent to which participants experience drinking-induced disinhibition.

3.1 Abstract

People often drink similar amounts of alcohol as their peers and low levels of behavioural regulation, specifically trait self-control and urgency, have been shown to moderate this peer effect. Critically, acute alcohol consumption can impair behavioural regulation, leading to disinhibited behaviour. However, no study has investigated the effect of self-report drinking-induced disinhibition on the peer effect. The current study investigated the extent to which the association between peer and personal drinking is moderated by self-control and urgency. In addition, whether this is in turn moderated by general, and affective, drinking-induced disinhibition was explored. Two-hundred and ten participants completed an online study. Self-reported drinking-induced disinhibition, trait self-control, urgency, peer and personal drinking were measured. Trait self-control was not found to moderate the peer effect. However, the relationship between peer and personal drinking was moderated by urgency, although this occurred only when affective drinking-induced disinhibition was low. This suggests that individuals high in urgency may be more likely to drink like their peers although this only occurs if they also have low levels of affective drinking-induced disinhibition.

3.2 Introduction

Perceived peer alcohol use has been consistently shown to be associated with personal alcohol use (e.g. Jones-Webb et al., 1997; Larimer, Turner, Mallett, & Geisner, 2004; Lee et al., 2012; Robinson, Jones, Christiansen, & Field, 2014b) and research suggests this relationship is causal rather than being the result of peer selection (Cruz, Emery, & Turkheimer, 2012). One explanation for this association may be that individuals use their perceptions of how their peers drink (i.e. 'descriptive norms') as an indication of how they should drink themselves (Cialdini, Reno, & Kallgren, 1990; Deutsch & Gerard, 1955; Rimal, Lapinski, Cook, & Real, 2005). Furthermore, experimental research suggests that people imitate the drinking behaviour of both previously unknown confederates (Larsen, Engels, Granic, & Overbeek, 2009; Larsen, Engels, Souren, Granic, & Overbeek, 2010; Quigley & Collins, 1999; Robinson et al., 2016) as well as friends (Dallas et al., 2014).

Although the effect of peer drinking is robust, a significant amount of cross-sectional research suggests that individual differences in behavioural regulation may moderate the extent to which people drink like their peers (Robinson et al., 2015; Stautz & Cooper, 2014b). Self-control (the ability to successfully regulate behaviours, thoughts, and emotions; de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2011), is consistently associated with problematic alcohol use (Costello, Anderson, & Stein, 2014) and implicated in the peer drinking effect (Robinson et al., 2015). Previous research has shown self-control moderates the relationship between peer and personal drinking among adolescents under legal drinking age, with individuals with lower self-control drinking more similar to their peers than those with higher levels of self-control (Wills et al, 2011). Conversely, this was not found among a sample of Dutch adolescents (Visser, de Winter, Veenstra, Verhulst, & Reijneveld, 2013) of legal drinking age (mean age=16.27± 0.73) - legal drinking age in the Netherlands being 16 at the time of the study (Monshouwer, Smit, de Zwart, Spruit, & van Ameijden, 2003). However, the moderating effect of self-control may be more apparent among those who are more frequently exposed to social drinking contexts. In particular, alcohol consumption has been shown to increase between the ages of 18-25 (Arnett, 2005) and this occurs regardless of university/college attendance (Bingham, Shope, & Tang, 2005). Consistent with this, Robinson et al.

(2015) found self-control to moderate the relationship between peer and personal drinking behaviours among a sample of UK university students (mean age 21.70 ± 4.50), such that lower levels of self-control were associated with personal drinking behaviour more similar to that of peers.

This research has, however, neglected to consider the potential consequence of alcohol's acute effects on the extent to which individuals drink similar to their peers. This is particularly important given that the majority of alcohol during a heavy drinking session will be consumed while subject to alcohol's effects. Indeed, it is well documented that acute doses of alcohol can lead to a loss of control over drinking resulting in increased alcohol craving and further drinking (e.g. Christiansen et al., 2013; de Wit, 1996; Rose et al., 2014). This 'alcohol priming effect' may, at least in part, be the product of alcohol-induced disinhibition as acute doses can lead to deficits in the neuro-cognitive substrates of self-regulation (e.g. de Wit et al., 2000; Field et al., 2010) and the magnitude of this impairment may be related to subsequent alcohol consumption (Weafer & Fillmore, 2008). Therefore, given that poor self-regulation may strengthen the peer effect, the effect of alcohol on the ability to self-regulate should moderate the relationship between peer and personal drinking. For example, while an individual may have relatively high trait self-control (and resist the urge to commence drinking when their peers do), following an initial drink their ability to self-regulate may be impaired, making them more likely to replicate the heavy drinking of their peers.

Another trait which may determine the strength of the peer effect is urgency. Urgency is considered to be a facet of impulsivity and refers to the tendency to act impulsively dependent on mood (i.e. mood- based rash action; Cyders and Smith, 2007). Urgency is composed of two factors; with positive urgency being the tendency to act impulsively when in a positive mood and negative urgency referring to the tendency to act impulsively when in a negative mood. Both facets of urgency are associated with greater alcohol consumption, problematic alcohol use (Coskunpinar, Dir, & Cyders, 2013; Stautz & Cooper, 2013) and, critically, a reduced ability to resist peer influence (Stautz & Cooper, 2014a). Indeed, among adolescents, positive and negative urgency have been shown to moderate the relationship between peer and personal alcohol use (Stautz & Cooper, 2014b); with greater positive and negative urgency being associated with a tendency to drink more like peers.

Stautz and Cooper (2015) argued the tendency to be guided by affective stimuli may be potentiated among individuals with high levels of urgency, although this may only be true for adolescents. Importantly, the association between urgency and alcohol consumption may be dependent upon affect. For example, the relationship between positive urgency and alcohol use has been found to be apparent only when a positive mood state is experienced (Dinc & Cooper, 2015). In addition, positive urgency has been found to moderate the relationship between positive affect and drinking to intoxication, while negative urgency has been shown to moderate the relationship between negative affect and drinking to intoxication (Bold et al., 2017).

As well as leading to a loss of control over drinking, previous work has suggested that acute alcohol consumption can lead to euphoric and dysphoric disinhibition (Leeman, Toll, et al., 2009; Leeman, Toll, & Volpicelli, 2007) wherein individuals are more likely to experience and express positive and negative emotional states. Therefore, given that previous work shows affect to moderate the relationship between urgency and alcohol consumption (Bold et al., 2017; Dinc & Cooper, 2015) and the tendency to imitate peers (Stautz & Cooper, 2014a; Stautz & Cooper, 2015), individuals high in urgency may drink more similar to peers if they are sensitive to the affective disinhibiting effects of alcohol.

The current study investigated the relationship between peer and personal alcohol use and the extent to which this relationship is moderated by trait self-control and urgency. Whether the influence of these moderators was dependent on affective and overall drinking-induced disinhibition was also assessed. A sample of young adult social drinkers (aged 18-25) was recruited. Participant's perceptions of their friends drinking (rather than peers) was measured as young adults have been shown to report drinking behaviour more similar to their friends than other more distal groups (Cox & Bates, 2011). As drinking-induced disinhibition is dependent on experiencing the acute effects of alcohol the relationship between peer and personal drinking quantity (i.e. the amount of alcohol consumed in a typical drinking day) was assessed along with, trait self-control and urgency. The drinking-induced disinhibition scale (DIDS; Leeman et al., 2007), which assesses euphoric and dysphoric disinhibition following alcohol, was also administered. As the effect of urgency on drinking may be dependent on affect (Dinc &

Cooper, 2015), an overall affective drinking-induced disinhibition scale was created by combining euphoric and dysphoric subscales, mean overall drinking-induced disinhibition scores were also calculated.

It was predicted that; the peer effect would be moderated by trait self-control so that the association between peer and personal drinking would be stronger when self-control was low and that this would only occur when overall drinking-induced disinhibition was high. In addition, it was expected that the peer effect would be stronger when urgency was high this would only be apparent when affective drinking-induced disinhibition was high.

3.3 Method

3.3.1 *Participants*

Two-hundred and ten participants (44 male) with a mean age of 21.44(±2.56) completed an online survey. Participants were required to be aged 18-25 and fluent in English. Recruitment took place via university intranet, social media, and poster advertisements. Participants were entered into a prize draw for their participation. The study was approved by the University of Liverpool Research Ethics Committee. Although previous work (Robinson et al., 2015), only found a small moderating effect of self-control the effect in this study was expected to be greater as questions which concerned more proximal peers were used. We, therefore, conducted a power calculation (α=.05, 95% power), using G*POWER (Faul, Erdfelder, Lang, & Buchner, 2007), to detect a medium effect size (f^2=0.15) for a hierarchical regression with five tested predictors and nine total predictors. A sample size of 138 was recommended, although more participants were recruited to account for responses which did not meet the age criteria, participants who did not complete all items and did not take due diligence when answering.

3.3.2 *Design*

This study used a cross-sectional correlational design to assess the association between peer and personal drinking. In addition, the moderating effects of urgency, self-control and drinking-induced disinhibition on this association were also assessed.

3.3.3 *Materials*

3.3.3.1 Leeds Dependence Questionnaire (LDQ)

The Leeds dependence questionnaire (Raistrick et al., 1994) is a diagnostic tool used to assess severity of dependency regardless of substance. It consists of 10 items scored from 0(never) to 3(always). LDQ scores are calculated as the sum across all items. Higher total scores suggest more severe dependency; with scores below 10 indicative of low dependence, scores from 10-22 suggesting medium dependence and, scores above 22 suggesting high dependence. Internal reliability for the current study was acceptable (α=.84; all subsequent alphas reflect the current data also).

3.3.3.2 Time Line Follow-Back (TLFB)

Previous two-week alcohol consumption was measured using the TLFB questionnaire (Sobell & Sobell, 1992b). Participants were required to retrospectively record the amount of alcohol, in UK alcohol units (1 UK unit= 8g of alcohol) that they had consumed over the previous two weeks. Participants were presented with information regarding the alcohol content of a number of drinks to aid completion.

3.3.3.3 Alcohol Use Disorder Identification Test (AUDIT; typical drinking quantity)

The AUDIT (Saunders et al., 1993) is a diagnostic tool used to identify harmful drinking patterns. The first eight items are scored on 5-point scales (from 0-5). The final two items consist of 3-points (scored as 0, 2 or 4). Scores range from 0-40 with scores above 8 indicative of hazardous drinking patterns. To test the association between peer and personal drinking on occasions when more than one alcoholic drink would be consumed, the second item of the AUDIT "How many drinks containing alcohol do you have on a typical day when you're drinking?" was used as a measure of typical drinking quantity (α=.85).

3.3.3.4 AUDIT-C (peer drinking)

An adapted version of the AUDIT-C *(Bush et al., 1998)* was used to assess perceptions of friends drinking. The AUDIT-C consists of the first three questions of the full AUDIT and in this case, the wording of the questions were altered to pertain to friends drinking e.g. "How often do you have a drink containing alcohol?" became "How often do your friends have a drink containing alcohol?". As with the AUDIT, "How many drinks containing alcohol do your friends have on a typical day when you're drinking?" was used as the measure of drinking quantity (α=.63).

3.3.3.5 Brief Self-control scale (SCS)

The brief self-control scale consists of 13-items scored from 1(not at all) to 5(very much). Scores can range from 13-65. The scale purports to measure the ability of individuals to resist temptation and control their behaviour. The scale has previously been shown to have good reliability, construct and predictive validity (Tangney, Baumeister, & Boone, 2004). However, the reliability of this scale in the current study is below acceptable levels (α =.41). The total score across all items was used as the primary measure of self-control.

3.3.3.6 SUPPS-P (Appendix 8)

The short version of the UPPS-P (α=.63) Impulsive Behaviour Scale (Cyders, Littlefield, Coffey, & Karyadi, 2014) consists of 20 items and measures 5 factors of impulsivity; lack of perseverance (α=.68), lack of premeditation (α=.81), sensation seeking (α=.73) & negative (α=.77) and positive urgency (α=.78). Items are presented as 4-points scales from 1(strongly agree) to 4 (strongly disagree). Mean scores for each factor were calculated. Previous findings suggest that the two urgency facets add little unique variance to indices of substance use (Stautz et al., 2017) both of these facets were combined into a single urgency factor (α=.83).

3.3.3.7 Drinking- induced disinhibition scale (DIDS)

The DIDS (Leeman et al., 2007) measures the extent to which individuals expect disinhibition to occur more so when drinking than when not drinking. Three types of disinhibition are assessed by

the scale; euphoric (α=.69), dysphoric (α=.75) and sexual (α=.86). The scale consists of nine items with three items for each category of disinhibition. Participants are provided with a statement or behaviour (e.g. "Greater feelings of personal freedom than when not drinking") and are asked to indicate how likely or unlikely from 1 (highly unlikely) to 6 (highly likely) they are to experience such an occurrence following alcohol consumption. Participants were told that a score of 1 would suggest such a behaviour or feeling has never occurred while a score of 6 would suggest a behaviour or feeling which occurs every time they drink. Mean scores for each of the three categories of disinhibition were calculated. An overall mean drinking-induced disinhibition measure was also calculated by calculating the mean across all items (α=.79). A single affective drinking-induced disinhibition factor was also created by combining euphoric and dysphoric disinhibition scales (α=.74), to reflect the combined urgency factor of the UPPS-P.

3.3.3.8 Awareness (Appendix 9)

Awareness was assessed using a single item open question: 'What do you believe the aims of the study to be?'

3.3.4 *Procedure*

Qualtrics was used to administer the questionnaires online. Upon accessing the online site participants provided informed consent. They were then asked to declare their intentions to answer diligently and were informed that several checks were in place throughout the questionnaire and that the quality of responses will be reviewed. They then provided basic demographic information (sex and age) before completing all the measures in a randomized order. Throughout the questionnaire, there were several checks to ensure that participants answered correctly wherein the 'question' informed the participant which response to select. At the end of the survey participants completed the awareness measure, were debriefed and thanked for their participation.

3.3.5 *Data reduction and analysis*

Data for this study is openly available on the Open Science Framework (https://osf.io/s5r3e/). Overall two-hundred and twenty participants completed the questionnaire. However, after removal of those who were aged over 25 (n=9) and who had not passed the checks (n=1) two hundred and ten participants remained. The data was analysed with two hierarchical regressions. The first model tested whether the relationship between peer and personal typical drinking quantity was moderated by trait self-control (SCS) and whether this is in turn moderated by general drinking-induced disinhibition (means DIDS). The second model tested whether the relationship between peer and personal typical drinking quantity was moderated by urgency and if this was, in turn, moderated by affective drinking-inducted disinhibition. All variables were standardized (z-scores) prior to analysis and computing interaction terms. Both models controlled for age and sex in the first step. The second step of each model contained the variables of interest and the third step contained all first-order interaction terms. The final step of each model contained the second-order interaction term.

3.4 Results

Correlations between variables and descriptive statistics are presented in table 1. Mean AUDIT scores were above 8 suggesting a preponderance of hazardous drinkers within the sample while scores on the LDQ on average were below 10 suggesting low levels of dependency. The mean amount of alcohol units consumed over the previous two weeks was 25.08 (±30.17).

3.4.1 *Self-control and drinking-induced disinhibition*

Overall the trait self-control and mean drinking-induced disinhibition model predicted approximately 50% of the variance in personal typical drinking quantity, R^2=.52, ΔR2= .50, F (9, 200) = 23.72, p<.001. Age and sex accounted for approximately 4% of variance but neither were directly associated with drinking quantity. The second step accounted for 47% of variance with lower self-control, greater drinking-induced disinhibition and peer drinking quantity being associated with increased personal drinking quantity. Step 3 and 4 each accounted for approximately 1% of the variance; none of the first or second-order interaction terms were significant (see table 2).

3.4.2 *Urgency and affective drinking-induced disinhibition*

The urgency and affective drinking-induced disinhibition model predicted approximately 52% of the variance in personal drinking quantity, R^2=.54, ΔR^2=.52, F (9, 200) = 25.67, p<.001. Step 1 was the same as previously described; step 2 predicted approximately 46% of the variance with greater urgency and greater peer drinking quantity being associated with greater personal drinking quantity although affective drinking-induced disinhibition was not associated with drinking quantity. Step 3 accounted for an additional 1% of the variance but no first-order interaction terms were significant. Step 4 explained 3% of the variance in personal drinking and the second-order interaction, urgency x affective drinking-induced disinhibition x peer drinking quantity, was significant. See table 2.

3.4.3 *Interaction between urgency, affective drinking-induced disinhibition and typical peer drinking quantity*

A simple slope analysis (Aiken, West, & Reno, 1991) revealed the relationship between peer and personal drinking quantity to be significant regardless of differing levels of urgency and affective drinking-induced disinhibition (all *p's*<.001). However, a slope difference test (Dawson & Richter, 2006) revealed the strength of the association between peer and personal drinking quantity differed depending on levels of urgency and affective drinking-induced disinhibition.

These significant differences were carried by those reporting low levels of affective disinhibition (-1 SD) in combination with high urgency (+1 SD) having a significantly stronger association between peer and personal drinking (slope 2, see figure 3; β= .94) than those with high affective disinhibition/high urgency (slope 1; β=.48) , t (209) = 4.16, p<.001, and those with low affective disinhibition/low urgency (slope 4; β=.55), t(209)= 2.77, p=.006. However, there was no difference between those with low affective disinhibition in combination with high urgency and those with high affective disinhibition/low urgency (slope 3; β=.70), t (209) = 1.36, p=.174. No other slopes significantly differed from one another, p's≥.135. See figure 3.

These findings suggest that individuals with low levels of drinking-induced disinhibition but high levels of urgency drink quantities of alcohol more like their friends than those with high urgency/

high disinhibition and those with low urgency/low disinhibition. However, the peer effect was similar for these individuals and those with high disinhibition/low urgency.

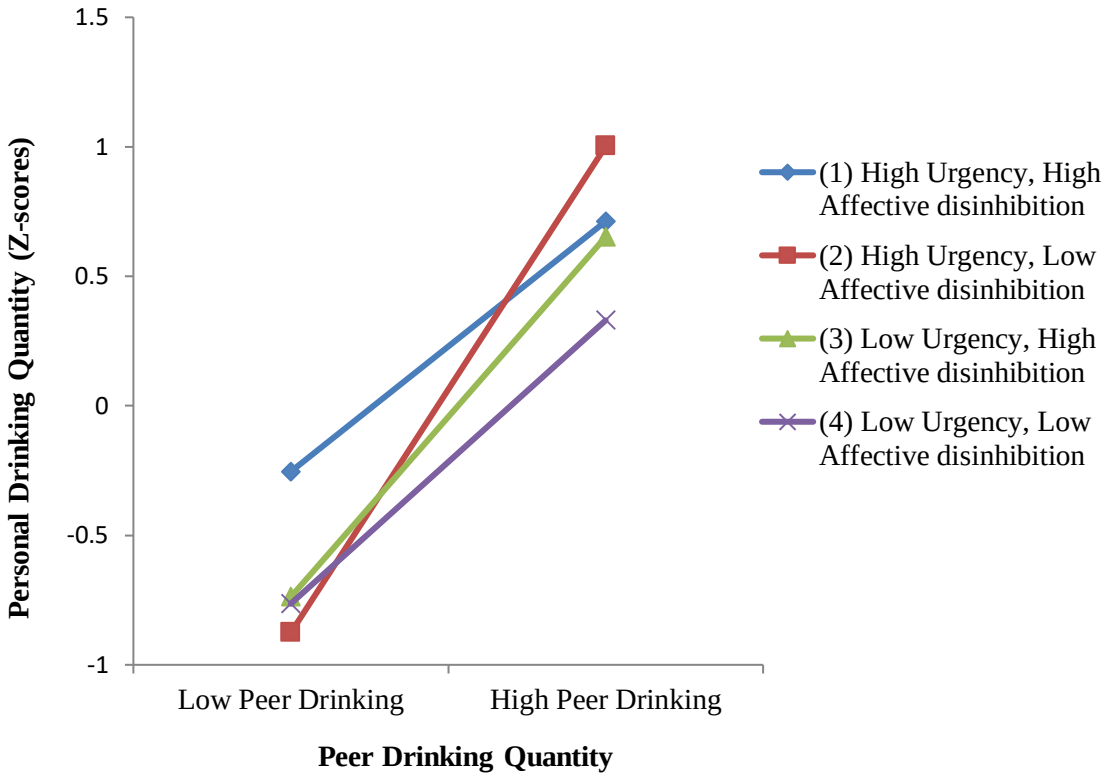

Figure 3. Slopes for relationship between peer drinking quantity- personal quantity across high and low levels of urgency and affective disinhibition. High levels +1 SD, low levels -1 SD. Standardized values (z-scores) presented.

Table 1. Study 1 descriptive statistics (values mean ±SD) and Pearson's correlations

	Mean (±SD)	1	2	3	4	5	6	7	8	9	10	11	12
1.Sex	-	-											
2.Age	21.44 (±2.56)	-.02	-										
3. DID	3.07 (±0.94)	-.05	-.04	-									
4.Affective DID	3.31 (±0.94)	.02	-.09	.87***	-								
5. Urgency	2.24 (±0.59)	.09	-.16*	.48***	.43***								
6. SCS	39.36 (±9.22)	<.00	.17*	-.37***	-.39***	-.61***	-						
7. AUDIT	8.89 (±5.59)	-.10	-.25***	.39***	.31***	.38***	-.44***	-					
8. Personal typical drinking quantity	1.50 (±1.17)	-.12	-.16*	.31***	.23***	.31***	-.30***	.70***	-				
9. Peer typical drinking quantity	1.98 (±1.04)	-.10	-.10	.22**	.124	.24***	-.18**	.49***	.67***	-			
10. Units consumed	25.08 (±30.17)	-.19***	-.15*	.23**	.17*	.21**	-.31***	.66***	.47***	-.31**	-		
11. LDQ	4.32 (±4.23)	-.06	-.14*	.43***	.38***	.46***	-.45***	.61***	.45***	.26***	.45***	-	
12. Peer AUDC	9.77 (±2.08)	-.17*	-.20**	.27***.	.17*	.23**	-.23***	.62***	.55***	.76***	.50***	.33***	-

Notes. DID= mean scores on DIDS, Affective DID= Combined dysphoric and euphoric subscale of DIDS (all scored from 1 to 6). Urgency= combined positive and negative urgency subscale of SUPPS-P. SCS= total SCS scores (scores can range from 13 to 65). AUDIT= total AUDIT scores. Personal typical drinking quantity= AUDIT item 2. Peer typical drinking quantity= adapted AUDIT-C item 2. Units consumed= amount of alcohol in UK units (1unit= 8g alcohol), retrospectively over two weeks. Peer AUDC= Total peer AUDIT-C scores. LDQ= Leeds dependence questionnaire (scored 0-30; with higher scores indicative of greater dependency). Sex coded as 1= male, 2= female. . *p<.05, **p<.01, *** p<.001.

Table 2. Study 1 hierarchical regression analyses for relationship between peer and personal typical drinking quantity

Predictors	Cumulative		Simultaneous		
	R^2-Change	F-Change	β	P	95% CI
SCS & mean drinking-induced Disinhibition					
Step 1	.04	4.30*			
Sex			-.05	.287	[-.15,.05]
Age			-.07	.153	[-.17, .03]
Step 2	.47	63.98***			
DID			.13	.022	[.02, .23]
SCS			-.14	.013	[-.25, -.03]
Peer quantity			.64	<.001	[.53, .75]
Step 3	.01	.656			
SCS x Peer quantity			-.03	.593	[-.12, .07]
DID x Peer quantity			-.05	.315	[-.15, .05]
DID x SCS			-.07	.208	[-.16, .04]
Step 4	.01	2.60			
SCS x DID x Peer quantity			.09	.109	[-.02, .18]
Urgency & affective drinking-induced disinhibition					
Step 1	.04	4.30*			
Sex			-.06	.169	[-.17, .03]
Age			-.05	.237	[-.16, .04]
Step 2	.46	61.75***			
Urgency			.14	.016	[.03, .25]
Affective DID			.09	.137	[-.03, .20]
Peer quantity			.67	<.001	[.56, .77]
Step 3	.01	1.97			
Urgency x Peer quantity			.04	.399	[-.06, .15]
Affective DID x Peer quantity			-.08	.128	[-.18, .02]
Affective DID x Urgency			-.05	.964	[-.09, -.09]
Step 4	.02	10.66**			
Affective DID x Urgency x Peer quantity			-.18	.001	[-.24, -.06]

Notes: Peer quantity= Peer typical drinking quantity, item 2 on adapted AUDIT-C. DIDS= Mean scores on drinking-induced disinhibition scale, urgency= mean score of positive and negative subscale of the SUPPS-P, affective DID= affective drinking-induced disinhibition (mean score of euphoric and dysphoric disinhibition subscale of DIDS) * p<.05, **p<.01, *** p<.001.

3.5 **Discussion**

This study investigated whether the relationship between perceived peer drinking quantity and personal drinking quantity was moderated by trait self-control and urgency. Whether these effects was further moderated by drinking-induced disinhibition was also assessed. Overall, lower trait self-control, higher overall drinking-induced disinhibition, and urgency were directly associated with greater alcohol consumption. It was also found the amount of alcohol peers consumed to be associated with personal alcohol consumption. No moderating effect of self-control or overall drinking-induced disinhibition was found. However, urgency did moderate the peer effect and this was dependent on affective drinking-induced disinhibition. Specifically, those higher in urgency drank more similar to their peers but only when affective drinking-induced disinhibition was low. Although there was no difference between those low in urgency and high in affective drinking-induced disinhibition and any other group.

The current findings support previous work suggesting that urgency moderates the relationship between peer and personal drinking (Stautz & Cooper, 2014b). It also supports research suggesting greater urgency to be associated with a reduced ability to resist peer influence (Stautz & Cooper, 2014a). One explanation for this is that individuals with high urgency are more likely to employ an 'affect heuristic' when making decisions i.e. basing their decisions on the perceived valence of those decisions. They may, therefore, be more likely to adhere to descriptive norms and/or match the drinking behaviour of present friends as this information is positively valenced. Furthermore, it is argued that individuals high in urgency may also be more likely to consider that their substance use/drinking is accepted by peers (Stautz & Cooper, 2014a).

In addition, this study suggests that the influence of urgency on the peer effect is only apparent when affective drinking-induced disinhibition is low. This is contrary to our hypothesis that urgency would moderate the peer effect only when affective drinking-induced disinhibition was high. One explanation for this is that high-urgency individuals, who are more likely to employ an affect heuristic (Stautz & Cooper, 2014a), who also experience the affective disinhibiting effects of alcohol may attribute greater valence to alcohol than that attributed to social cues. This may serve to disrupt

the valence attributed to social cues. These individuals may be more likely to experience an alcohol priming effect wherein acute alcohol consumption increases further use (Christiansen et al., 2013; De Wit, 1996; Rose et al., 2014), regardless of the amount their peers consume. This is consistent with the notion that drinking-induced disinhibition underlies the alcohol priming effect (de Wit et al. 2000; Field et al. 2010). Conversely, those high in urgency and low in affective disinhibition may be less likely to experience a priming effect and attribute valence onto social cues, subsequently drinking similar to their friends.

Interestingly, the magnitude of the peer effect was similar for those with high urgency/low affective disinhibition and those with low urgency/high affective disinhibition. Indeed the latter individuals, being low in urgency traits, may be less inclined to employ an affect heuristic. However, as they experience the disinhibiting effects of alcohol, and their ability to self-regulate is impaired, these individuals may be more likely to employ descriptive norms and so drink similar to their peers (Robinson et al, 2015). This may explain why these individuals did not differ from participants with any other combination of urgency traits. Future research should aim to further disentangle this complex relationship between urgency, affective drinking-induced disinhibition and peer drinking. In addition, the effect of alcohol administration and urgency on modelling of alcohol consumption under lab conditions and the effect of acute intoxication on the valence of alcohol among high urgency individuals should be assessed.

The current results do not support previous research which suggests trait self-control to moderate the peer effect (e.g. Robinson et al., 2015; Wills et al., 2011). However, these previous studies used sample sizes much greater than the current study and, although results are significant, effect sizes are small. While the effect size was expected to be greater in this study, as the relationship between personal drinking and drinking of people's friends was assessed, the study may have been have been underpowered to detect a moderating effect of self-control. Alternatively, self-control may not moderate the relationship between personal and friends drinking but may moderate the relationship between personal drinking and the perceived drinking of more distal reference groups (i.e. 'other students', see Robinson et al. 2015).

There are a number of additional limitations of the current study. Firstly, this study was cross-sectional and so no conclusions can be made regarding causal relationships. Furthermore, a sample of 18-25 year olds were recruited in order to gain a sample of participants who are highly likely to be exposed to social drinking contexts, but this limits the generalisability of our findings. Finally, acute alcohol effects are biphasic exerting a stimulant effect during the ascending limb of the blood alcohol curve and a sedative effect on the descending limb (Sutker, Tabakoff, Goist, & Randall, 1983). While the DIDS is a useful scale, and is the only measure, to our knowledge, which assesses disinhibition when intoxicated relative to when sober, it may pertain more to the propensity to experience alcohol's biphasic effects rather than disinhibition *per se*. Future research should aim to use measures which specifically address the disinhibiting effects of alcohol or use direct neurocognitive measures of disinhibition (e.g. stop-signal task; Logan & Cowan, 1984) and also investigate whether the biphasic effects of alcohol moderate the relationship between peer and personal drinking.

In summary, the current findings suggest that urgency moderates the relationship between peer and personal typical drinking quantity and that this occurs when affective drinking-induced disinhibition is low. This indicates that those high in urgency traits but who are less susceptible to the affective disinhibiting effects of alcohol may drink more similar to their friends. In addition, individuals low in urgency may be more likely to employ descriptive norms when experiencing the disinhibiting effects of alcohol.

4 Chapter 4: Study 2 The effect of a light-drinking confederate on the alcohol priming effect

The previous study demonstrated that the association between peer and personal drinking behaviour is moderated by both trait factors of impulsivity (specifically urgency) and drinking-induced disinhibition. However, the previous study was cross-sectional in nature and it is therefore not possible to infer causation. In addition, the previous study used a self-report measure of drinking-induced disinhibition which may reflect the biphasic effects of alcohol rather than disinhibition *per se*. The current study, therefore, employed an experimental design so that causation can be inferred and used a cognitive task which explicitly assesses neurocognitive mechanisms of disinhibition (the SST). Specifically, given the previous study's findings, the current study investigated whether the alcohol priming effect can be mitigated by exposure to a light-drinking confederate. This study also aimed to investigate whether imitation of alcohol consumption is underwritten by alcohol-induced impairments of inhibitory control.

4.1 Abstract

Participants have been shown to imitate the amount of alcohol an unfamiliar confederate consumes; suggesting peer drinking has a strong effect on personal alcohol consumption. However, no study has investigated the effect of an acute dose of alcohol on imitation of alcohol consumption. This is potentially important as acute doses of alcohol can prime alcohol-seeking behaviour leading to increased alcohol consumption. As the effect of peer drinking appears to be strong, exposure to light-drinking peers may mitigate this alcohol priming effect. To investigate this, participants (N=129) were given a priming dose of alcohol or a placebo and completed an *ad lib* drinking session. This occurred either with a friend who had been allocated to be a light-drinking confederate or in isolation. Overall, participants reported greater increases in craving within the alcohol condition suggesting the presence of a priming effect. However, there was no effect of confederate condition or drink type on the amount of alcohol consumed and additional measures of alcohol-seeking. Exploratory analysis of sex differences revealed males consumed significantly more alcohol and ordered more drinks, when in the presence of a confederate relative to those in isolation. This suggests male alcohol consumption to be elevated when in the presence of a friend.

4.2 Introduction

A substantial amount of research suggests that individuals imitate the drinking behaviour of their peers during drinking sessions. To assess this, previous research has predominately used confederate paradigms wherein participants are exposed to a confederate that they are unfamiliar with and who is trained to consume a predetermined amount of alcohol. A meta-analysis of 13 studies using this method (Quigley & Collins, 1999) found participants drank more alcohol, had higher blood alcohol levels, took more sips and consumed a greater volume of alcohol per sip when exposed to a heavy-drinking confederate relative to a control condition (either a low-drinking confederate or a no-confederate condition). More recent research has found this effect to persist regardless of engagement with the confederate and levels of stress, and has been replicated in real bars (Larsen, Engel, et al 2013; Larsen Lichtwarck-Aschoff et al, 2013; Larsen et al, 2012, 2010).

Previous confederate paradigms have a number of issues. For example, they are unlikely to reflect 'natural' drinking occasions since people usually drink with friends (not strangers; Ally et al, 2016). Indeed, imitation of alcohol consumption has been argued to increase when participants have a need to ingratiate themselves (Robinson et al, 2016), which may be enhanced in the presence of a stranger. To my knowledge, only one study has attempted to address this by recruiting pairs of friends and secretly enlisting one member of the pair to act as a confederate (Dallas et al, 2014). In this study, it was found that participants were more likely to choose to drink alcohol if their (confederate) friend had also chosen to drink alcohol. Similarly, if their friend chose to consume soft drinks then the participant was more likely to choose soft drinks.

Secondly, there are inconsistent findings regarding sex differences and sex composition of the dyads which have been used. For example, in their meta-analysis, Quigley and Collins (1999) report a greater increase in sipping in heavy-drinking confederate conditions relative to controls when the dyads were mixed-sex and for female participants. The volume of alcohol consumed per sip was greater in heavy-drinking conditions relative to controls when the sex dyad was the same, but this effect was not present for mixed-sex dyads. There was, however, no moderating effect of sex on the amount of alcohol consumed. More recent research has also found the sex of the confederate has no

effect on the extent to which participants imitate confederate drinking (Larsen et al, 2010). However, males have been found to imitate sips more so than females even after controlling for the total amount of alcohol consumed (Larsen et al, 2010). These findings are contrary to recent ecologic momentary assessment (EMA) research which has shown sex compositions of natural drinking groups to affect alcohol consumption; with both sexes being found to drink less alcohol when in a group consisting entirely of the opposite sex and more alcohol within mixed-sex groups (Thrul et al, 2017).

Finally, as previously described, acute alcohol consumption has been shown to increase *ad lib* alcohol consumption (see de Wit et al, 1993; Rose et al, 2013). This alcohol priming effect may underlie binge drinking (Field et al, 2010) and so understanding factors that may moderate this effect is important for the development of potential interventions. Importantly, the majority of binge drinking episodes take place in the presence of others and when subject to alcohol's acute effects, however, there is a paucity of research that has examined the effect of acute alcohol consumption on subsequent alcohol-seeking in the company of other people. This may explain inconsistent findings between lab and recent EMA findings (Thrul et al, 2017) such as sex effects.

Acute alcohol consumption may affect imitation of drinking behaviour in a number of ways. Firstly, alcohol may impair inhibitory control (e.g. de Wit et al, 2000; Caswell et al, 2013; Gan et al., 2014; Mulvihill et al., 1997; Reynolds, Richards, & de Wit, 2006) and this impairment may lead to an increased reliance on social cues to guide behaviour. Indeed, previous cross-sectional work has suggested the theoretically similar construct of 'self-control' and aspects of impulsivity to moderate the relationship between self-reported peer and personal drinking (e.g. Robinson et al 2015; Stautz et al, 2014b). Secondly, the alcohol priming effect may render the effect of peer alcohol use redundant with participants going on to drink heavily regardless of other's drinking behaviour. Finally, given the strong effect of peer drinking, it is possible that exposure to low-drinking confederates may lead to a mitigated alcohol priming effect. If this is the case, then interventions that aim to reduce the influence of peers while individuals are intoxicated may be a novel method of reducing alcohol consumption and binge drinking.

Taken together, previous research has shown exposure to confederates influences the amount of alcohol participants consume. However, these studies often employ confederates that participants are not familiar with. The effect of acute alcohol consumption on imitation of alcohol consumption has not been assessed but is likely to be important as acute alcohol can prime further alcohol-seeking and impair inhibition. In particular, given the strong effect of confederate drinking, the current study assessed whether exposure to a light-drinking confederate can mitigate the alcohol priming effect. To do this, a 2x2 between-subject design was used wherein participants consumed an acute dose of alcohol or a placebo at the start of the session. Subsequently, participants were either exposed to a light-drinking confederate or were alone during an *ad lib* alcohol drinking session. The effect of alcohol on inhibition was also measured to assess the indirect effect of an acute dose of alcohol on subsequent drinking via alcohol-induced inhibitory impairments. In order to increase the validity of the current study further, a novel confederate paradigm (based on Dallas et al, 2014) was used wherein pairs of friends were tested with one allocated to be a confederate. The confederate was covertly trained to consume a small amount of alcohol (confederate condition) or left the study early (isolation condition).

It was expected that participants within the isolation condition would consume significantly more alcohol overall than the confederate condition. It was also hypothesised that there would be no difference in the amount of alcohol consumed between alcohol and placebo conditions within the confederate condition. The effect of acute alcohol consumption and confederate was also assessed for the number of additional drinks ordered and micro-drinking behaviours (latency to first sip, total number of sips and volume per sip). It was expected that these micro-drinking behaviours would increase following alcohol within the isolation, but not within the confederate condition. Furthermore, this study aimed to assess whether inhibitory control mediated the effect of acute alcohol consumption on *ad lib* alcohol consumption and whether this mediation was moderated by the presence of the confederate. Finally, separate exploratory analyses of the effect of participant sex on alcohol consumption and micro-drinking behaviours were conducted.

4.3 Method

4.3.1 *Participants*

The sample size for this study was determined by way of a power calculation conducted in G*POWER (Faul et al, 2007) to detect a medium effect size (f=0.25). According to this analysis 128 participants were needed to obtain 80% power. Overall, 129 pairs of participants were recruited (due to slight over recruitment, prior to termination of the study, to tackle participant nonattendance) those allocated to be participants were on average aged 19.36(±2.60), 63 participants were male and 66 were female. Those allocated to be confederates were aged 19.24(±1.74), 13 confederates were male and 116 were female. Due to recruitment issues, the sex compositions of the dyads were not able to be properly balanced. The majority of dyads were female/female (66) and female/male (with the female as the confederate; 50). There were 13 male/male dyads but 0 male/female dyads (with males as confederates).

In order to participate all participants were required to be aged 18-25, drink at least 10 UK Units (1 UK Unit= 8g alcohol) on an average week, be fluent in English and drink vodka at least occasionally. Participants were unable to take part if they were on medication which may be affected by alcohol or have a current illness which may increase their sensitivity to alcohol or have ever received treatment for a past or present alcohol disorder. Females that were pregnant or breastfeeding were not permitted to partake. The study received ethical approval by the University of Liverpool's ethics committee. All participants provided informed consent.

4.3.2 *Design*

This study used a factorial design with two between-subject factors. The first factor was drink type (alcohol/placebo) and the second was condition (confederate/isolation). Participants were allocated to these conditions using a fixed block allocation procedure. Drink content was single blinded.

4.3.3 *Materials*

4.3.3.1 Drinks preparation

The alcohol prime was calculated at 0.50g/kg (ASDA Triple Distilled Vodka, 37.5% alcohol by volume; ABV) mixed with lemonade (ASDA Diet Lemonade) in the ratio of one-part vodka to two parts lemonade. The placebo drink was lemonade of an equivalent volume. An atomizer was used to spray vodka mist on the surface and rim of the glass, as well as on top of the liquid for both alcohol and placebo drinks (Christiansen et al, 2017; Christiansen, Jennings et al, 2016).

4.3.3.2 AUDIT

The AUDIT (Saunders et al, 1993) was used to assess baseline hazardous drinking. The total AUDIT score was calculated and is presented here (current study following data reduction; α= .70).

4.3.3.3 TLFB

The TLFB (Sobell & Sobell, 1992) was used in the current study to assess alcohol consumption over the preceding two weeks.

4.3.3.4 LDQ

The LDQ (Raistrick et al, 1994) was used to assess baseline dependency (α=.79).

4.3.3.5 DIDS

The DIDS (Leeman et al, 2007) measures the extent to which euphoric, dysphoric and sexual disinhibition occurs during intoxication. The overall mean drinking-induced disinhibition measure was used (α=.68).

4.3.3.6 SIS

SIS's (Duka et al, 1998) were used to assess differences in subjective intoxication at three-time points at baseline post-priming drink and prior to the *ad lib* drinking session (end of session).

These scales were administered in both the alcohol and placebo conditions. Consistent with previous work (e.g. Christiansen et al, 2016) light-headedness was used as the primary measure of subjective intoxication (α=.62).

4.3.3.7 DAQ

The DAQ (Love et al, 1998) was used to assess craving across three-time points, baseline, post-priming drink and prior to the *ad lib* drinking session (end of session). While the DAQ is designed to assess different craving factors, previous research has shown the factor structure to be inconsistent, therefore, (Pasche et al, 2013; Kramer et al, 2010) mean craving scores across the entirety of the scale were used (α=.93).

4.3.3.8 Friend questions (Appendix 10)

Participants were also asked to complete a number of questions regarding their friendship with the other participant. There were provided with the open question 'How do you and the other participant know each other?' They were also asked to indicate how long they had known each other for and to indicate on a 5-point Likert scale (ranging from strongly disagree to strongly agree) to what extent they believed the other person to be their friend.

4.3.3.9 Unit estimation (Appendix 11)

Completed at the end of the experimental session, the unit estimation questionnaire asked participants to indicate how many units of alcohol they believed were in the priming drink (ranging from 0-9+).

4.3.3.10 Awareness (Appendix 12)

Participant awareness was assessed using a funnelled debriefing. They were first asked to openly record what they believed the aims of the study to be. They were then presented with a number of 5-point Likert scales (ranging from Strongly disagree- strongly agree) which assessed whether they believed that the confederate behaved normally, whether they believed the amount they drank to be

odd, the extent to which they believed the other participants drinking to influence their own, whether they would normally drink the drink they were provided with during the *ad lib* alcohol session and whether they liked the drink they were given. They also indicated (yes or no) whether they noticed the other participant's drinking.

4.3.4 *Ad lib drinking session*

During the *ad lib*, drinking session participants were provided with 235ml of vodka and cola presented in 250ml glasses. This drink during this session consisted of 15ml's of vodka mixed with 220ml of cola (ASDA Diet Cola). The *ad lib* drinking session lasted for 30 minutes, during this time they watched an edited version of the British comedy programme QI (Episode title: 'Just the job') which did not contain any mention of alcohol. Participants were presented with an electronic doorbell which they were told they could press at any time if they wished to order an additional vodka and cola (15ml vodka, 220ml cola) and that they could do this as many times as they wished.

4.3.4.1 SST

The SST was a replication of the STOP-IT program developed by Verbruggen et al (2008) and was ran using Inquisit 2.0 (Millisecond Software, 2002). This was presented on a 12-inch monitor. At the beginning of each trial a white fixation cross appeared for 500ms this was followed by an arrow pointing either left, on 50% of occasions, or right, on 50% of occasions. Participants responded by pressing the appropriate keyboard key. On 25% trials, an auditory stop signal was present, on these occasions participants were required to inhibit responding. There were four blocks, including the practice block of 64 trials. Stop signals were presented in a pseudo-randomized order. A tracking algorithm is incorporated into this version of the SST which adjusts the delay between the go stimuli and stop signal. SSRT was calculated as the main measure of inhibitory control and was calculated using the integration method (Verbruggen et al, 2013). Go reaction times and inhibition errors are also analysed and presented.

4.3.5 *Procedure*

Participants were recruited in pairs of friends with potential participants being asked to bring a friend to take part. The study ostensibly assessed the effect of alcohol on social interaction, comedy perception, and emotional expression. They were told that in order to assess this, their emotional expressions would be recorded using a webcam while they watched a comedy programme. Given the anticipated difficulty in recruiting sufficient males, it was decided that for female/male dyads the confederate would always be the female. However, for same-sex dyads, a random number generator (SPSS version 21) was used in order to allocate one participant to be the confederate. A schematic overview of the procedure is presented (see figure 4).

Testing took place in a semi-naturalistic 'lounge lab' which contains items designed to reflect a home lounge. This includes a couch, bookshelf, table and a television. Participants first provided informed consent, were breathalyzed and weighed so that the correct priming dose could be calculated. Following this they both completed baseline questionnaires (AUDIT, TLFB, LDQ, DIDS, DAQ, SIS, the questions regarding their friend and a bogus mood scale and TV viewing questionnaire used to corroborate the study's cover story), during this time the confederate was moved to a separate (neutral) lab under the pretence of protecting the anonymity of their questionnaire responses. The participant remained in the lounge lab.

Once the confederate had completed their questionnaires they were made aware of their role in the study. The confederate was instructed to consume two small sips of a drink that they would be provided with while watching a comedy programme. They were told that halfway through the comedy programme the researcher would enter to ensure they were happy to continue and following this they should take an additional two small sips. They were told to place the drink back on the table in between sips, not to let their friend know what they have been told to do, not to draw attention to their drinking and not to influence their drinking in any way. They were told that they were under no obligation to do this and could opt not to follow instructions if they so wished.

Confederates within the isolation condition were told that they had been randomly allocated to finish the study early. They were told that their friend would be watching a comedy programme in the lounge lab alone and that they will be led to believe they are doing the same in a different room. They were told not to let their friend know that this is the case.

Following this, both the confederate and participants were reunited in the lounge lab and provided with the priming drink. They consumed this drink together in the same room with the researcher present. They were provided with 10 minutes to consume the drink in its entirety and then rested during a 10-minute absorption period. Following the priming drink, they were separated again and provided with a small glass of water to sip prior to providing a breathalyzer sample. They then completed the second DAQ and SIS and the confederate was reminded of their role. Subsequently, both participants completed the SST followed by an additional breathalyzer sample, DAQ, and SIS.

Within the confederate condition, both participants were reunited in the lounge lab. They were told that their emotional expressions would be recorded using a webcam. In reality, they were recorded so that micro-drinking behaviours (latency to first sip, number of sips and volume per sip) could be recorded and so that the confederate could be observed to ensure instructions were adhered to. To corroborate the cover story, those present in the room were asked to hold a neutral facial expression and to look towards the camera for three seconds so that a baseline facial expression could be recorded. They were then presented with an initial vodka and cola drink and made aware that they could use the doorbell to order additional drinks. They were then left in the room to watch a comedy programme for 30 minutes. Halfway through the programme, the experimenter re-entered to ensure participants were content and to covertly indicate that the confederate should take two additional small sips. Within the isolation condition the above procedure remained the same, however, the confederate was not present in the room. The confederate was debriefed, received compensation and could leave following signing a waiver (Appendix 13).

Following the *ad lib* alcohol consumption session, in the confederate condition, the confederate and participant were separated into separate rooms again. The participant was then provided with a bogus mood scale and comedy perception questionnaire which asked a number of

questions regarding the content of the comedy programme. They were then provided with the unit estimation scale and the awareness measure. Subsequently, the participant, along with the confederate (within the confederate condition), were fully debriefed, signed a waiver and received compensation.

4.3.6 *Data reduction and analysis*

Three participants were deemed to be aware of the purpose of the study as they mentioned peer pressure or social influence within the awareness measure. These participants were excluded from all analyses. Out of the remaining participants, 3 video files were corrupted or lost due to a computer error. Their data was analysed but is absent for analysis of data gained from video recording. Videos were coded using ELAN 4.9.4. Consistent with previous work (Verbruggen et al, 2007; McGrath et al, 2016) reaction time data on the SST was trimmed with reaction times faster than 100ms, slower than 2000ms and more than three standard deviations above the mean being removed. Participants with negative SSRT's or those below 50ms were to be removed but there were no instances of this. Data from this study is available on the Open Science Framework (https://osf.io/s5r3e/).

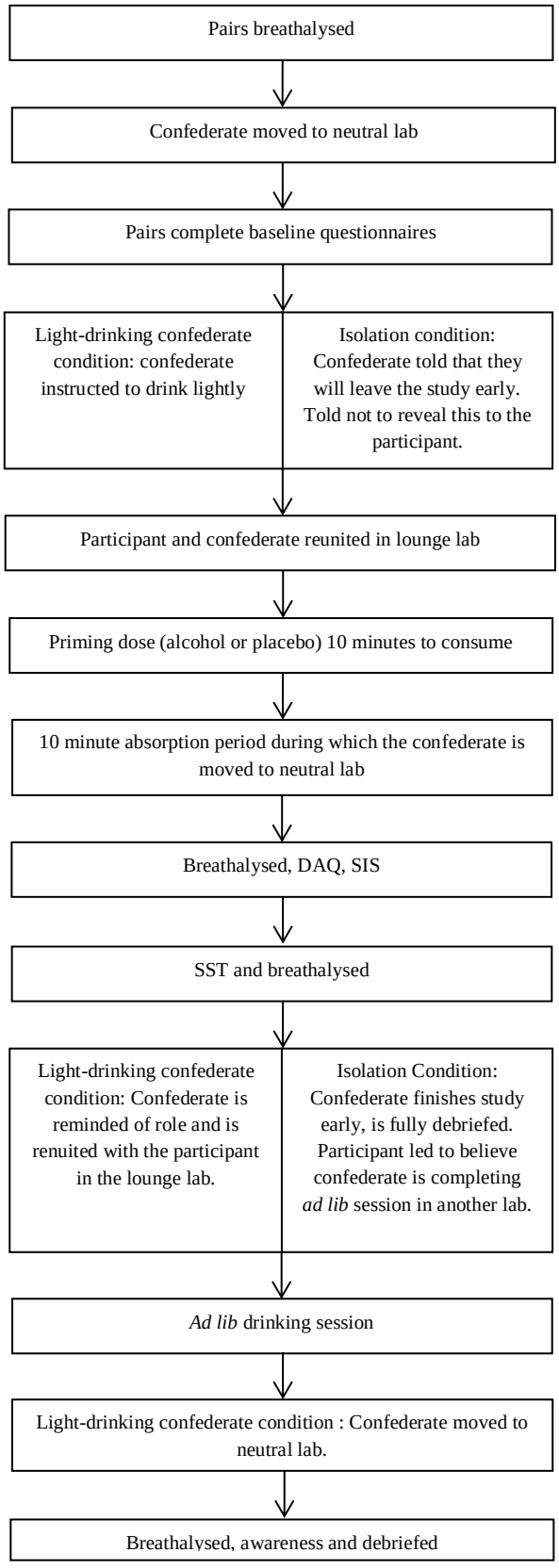

Figure 4 Study 2: schematic overview of experimental procedure

4.4 Results

4.4.1 *Participant Characteristics*

Following data reduction, 125 pairs remained in the analysis, 63 female/female dyads, 50 female/male dyads with the female as the confederate and 12 male/male dyads. Overall there were 62 male and 63 female participants with an average age of 19.36 (±2.60). There were 12 male confederates and 113 female confederates who had an average age of 19.24(±1.74). Pairs had known each other for an average of 82.97 (±160.10) weeks. Responses on the friend perception scale ranged from 3-5 with a mean of 4.78 (±.43). The majority of participants, 116, reported the confederate to be a friend, 8 reported the confederate to be a partner and 1 reported knowing the confederate only since the afternoon of testing.

A series of 2X2 ANOVA's with condition (confederate vs isolation) and drink type (alcohol vs placebo) as between-subject factors revealed no significant differences in age, alcohol consumption (TLFB), AUDIT, LDQ, mean DID, friend perception, liking of the drink in the *ad lib* session and perceptions of social influence (see table 3). This was the case for participants (all *p's*≥.151) and confederates (all *p's*≥.101). However, there was a significant difference between light-drinking and isolation conditions on the amount of time participants had known each other, F (1,121) =8.61, p=.004, η_p^2=.07. With participants in the confederate condition (123.27± 197.23) knowing the confederates for significantly more weeks than those within the isolation condition (43.32±98.84).

4.4.2 *Perceived alcohol content*

Perceived alcohol content (table 3) was assessed using 2x2 between-subjects ANOVA with the factors condition (confederate vs isolation) and drink type (alcohol vs placebo). There was a main effect of drink F (1,121) = 76.87, *p*<.001, η_p^2=.39. Participants estimated there to be significantly more units of alcohol in the alcohol drink (3.74 ± 1.44) than the placebo drink (1.78±1.04). Unit estimation did not differ between confederate and isolation conditions and there was no significant interaction (*p*'s >.220). A one sample t-test with a test value of 0 did, however, find participants

within the placebo condition to perceive there to be significantly more than 0 units of alcohol within the placebo, t (59) = 13.18, p<.001, d=1.70.

4.4.3 *Breath alcohol readings (BrAC)*

All participants provided a breath reading of 0.00g/100ml at baseline. Following alcohol, the mean BrAC was 0.19g/100 ml (±.08). At time 3 this increased significantly, t (63) = 2.77, p=.007, d=0.35, to 0.21g/100ml (±.07). BrAC significantly, t (63) =5.04, p<.001, d=0.63, increased again following the *ad lib* alcohol session to 0.25g/100ml (±.07). Within the placebo condition, BrAC remained at 0 until the *ad lib* drinking session following which BrAC was on average 0.04g/100ml (±0.4). A series of independent sample t-tests revealed no differences in BrAC between the confederate and isolation conditions at any time point (all p*'s*≥.534).

4.4.4 *Subjective Intoxication*

Light-headedness was used as the primary measure of subjective intoxication (see table 4). This was assessed using a 2X2X3 mixed ANOVA with condition (confederate vs isolation) and drink type (alcohol vs placebo) as between-subject factors, and time (baseline, post-drink, end of session) as a within-subject factor. There was a significant drink x time interaction, F (2, 240) = 26.16, p<.001, η_p^2=.18, which was further analysed using Least Significant Difference (LSD) tests. This revealed that following alcohol, light-headedness increased from baseline to post-drink (p<.001), from baseline to end of session (p<.001) and from post-drink to end of session (p=.004). However, within the placebo condition, light-headedness increased from baseline to post-drink (p=.001) and from baseline to end of session (p=.005) but not from post-drink to end of session (p=.830). At baseline, light-headedness did not differ between alcohol and placebo conditions (p=.951) but was greater following alcohol at post-drink (p<.001) and at end of session (p<.001).

Secondary measures of subjective intoxication were also analysed, main effects, interactions and post-hoc tests other than those reported were not significant (all p's≥.061). There was a main effect of time on irritableness, F (2, 240) = 12.37, p<.001, η_p^2=.09, and a significant drink x time interaction, F (2, 240) = 3.66, p=.027, η_p^2=.03. For participants who consumed alcohol, irritableness

significantly decreased from baseline to post-drink (p=.008) and increased from post-drink to end of session (p=.019). Within the placebo condition, irritableness increased from post-drink to end of session (p<.001) and from baseline to end of session (p=.001). Irritableness was greater within the placebo relative to alcohol condition at end of session (p=.021). Regarding subjective stimulation, there was a main effect of time, F (2, 240) = 30.18, p<.001, η_p^2=.20, and a significant drink x time interaction, F (2, 240) = 3.15, p=.045, η_p^2=.026. Regardless of drink type, stimulation increased from baseline to post-drink and from baseline to end of session (p's< .017). For alertness, there was a main effect of time only, F (2, 240) = 3.19, p=.043, η_p^2=.03, with a decrease from baseline to post-drink only (p=.017). For relaxation, there was a main effect of drink, F (1, 120) = 5.80, p=.018, η_p^2=.05, with participants within the alcohol condition reporting higher levels of relaxation than those within the placebo condition. There was also a main effect of time, F (2, 240) = 8.23, p <.001, η_p^2= .06, with relaxation increasing from baseline to post-drink (p<.001) and then decreasing from post-drink to end of session (p=.007).There was a main effect of time, F (2, 240) = 9.86, p<.001, η_p^2=.08, and drink, F (1, 120) = 5.14, p=.025, η_p^2=.04, on subjective feelings of contentedness. This was superseded by a drink x time interaction, F (2, 240) = 5.93, p=.003, η_p^2=.05, with contentedness increasing from baseline to post-drink within the alcohol condition (p<.001) and from baseline to end of session (p=.001). Within the placebo condition contentedness significantly decreased post-drink to end of session (p=.010). Contentedness was greater within the alcohol condition at post-drink (p=.022) and end of session (p=.001). All other main effects, interactions and post-hoc tests, other than those reported, were not significant (all p's >.061).

4.4.5 *Craving*

A 2X2X3 mixed ANOVA was conducted to assess the effect of condition and drink type on craving (mean DAQ scores; table 4). Overall, there was no main effect of drink F (1,121) = 1.23, p=.600, η_p^2=.002. But there was a main effect of time F (2, 242) = 32.02, p<.001, η_p^2= .21, which was subsumed by a significant drink x time interaction, F (2, 242) = 3.80, p=.024, η_p^2= .03. LSD tests revealed craving to increase from baseline to post-drink for both alcohol (p<.001) and placebo (p=.005), craving from post-drink to end of session did not differ for alcohol (p=.462) or placebo

(p=.914) but was greater than at baseline (alcohol: p<.001; placebo: p=.005). Comparisons between the drinks did not yield any significant differences (all $p's$≥.292). Change scores were calculated, and an additional ANOVA was conducted to investigate this interaction further.

Analysis of craving change scores yielded a significant main effect of time F (2,242) = 26.09, p<.001, η_p^2=.18 and drink, F (1,121) = 4.04, p=.047, η_p^2= .03, which was qualified by a significant drink x time interaction, F (1,121) =3.53, p=.031, η_p^2= .03. Comparisons between the two drinks revealed the increase in craving from baseline to post-drink to be greater within the alcohol condition (p=.024), there was no difference between drinks in craving change from post-drink to end of session (p=.666), but the increase in craving from baseline to end of session was greater following alcohol (p=.047). Taken together, this suggests that both alcohol and placebo drinks led to increased craving. However, this increase was greater when alcohol was consumed relative to placebo.

4.4.6 *Alcohol consumption*

A series of 2x2 between-subject ANOVA's were used to assess the effect of condition and drink type on a number of alcohol-seeking behaviours (table 4). Concerning volume of alcohol consumed during the *ad lib* session, there was no effect of drink type, F (1, 121) = 1.47, p=.228, η_p^2=.01, condition, F (1, 121) = .986, p=.323, η_p^2= .01 and no drink x condition interaction, F (1, 121) = .207, p=.650, η_p^2=.002. In addition, there was no effect of drink type, F (1, 121) = 3.29, p=.072, η_p^2=.026, condition F (1, 121) = .931, p=.336, ηp^2=.01, and no drink x condition interaction, F (1, 121) = .465, p=.496, η_p^2= .004 on the number of additional drinks ordered.

4.4.7 *Micro-drinking behaviours*

Using the recorded footage, micro-drinking behaviours (latency to first sip, total sips and average volume per sip) were assessed. Additional 2x2 ANOVA's found no effect of drink, F (1, 112) = .149, p=.700, η_p^2=.001, and no drink x condition interaction, F (1, 112) = .217, p=.642, η_p^2= .002 on latency to first sip. However, there was a significant main effect of condition on latency to first sip, F (1, 112) = 7.26, p=.008, η_p^2= .06, with participants exposed to a light drinking confederate having shorter latencies to first sip (in seconds; 46.38±96.13) than those who were alone (126.45±204.56).

Average number of sips was not affected by drink, F (1, 118) = .990, p=.322, η_p^2= .01, or condition, F (1, 118) = .860, p=.356, η_p^2=.01, nor was there a significant drink x condition interaction F (1, 118) = .339, p=.562, η_p^2= .003. Finally, regarding volume per sip, there was no significant effect of drink, F (1, 118) = .040, p= .842, η_p^2<.001, condition, F (1, 118) = 2.27, p=.135, η_p^2= .02 and no significant interaction, F (1, 118) = .002, p=.962, η_p^2<.001.

4.4.8 *Inhibitory control*

Three between-subject ANOVA's were used to assess the effect of condition and drink type on SSRTs, go reaction times and inhibition errors. There was no significant effect of drink, F (1, 121) = 1.97, p=.163, η_p^2=.02, condition, F (1, 121) = .019, p=.889, η_p^2<.001 and no drink type x condition interaction, F (1, 121) = .413, p=.522, η_p^2=.003, on SSRTs. The same pattern of results was found for inhibition errors with no effect of drink, F (1, 121) = .021, p=.885, η_p^2<.001, condition, F (1, 121) = .139, p=.710, η_p^2=.001 and no interaction F (1, 121) = .139, p=.710, η_p^2=.001. Finally, there was also no effect of drink, F (1, 121) = .265, p=.608, η_p^2= .002, condition, F (1, 121) = .016, p=.900, η_p^2<.001 and no interaction F (1, 121) = .415, p=.520, η_p^2= .003 for go reaction times.

Additionally, correlational analyses were conducted to assess the association between each of these measures and *ad lib* alcohol consumption. There were no significant correlations between go reaction times, τ_b (125) = .001, p=.983, inhibition errors, τ_b (125) = -.026, p=.698, or SSRT's r (125) = -.098, p=.275, and *ad lib* alcohol consumption. As there was no effect of acute alcohol consumption on any measure of inhibition, which occurred regardless of confederate condition, and no correlation between any of these measures and alcohol consumption, the planned mediation analysis was not conducted.

Table 3. Study 2 participant characteristics split by condition and drink type (values mean ±SD)

Characteristic	Confederate (n=62)		Isolation (n=63)	
	Alcohol (n=31)	Placebo (n=31)	Alcohol (n=34)	Placebo (n=29)
Gender (male: female)	15:16	16:15	17:17	14:15
Age	19.65(±2.67)	19.32(±1.54)	19.52(±2.03)	18.86(±3.82)
Alcohol consumption	46.70(±28.29)	54.45(±36.78)	50.47(±35.37)	55.55(±37.66)
AUDIT	14.32(±6.36)	15.06(±5.46)	14.32(±5.68)	14.93(±5.51)
LDQ	6.29(±4.12)	7.10(±3.70)	5.94(±3.30)	6.46(±4.90)
DID	3.13(±.79)	3.19(±.56)	3.07(±.82)	3.26(±.60)
Friend Perception	4.90(±.30)	4.77(±.43)	4.76(±.50)	4.69(±.47)
Weeks known	153.56(±236.53)	92.98(±145.77)	60.08(±125.51)	23.67(±48.21)
Liking (*ad lib* drink)	3.39(±1.28)	3.52(±1.09)	3.24(±1.10)	3.41(±1.15)
Perception of social influence	1.93(±1.26)	2.23(±1.06)	2.06(±1.00)	2.11(±0.93)

Alcohol consumption= Total amount of UK units (1 unit=8g alcohol) reported in the TLFB. AUDIT= Total scores on AUDIT questionnaire. LDQ= Total score LDQ. DID= Total scores DIDS, Friend perception= scores range from 1-5 on a single item 'The other participant is my friend' anchored with Strongly Agree- Strongly Disagree. Weeks known= how many weeks participants reported knowing the confederate. Liking (*ad lib* drink) = Score on one item 'I liked the drink I was given during the comedy programme' scores range from 1-5 anchored strongly disagree-strongly agree. Perception of social influence= 'The other participant influenced my drinking' scores 1-5 anchored strongly disagree-strongly agree.

4.4.9 *Sex differences: an exploratory analysis*

Given that the literature suggests there may be sex differences in the extent to which individuals are affected by other's drinking additional exploratory analysis of the *ad lib* drinking session variables with the inclusion of sex (male/female) as an additional between-subject variable was conducted. Regarding overall alcohol consumption this analysis yielded a significant main effect of sex, F (1, 117) = 21.02, $p<.001$, $\eta_p^2= .15$, which was superseded by a significant condition x sex interaction, F (1, 117) = 9.53, $p=.003$, $\eta_p^2=.08$. This interaction was the result of males drinking significantly more ($p=.004$) in the presence of a light-drinking confederate (319.35±166.04) than when alone (211.29±170.97). However, alcohol consumption did not differ (p=.165) between conditions for females when alone (173.44±143.15) relative to in the presence of a confederate (120.74± 92.32; see figure 5). There was, however, no main effect of condition, F (1, 117) = 1.26, $p=.265$, $\eta_p^2= .01$ or drink, F (1, 117) = 2.11, $p=.149$, $\eta_p^2= .02$. There was also no significant drink x condition, F (1, 117) = .422, $p=.517$, $\eta_p^2=.004$, drink x sex, F (1, 117) = .478, $p= .491$, $\eta_p^2= .004$ and no drink x condition x sex interaction, F (1, 117) = 2.19, $p=.141$, $\eta_p^2=.02$.

With the inclusion of sex, there was a significant main effect of drink type on the number of additional drinks ordered F (1, 117) = 4.09, $p=.046$, $\eta_p^2= .03$ with more drinks being ordered following alcohol (0.28 ± 0.54) relative to placebo (0.11± 0.32). There remained no significant main effect of condition, F (1, 117) = 1.13, $p= .291$, $\eta_p^2= .01$. However, there was a main effect of sex, F (1, 117) = 13.92, $p<.001$, $\eta_p^2= .11$, which was superseded by a significant condition x sex interaction, F (1, 117) = 4.45, $p=.037$, $\eta_p^2= .04$. This interaction was driven by males ordering significantly ($p=.028$) more additional drinks in the confederate condition (0.48±0.72) than when alone (0.23±0.50). There was no significant difference in the amount that females ordered between isolation (0.09± 0.39) and confederate (0.00±0.00) conditions ($p=.458$). There was no significant drink x condition, F (1, 117) = .720, $p=.398$, $\eta_p^2= .01$, or drink x condition x sex interaction, F (1, 117) = 3.60, $p=.060$, $\eta_p^2= .03$.

Regarding micro-drinking behaviours, there was a significant main effect of sex on latency to first sip, F (1, 108) = 5.15, $p=.025$, $\eta_p^2=.05$, with reduced latencies in males (53.75 ± 129.66) relative

to females (116.62 ± 186.12). As with the initial analysis, there remained a significant effect of condition on sip latencies, F (1, 108) = 6.57, p=.012, η_p^2= .06. There was no drink x condition interaction, F (1, 108) = .081, p=.776, η_p^2 =.001, condition x sex interaction, F (1, 108) = .272, p=.603, η_p^2=.003, drink x sex interaction, F (1, 108) = 1.87, p=.175, η_p^2= .02 and no drink x condition x sex interaction, F (1, 108) = 3.63, p=.059, η_p^2= .03. There was no change in the pattern of results regarding number of sips (all p's≥.067). However, there was a significant main effect of sex on volume per sip, F (1, 114) = 7.10, p=.009, η_p^2= .06, with males consuming greater volumes of liquid per sip (21.22 ± 10.41) than females (15.67 ± 12.21). Within this analysis there remained no effect of condition, F (1, 114)= 2.49, p= .118, η_p^2= = .02, no drink x condition interaction, F (1, 114)= .000, p=.984, η_p^2<.001, and there was no drink x sex, F (1,114)= .403, p=.527, η_p^2= = .004, condition x sex, F (1, 114)= .403, p=.527, η_p^2= .02 and no drink x condition x sex interactions, F (1, 114)= .077, p=.782, η_p^2=.001.

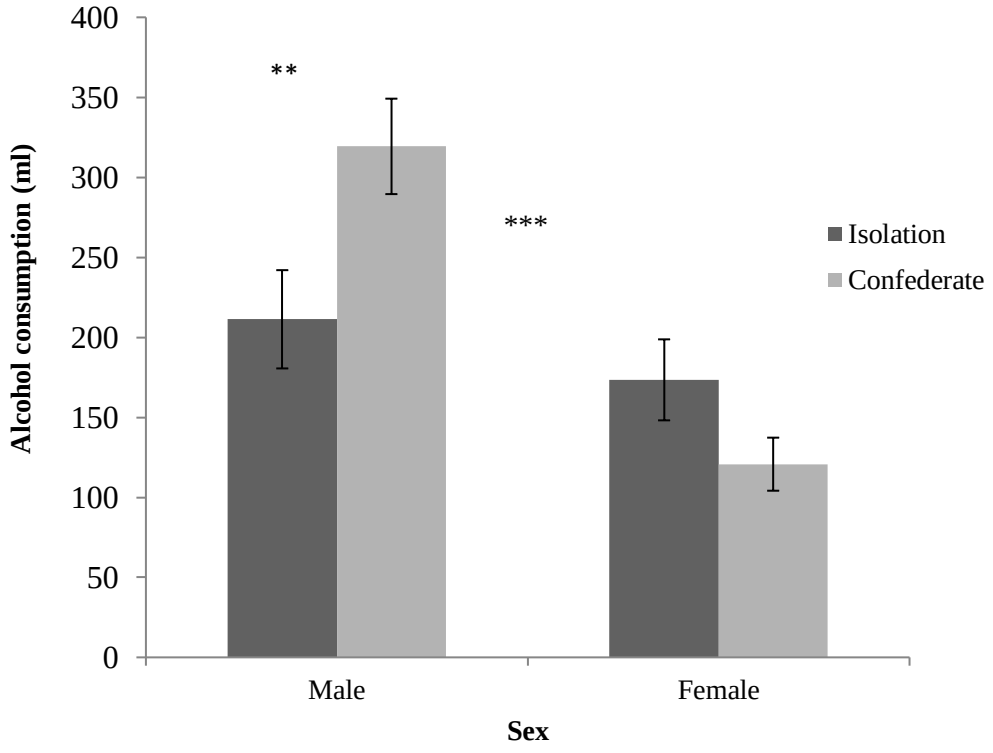

Figure 5. Mean alcohol consumed (in ml) for males and females split by condition (isolation/confederate). Values are mean ± SEM. (***p<.001; ** p=.004).

Table 4. Study 2 descriptive statistics for craving, light-headedness, unit estimation and alcohol consumed in *ad lib* session (values mean ± SD)

	Sample						Isolation						Confederate					
	Placebo			Alcohol			Placebo			Alcohol			Placebo			Alcohol		
	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session
Light-headed	9.07 (±15.24)	16.71 (±17.51)	16.25 (±15.92)	9.37 (±14.96)	33.08 (±20.35)	39.02 (±24.02)	10.28 (±19.45)	18.34 (±19.88)	18.10 (±19.21)	11.74 (±17.89)	33.97 (±20.85)	39.62 (±21.51)	7.90 (±9.83)	15.13 (±15.06)	14.47 (±12.00)	6.77 (±10.59)	32.10 (±20.09)	38.35 (±26.85)
DAQ	2.60 (±.71)	2.94 (±.87)	2.94 (±.97)	2.54 (±.67)	3.13 (±1.19)	3.09 (±1.15)	2.64 (±.75)	2.90 (±1.03)	2.94 (±.97)	2.49 (±.69)	2.94 (±1.00)	3.00 (±1.11)	2.67 (±.67)	2.98 (±.72)	3.04 (±.85)	2.60 (±.66)	3.34 (±1.36)	3.18 (±1.20)
Unit est	_	_	1.78 (±1.04)	_	_	3.74 (±1.44)	_	_	1.76 (±1.19)	_	_	3.47 (±1.24)	_	_	1.79 (±.90)	_	_	4.03 (±1.60)
Ad lib drinking (ml)	_	_	188.25 (±132.47)	_	_	222.28 (±184.64)	_	_	180.17 (±132.88)	_	_	202.21 (±176.91)	_	_	195.81 (±133.82)	_	_	244.29 (±193.26)

Light-headed scores range from 0(not at all) to 100(extremely). DAQ denotes Desire for Alcohol Questionnaire mean scores range from 1(minimum) to 7 (maximum). Unit est= Unit estimation, number of 25ml vodka measures participants believed the priming drink contained from 1 to 9+ (8g of alcohol= 1 UK unit).

4.5 Discussion

The current study aimed to assess the effect of a light-drinking confederate on the alcohol priming effect. It was hypothesised that the alcohol priming effect would be mitigated among those exposed to a light-drinking confederate, relative to participants in isolation. However, there is no support for this hypothesis. Overall, there was no effect of drink (alcohol/placebo) or condition (confederate/isolation) on the amount of alcohol consumed, the number of additional drinks ordered, the number of sips participants took or volume per sip. There was an effect of condition on latency to first sip with participants within the confederate condition having shorter latencies. An alcohol priming effect was observed in that craving increased to a greater extent following alcohol relative to placebo. Another aim was to assess whether inhibitory control mediated the alcohol priming effect and whether this was affected by the presence of a confederate. The lack of an effect of the alcohol prime on inhibition and correlations between inhibitory measures and alcohol consumption meant that there was no evidence for this mediation.

Findings differed, however, when sex was considered, with males consuming significantly more alcohol in the presence of a light-drinking confederate relative to the isolation condition, while the amount that females drank did not differ. This finding was reflected for the number of additional drinks ordered and the inclusion of sex also revealed there to be more additional drinks ordered when alcohol was consumed. Furthermore, males had shorter latencies to first sip and consumed a greater volume of alcohol per sip than females.

The current findings support previous alcohol priming research, in that an acute dose of alcohol increased craving relative to placebo (see de wit et al, 1996; Rose et al, 2013). However, it is notable that craving increased over time for both drinks, suggesting the anticipated effects of alcohol may also lead to increases in craving (Christiansen et al, 2016; 2017). Furthermore, when sex was considered, the alcohol prime increased the number of additional drinks which were ordered. Despite this, there was no overall effect of the alcohol prime on *ad lib* alcohol consumption.

There was no effect of alcohol on SST performance which is contradictory to previous experimental findings (e.g. de Wit et al, 2000; Caswell et al, 2013; Gan et al., 2014; Mulvihill et al., 1997; Reynolds, Richards, & de Wit, 2006) and theoretical models which suggest alcohol-induced impairments of inhibition to underlie the alcohol priming effect (Field et al, 2010). This is particularly notable as an effect of alcohol was found on craving, and drink orders, in the absence of an effect of alcohol on inhibition. This concurs with a growing body of research that suggests alcohol-induced impairments of inhibitory control do not underlie the alcohol priming effect (e.g. Christiansen et al, 2013; Baines et al, 2019).

As no alcohol priming effect was detected for *ad lib* alcohol consumption it was not possible to assess whether exposure to a light-drinking confederate would mitigate the priming effect. However, the effect of an alcohol prime on drink orders was significant, when sex was considered, and this effect was not moderated by confederate condition. This finding is contrary to previous research which has found participants to match the alcohol consumption of confederates (Quigley & Collins, 1999). One explanation for this discrepancy is that the current study used participant's friends as a confederate rather than an unfamiliar confederate. Ingratiation motives have been shown to moderate imitation of alcohol consumption when an unknown confederate is used and, as there may be less need to ingratiate oneself with existing friends, this may serve to reduce imitation (Robinson et al, 2016). Therefore, previous research using confederates may have exaggerated the extent to which people imitate alcohol use.

Males were found to drink significantly more alcohol, order more additional drinks and have shorter latencies to first sip within the confederate condition, while there was no difference between conditions for females on any of these measures. This suggests the mere presence of a friend may serve to increase alcohol consumption among males but not females. This is consistent with previous work which has shown the presence of others, particularly for males, to increase alcohol consumption in the 'real-world' (Thrul et al, 2017). This finding may be explained by the effect of social context on subjective response to alcohol. Indeed, previous research has demonstrated liking of alcohol and subjective response to be increased within social contexts relative to when alone (for a review see; de

Wit & Sayette, 2018). However, in the current study there were no observed differences in liking between conditions, and measures of subjective intoxication were taken prior to the *ad lib* drinking session but not during. Therefore, it is not possible to test whether differences in subjective response to alcohol between confederate and isolation conditions underlie increased alcohol consumption. Furthermore, the current findings are inconsistent with those suggesting light-drinking confederates reduce alcohol consumption relative to isolation (Quigley & Collins, 1999). This may be due to the use of a friend in this study, rather than an unknown confederate.

There are several issues with the current study. Firstly, one explanation for the absence of an alcohol priming and confederate effect on *ad lib* consumption, as well as the unexpected sex differences, may be due to the use of a placebo priming drink. This was done to isolate the pharmacological effects of alcohol on alcohol-seeking, however, research has shown that placebo-alcohol may also increase alcohol-seeking behaviour (Christiansen et al, 2016; 2017), indeed within the current study, craving was found to increase over time regardless of drink. As alcohol consumption in the 'real-world' is subject to both the pharmacological and anticipated effects of alcohol, a more ecologically valid comparison would be to compare a priming dose of alcohol to a control drink, which participants are aware contains no alcohol.

Furthermore, due to practical reasons, it was not possible to balance the gender composition of the dyads. Therefore, the majority of participants were exposed to a female confederate. It may be that the observed sex differences are not sex-specific but instead are the result of the majority of males being exposed to a member of the opposite sex while females were exposed to a same-sex confederate. In addition, confederates were instructed to take a total of 4 sips throughout the session and the extent to which this could be perceived to be 'light' drinking is unknown.

Future work is needed to investigate the moderating role of sex on imitation of alcohol consumption. These studies should aim to balance the gender composition of dyads and include a heavy-drinking confederate condition. Further work is also needed to assess the relative importance of the alcohol priming effect when drinking with others. Indeed, the next study of this thesis (study 3),

investigates whether priming doses of alcohol have an additive effect when participants are exposed to a heavy-drinking confederate.

In summary, the current study found that, overall, an alcohol prime increases craving and, when sex was considered, the number of additional drinks participants ordered, although it did not affect alcohol consumption or impair inhibitory control. Furthermore, exposure to a light-drinking confederate did not affect alcohol consumption. However, exploratory analysis of sex differences suggests males may consume more alcohol, order more drinks and consume more alcohol per sip in the presence of a light-drinking friend relative to when they are alone. Future studies should further explore the effect of acute doses of alcohol and the effect of sex on imitation of alcohol consumption among existing friendship dyads.

5 Chapter 5: Study 3 The effect of acute alcohol consumption on imitation of a heavy and light-drinking confederate.

The previous study found no effect of a light-drinking confederate or a dose of alcohol, relative to placebo, on *ad lib* alcohol consumption. However, when sex of participants was considered it was found that males consumed significantly more alcohol when exposed to the confederate. When sex was included there also a main effect of drink type on the number of additional drinks ordered, with more drinks being ordered following alcohol relative to placebo. In order to investigate the effect of acute alcohol consumption on imitation of alcohol consumption further, the current study addressed several limitations of the previous study. Firstly, rather than comparing a light-drinking confederate condition to an isolation condition the current study used light-drinking and heavy-drinking conditions. Secondly, the current study used alcohol and a control drink (which participants are aware contains no alcohol) comparison rather than an alcohol, placebo comparison. This is important as the use of an alcohol control comparison may more accurately reflect the real-world effects of alcohol (e.g. Christiansen et al, 2016). Finally, to provide greater standardisation, and given that the effect of confederate on drinking was found in the previous study only among males, only males were recruited as participants and females as confederates.

5.1 Abstract

The current study assesses the effect of an acute dose of alcohol on imitation of heavy and light-drinking friends. To do this, participants (*N*= 65) attended two sessions; in one session consuming an acute priming dose of alcohol and in the other a control drink. On a between-subject basis, participants were exposed either to a heavy or light-drinking friend who had been covertly recruited as a confederate. Due to the findings of study 2, mixed-sex dyads were used with males allocated as participants and females as confederates. *Ad lib* alcohol consumption was expected to be greater following alcohol relative to the control drink. Participant's *ad lib* alcohol consumption was expected to be increased when exposed to a heavy-drinking confederate following the control drink but not following an acute dose of alcohol. There was no effect of alcohol, relative to the control drink, on *ad lib* alcohol consumption although alcohol did increase craving. Despite this alcohol-induced increase in craving, *ad lib* alcohol consumption was greater within the heavy-drinking confederate condition regardless of whether alcohol or a control drink was administered. This finding suggests imitation of alcohol consumption to be a more important determinant of alcohol consumption than the alcohol priming effect.

5.2 Introduction

Given the theoretically strong effect of peer drinking (Quigley & Collins, 1999), the previous study investigated whether the alcohol priming effect would be mitigated when participants were exposed to a light-drinking confederate. However, while an alcohol priming effect was detected for craving, there was no effect of the priming dose on the amount of alcohol participants consumed during the *ad lib* drinking session. In addition, there was no overall effect of confederate condition, with alcohol-seeking behaviours being similar when exposed to a light-drinking confederate relative to when in isolation.

However, sex differences were observed; males consumed significantly more alcohol and ordered more alcoholic drinks when exposed to a confederate relative to when alone and when sex was considered, there was found to be an effect of alcohol on the number of additional drinks ordered. This contradicts previous experimental research which has suggested the effect of peers on alcohol consumption to be unaffected by the sex composition of drinking dyads (Quigley & Collins, 1999; Larsen et al, 2010). This finding is, however, consistent with recent EMA studies demonstrating sex composition of drinking groups to affect alcohol consumption (Thrul et al, 2017). Importantly, experimental research has been conducted only when participants are sober while the majority of drinking in the 'real world'- as assessed via EMA- takes place following an initial drink. It is, therefore, possible that the effect of sex on imitation of alcohol consumption has a more profound effect when individuals are intoxicated.

Study 2 is also limited in that it did not include a heavy-drinking confederate condition and, to my knowledge, no study has investigated the effect of a heavy-drinking confederate on alcohol consumption following an initial drink. This is important as both peer drinking (Creemers et al., 2017; Eisenberg et al., 2014; Elisaus et al., 2018; Kelly et al., 2016; Kuntsche et al., 2015; Scholly et al., 2014) and the alcohol priming effect (Field et al, 2010) have been suggested to underlie binge drinking. However, the relative importance of these mechanisms is unknown. For example, exposure to a heavy-drinking confederate may elevate alcohol consumption to a greater extent when individuals are also subject to the alcohol priming effect. Alternatively, as the peer effect is suggested to be

strong, the effect of an acute dose of alcohol on subsequent drinking may be negligible when exposed to a heavy-drinking peer.

In addition, placebo-alcohol can increase alcohol-seeking behaviour (e.g. Christiansen et al, 2017) and this increase in alcohol-seeking may mitigate the effect of other people's drinking. The lack of a peer effect in study 2 may, therefore, be due to increased alcohol-seeking, due to the belief that alcohol is being consumed, regardless of the actual contents.

The current study, therefore, aimed to assess the relative importance of the alcohol priming effect when exposed to a light-drinking and heavy-drinking peer. To do this, a similar method to Study 2 was used wherein an existing friend was covertly instructed to act as a confederate. The method was altered in a number of ways from the previous study. Firstly, to standardise the sex compositions of the dyads and, due to the sex differences found in males within Study 2, mixed-sex pairs of friends were recruited with the participant always being male and the confederate always being female. Secondly, a control drink was used rather than placebo as this offers a more ecologically valid comparison as it captures both the pharmacological and anticipated effects of alcohol which both exert an effect on 'real world' drinking (e.g. Christiansen et al, 2016). Thirdly, to simplify the instructions for the heavy-drinking confederate (who was instructed to drink the entirety of the drink), a shorter *ad lib* drinking session was used. Finally, a mixed-design was employed, with exposure to the light or heavy-drinking confederate being manipulated on a between-subject basis, and participants consuming alcohol or a control drink on a within-subject basis. A mixed, rather than between-subject design was used for practical purposes and to achieve sufficient power. One issue with this is that confederates may reveal the nature of the experiment in the interim between sessions. A number of measures were taken to reduce the impact of this. In the current study, only participants consumed the priming drink, although they were led to believe that the confederate was consuming the prime in another room. This was done in order to avoid the chance that the confederate may reveal the purposes of the study while intoxicated. In addition, a funnelled debrief was administered to both the participant and confederate, with the participant being asked if the confederate had revealed any information and the confederate being asked if they had revealed any information. Prior to these

questions being administered, participants were exposed to a bogus debrief which told them that the confederate had been told to perform an action during the study and the purpose of the study was to assess how well their friend could withhold this information.

On a between-subjects basis, participants were exposed to a light-drinking or a heavy-drinking peer. While, on a within-subject basis, the participant consumed a priming dose of alcohol in one session and, in the other, a control drink which they were aware contained no alcohol. Participants then completed an SST and a 10-minute *ad lib* drinking session with the confederate, who fulfilled the instructions, while watching a TV comedy programme. Because of the absence of a confederate effect in study 2, it was hypothesised that there would be no difference in alcohol-seeking (*ad lib* alcohol consumption and micro-drinking behaviours; latency to first sip, total number of sips and volume per sip) following the alcohol prime between confederate conditions. However, following the control drink, it was expected that participants would consume more alcohol when exposed to a heavy-drinking confederate relative to a light-drinking confederate.

5.3 Method

5.3.1 *Participants*

A power calculation using G*POWER (Faul et al, 2007) was used to determine the sample size. In order to detect a medium sized (f=0.25) within-between interaction, with 80% power, a sample size of 52 was recommended. Overall, 64 participants were recruited to account for potential exclusions. Mixed-sex pairs of friends were recruited exclusively with males always being allocated as the participant and the female allocated as the confederate. Participants were aged 21.47(±4.51) on average while confederates were aged 20.34(±2.78). Participants were all required to be aged over 18, drink at least 10 UK units (1 UK Unit= 8g alcohol) of alcohol on average per week and drink vodka at least occasionally. Participants were excluded from participation if they were on medication which may be affected by alcohol, had a current illness which may have increased their sensitivity to alcohol or having ever received treatment for an alcohol-related disorder. Females that were pregnant or breastfeeding were also excluded. The study received ethical approval from the University of Liverpool's ethics committee. The protocol for this study was preregistered in advance of data collection (https://aspredicted.org/nc7p5.pdf).

5.3.2 *Design*

This study used a mixed design; with a between-subject factor of confederate condition (heavy-drinking/light-drinking) and a within-subject factor of drink type (alcohol/control). Participants attended the lab on two occasions with at least 48hrs between each session; on one occasion they were provided with the alcohol prime and on the other the control drink (order counterbalanced). The order that these drinks were administered, and the experimental condition participants were allocated to was determined using a fixed block allocation procedure. Drink content was single blinded.

5.3.3 *Materials*

5.3.3.1 Drinks preparation

As with the previous study, the alcohol prime was calculated at 0.50g/kg (ASDA Triple Distilled Vodka, 37.5% ABV) mixed with lemonade (ASDA Diet Lemonade) at a ratio of one-part vodka to two parts lemonade. An atomizer was also used to spray vodka mist on the surface and rime of the glass and on top of the liquid. The control drink was lemonade of an equivalent volume to the alcohol drink (Christiansen et al, 2016, 2017).

5.3.3.2 Questionnaires

This study used the same questionnaire measures as study 2. This included; the AUDIT (α=.72), TLFB, LDQ (α=.80), DIDS (mean DIDS, α= .73), SIS's (α = .88), the DAQ (α =.90) and unit estimation scale.

5.3.3.3 Friend questions

Participants were asked a number of questions regarding their friend. They were provided with the open question 'How do you and the other participant know each other?' They were asked to indicate how long they had known each other, indicate whether they had ever consumed alcohol with their friend before and indicate on a 5-point Likert scale (ranging from strongly disagree to strongly agree) to what extent they believed the other person to be their friend.

5.3.4 *SST*

As with the previous study, a replication of the STOP-IT programme developed by Verbruggen et al (2008) was run using Inquisit 2.0 (Millisecond Software, 2006). SSRT was calculated using the integration method (Verbruggen et al, 2013) and was used as the main measure of inhibitory control. Go reaction times and inhibition errors were also analysed.

5.3.5 *Ad lib drinking*

During the *ad lib* drinking session, participants were provided with 245ml of vodka and cola presented in 250ml glasses. This drink consisted of a standard UK serving of vodka (25ml) mixed with 220ml of cola (ASDA Diet Cola). The *ad lib* drinking session lasted for 10 minutes, during this time they watched an edited version of the British comedy programme QI (Episode title: Series N, VG). Two different clips were used for each session; the presentation of which was counterbalanced, no mention of alcohol was present in either clip.

5.3.5.1 Awareness

Participant awareness was assessed using a funnelled debriefing procedure. The first section was the same for both the participant and the confederate. Within this section, participants were first asked the open question 'What do you believe the aims of the study to be?' They were then asked to rate on a 5-point Likert scale (Strongly disagree- Strongly agree) whether they would normally drink the drink provided during the *ad lib* session and whether they liked the drink. They then completed the unit estimation for the *ad lib* drink.

5.3.5.2 Awareness- participant specific (Appendix 14)

Following this, they rated how normal the confederate behaved during both sessions (if they disagreed they were provided with the option to openly record their reasons). Participants were then provided with a bogus debrief sheet which informed them that the study was assessed the extent to which alcohol consumption and personality is associated with the ability to keep secrets. Participants were informed that the confederate was told to perform an action which would make sense given the context of the experiment and to withhold this information during the study and during the gap between experimental sessions and that their ability to withhold this information was the main outcome measure.

Following exposure to this bogus debrief, participants were asked the open question 'What do you think your friend was asked to do/told?' A second open question asked 'Why do you think this?' Using this format, participants were also asked whether anyone else had revealed any information

about the study, whether their friend had said something which had made them suspicious and were finally asked to report any other relevant information.

Subsequent questions asked whether they had noticed, in both sessions, the confederates drinking while watching TV, whether the amount the confederate drank seemed odd and whether they believed the confederates drinking to influence their own.

5.3.5.3 Awareness- confederate specific (Appendix 15)

Confederates completed a different questionnaire which asked if they had revealed any information about their role in the study. If they responded in the affirmative they were asked to elaborate. Similarly, they were asked whether they had revealed any information about their role to someone who also knows their friend, whether they had intentionally, or unintentionally, made their friend suspicious about their role in the study and, finally, they were asked to report anything else they may have done which may have resulted in their friend knowing about what they were told to do.

5.3.6 *Procedure*

For a schematic overview of this procedure see figure 6. Participants were recruited in mixed-sex pairs. As with the previous study, this study ostensibly assessed the effect of alcohol on social interaction, comedy perception, and emotional expression. Testing took place in a semi-naturalistic 'lounge lab'. Within the first session, after providing informed consent, participants were breathalysed and weighed. The confederate (female) was then moved to a separate (neutral) lab under the pretence of completing baseline questionnaires anonymously. The participant remained in the lounge lab. Both the participant and confederate completed the AUDIT, TLFB, LDQ, DIDS, DAQ and SIS, the friend questions as well as a TV viewing questionnaire (to corroborate the cover story). The confederate was made aware of their role following the completion of the questionnaires. Specifically, they were either asked to consume one small sip of the *ad lib* session drink or to consume the drink in its entirety within the 10-minute period. They were asked not to reveal this to the participant during the session or during the time between the first and second session and not to influence the participants drinking behaviour in any way. They were told that they were under no obligation to do this and could opt not

to follow instructions if they wished. In the second session, participants were separated and completed only the DAQ and SIS at baseline, the confederate was reminded of their role, the rest of the procedure was identical in the second session.

Following this, the participant was provided with the priming drink (alcohol or the control drink; counterbalanced) and given 10 minutes to consume the drink which was followed by a 10-minute absorption period during which time they rested. The confederate was not presented with a priming drink but was provided with a sample drink to taste so that they were able to share their opinions about the drink if questioned by the participant. Following the absorption period, both participants were separated again, were provided with a small glass of water to sip before providing a breathalyser sample. They then completed the DAQ, SIS and the SST. Following the SST an additional breathalyser sample, DAQ and SIS were completed.

The participants were then reunited in the lounge lab and provided with the *ad lib* session drink. They were told that their emotional expressions would be recorded using a webcam. To corroborate this, they were asked to hold a neutral facial expression and look towards the camera for three seconds. They were then left alone in the room to watch the comedy programme for 10 minutes. At the end of the 10 minutes, the participants were separated again, breathalysed, completed the unit estimate for the priming drink, a bogus comedy perception questionnaire, and a waiver. At the end of the second session participants completed the awareness measures, were fully debriefed and compensated.

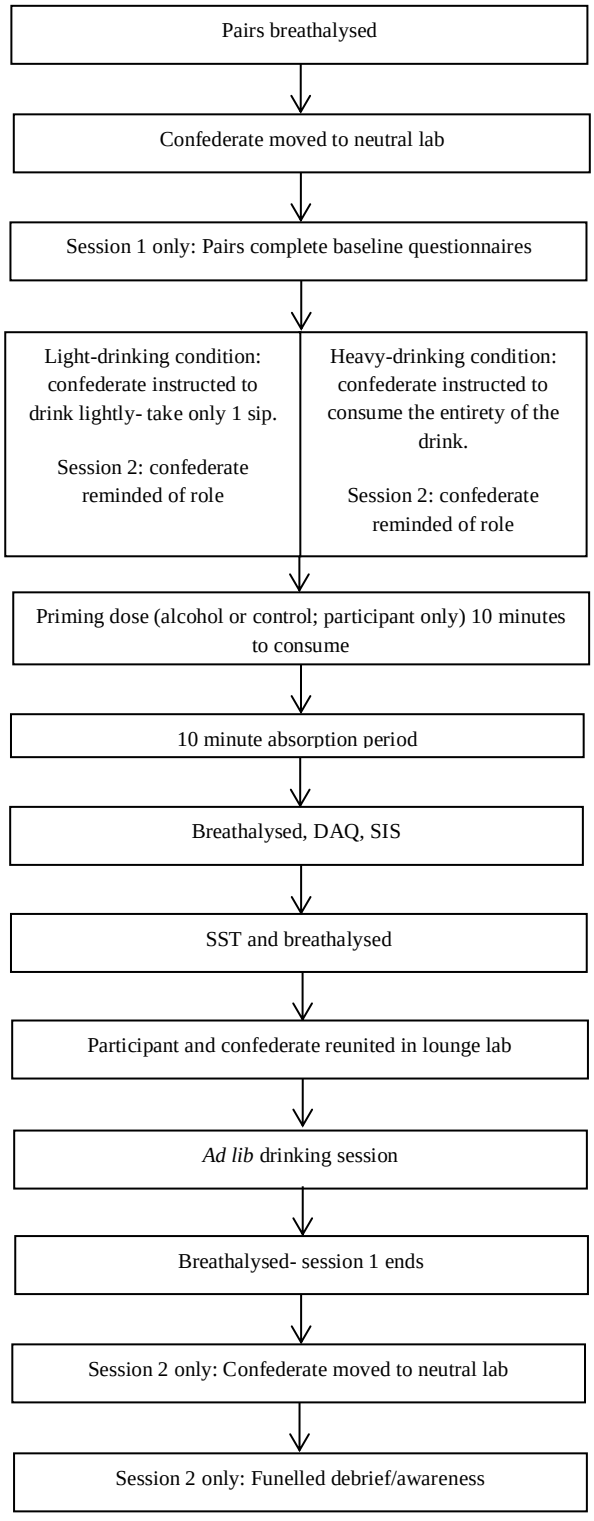

Figure 6 Study 3: schematic overview of experimental procedure

5.3.7 *Data reduction and analysis*

The awareness measures were independently rated by two researchers. Both the participant and confederate awareness measures were assessed. If either measure suggested that the participant was informed of the nature of the confederate's role they were excluded from all analysis. Similarly, if there was any indication that the confederate had attempted to influence the participants drinking in any way, other than that instructed they were also excluded from analyses. From this, two participants were identified as being aware of the confederate's role and one confederate did not follow instructions. In addition, one video file was lost due to a recording error. The data from this participant is analysed but is absent for analysis of data gained from video recording. Videos were coded using ELAN 4.9.4. Reaction time data on the SST was trimmed with reaction times faster than 100ms, slower than 2000ms and more than three standard deviations above the mean being removed. Participants with negative SSRT's or those below 50ms were to be removed but there were no instances of this (Verbruggen et al, 2007; McGrath et al, 2016). Data from this study is available on the Open Science Framework (https://osf.io/s5r3e/).

5.4 Results

5.4.1 *Participant characteristics*

Following data reduction 61 pairs of participants remained. All pairs were male/female dyads with females as the confederate. Participants had an average age of 21.52 (±4.60) while confederates had an average age of 20.34 (±2.83). Nine participants reported being in a relationship with the confederate while the majority of participants (52) reported being friends. According to the participant, pairs had known each other for an average of 27.39 (±48.69) weeks. Scores on the friend perception scale ranged from 4 to 5 with a mean of 4.82 (±.39). Only one participant reported never having consumed alcohol with the confederate before.

A series of independent samples t-tests with a between-subject factor of condition (heavy-drinking vs light-drinking) revealed no significant differences in baseline measures including age,

TLFB, AUDIT, LDQ, mean DID, friend perception and how long they had known the confederate (all p's >.065; see table 5).

5.4.2 *Awareness measures*

Further independent samples t-tests revealed there to be no significant differences between confederate conditions on awareness measures including; liking of, and whether they would normally consume, the *ad lib* drink, the extent to which they perceived their drinking to be influenced by the confederate, perceived normality of the confederates' behaviour, whether the drinking of the confederate was perceived to be odd, and estimates of alcohol units in the *ad lib* drink (see table 6; all *p*'s >.135).

5.4.3 *Perceived alcohol content*

The perceived number of units in the priming dose was assessed using a 2x2 mixed ANOVA with drink (alcohol/control) as a within-subject factor and condition (heavy/light-drinking) as a between-subject factor. There was a significant main effect of drink, $F(1, 59) = 431.47$, $p<.001$, $\eta_p^2=.88$, with participants reporting there to be a significantly greater number of units in the alcohol drink than in the control drink (see table 5).

5.4.4 *Breath alcohol readings (BrAC)*

A breath alcohol reading of 0.00g/100ml was recorded for every participant at baseline. Following alcohol, the mean BrAC was .18 (±.10), this then significantly increased, $t(60) = 4.41$, $p<.001$, $d=0.50$, to .21(±.10). Following the *ad lib* drinking session, BrAC significantly increased again, $t(60) = 8.57$, $p<.001$, $d=1.11$, to .27(±.11). For the control drink, BrAC's remained at 0 until following the *ad lib* alcohol session at which point mean BrAC's was .08(±.25).

5.4.5 *Subjective intoxication*

As with Study 2, light-headedness was used as the primary measure of subjective intoxication (see table 7). A 2X2X3 mixed ANOVA with condition (heavy vs light-drinking confederate) as a

between-subject factor and drink type (alcohol vs control) and time (baseline, post-drink, end of session) as within-subject factors. This analysis yielded a significant drink x time interaction, F (2, 118) = 42.40. $p<.001$, $\eta_p^2=.42$. LSD tests were used to explore this interaction further. This revealed light-headedness to increase following alcohol from baseline to post-drink ($p<.001$), from baseline to end of session ($p<.001$), but not from post-drink to end of session ($p=.442$). Within the control session, light-headedness did not increase from baseline to post-drink ($p=.103$) post-drink to end of session ($p=.558$) or from baseline to end of session ($p=.374$). Comparisons across the two drink sessions revealed there to be no difference in light-headedness between alcohol and control session at baseline ($p=.795$) but light-headedness to be greater within the alcohol condition as post- drink ($p<.001$) and at end of session ($p<.001$).

Secondary measures of subjective intoxication were also analysed, only significant effects are reported here, all other comparisons are not significant (all *p's*≥.055). There was a main effect of time on irritableness, F (2, 118) = 21.64, *p<.001*, $\eta_p^2=.24$, which was superseded by a drink x time interaction, F (1, 118) = 3.89, $p=.023$, $\eta_p^2=.06$. Within alcohol sessions, irritableness increased from post-drink to end of session ($p=.019$) and from baseline to end of session ($p=.037$). Within control sessions, irritableness increased from post-drink to end of session ($p<.001$) and from baseline to end of session ($p<.001$). Greater irritableness was reported within the control relative to alcohol session at end of session only ($p=.033$).

There was a main effect of time, F (2, 118) = 23.77, $p<.001$, $\eta_p^2=.29$, on subjective stimulation, with a significant increase in stimulation at every time point ($p<.001$). There was also a main effect of drink type, F (1, 59) = 5.53, $p=.022$, $\eta_p^2=.09$, which was superseded by a significant drink x confederate condition interaction, F (1, 59) = 4.99, $p=.029$, $\eta_p^2=.08$, this was characterised by greater stimulation within the alcohol, relative to control, sessions within the light-drinking confederate condition ($p=.002$).

For alertness there was a main effect of drink, F (1, 59) = 19.41, $p<.001$, $\eta_p^2 =.25$, and a significant drink x condition interaction, F (1, 59) = 7.72, $p=.007$, $\eta_p^2 =.12$, characterised by increased alertness between the heavy-drinking relative to light-drinking confederate condition within the

control session and greater alertness within the heavy-drinking condition between alcohol and control sessions (*p*'s<.001). There was also a main effect of time, F (2, 118) = 4.50, *p*=.013, η_p^2=.07, and a drink x time interaction, F (2, 118) = 4.78, *p*=.010, η_p^2 =.08. Following alcohol, alertness decreased from baseline to post-drink (*p*=.001), while within the control condition alertness increased from post-drink to end of session (*p*=.035). There were greater levels of alertness reported at post-drink and at end of session within control sessions relative to alcohol sessions (*p*'s<.001).

Table 5. Study 3 participant characteristics split by confederate condition (values mean ±SD)

Characteristic	Light-drinking confederate (n=29)	Heavy-drinking confederate (n=32)	Total (n=61)
Age	21.24(±4.21)	21.78(±4.99)	21.52(±4.60)
Alcohol consumption	43.49(±22.68)	39.97(±27.80)	41.65(±25.35)
AUDIT	14.72(±6.09)	12.03(±5.83)	13.31(±6.06)
LDQ	5.93(±4.28)	4.28(±3.00)	5.07(±4.13)
DID	3.32(±0.65)	2.95(±0.87)	3.13(±0.79)
Friend Perception	4.86(±0.35)	4.78(0.42)	4.82(±0.39)
Weeks known	18.41(±21.25)	35.52(±63.54)	27.39(±48.69)

Alcohol consumption= Total amount of UK units (1 unit=8g alcohol) reported in the TLFB. AUDIT= Total scores on AUDIT questionnaire. LDQ= Total score LDQ. DID= Total scores DIDS, Friend perception= scores range from 1-5 on a single item 'The other participant is my friend' anchored with Strongly Agree- Strongly Disagree. Weeks known= how many weeks participants reported knowing the confederate.

Table 6. Study 3 awareness measures split by confederate condition and drink type when appropriate (values mean ± SD)

Characteristic	Light-drinking confederate (n=29)			Heavy-drinking confederate (n=32)		
	Alcohol	Control	Total	Alcohol	Control	Total
Liking (*ad lib* drink)	-	-	3.52(±0.99)	-	-	3.16(±1.19)
Normally drink (*ad lib* drink)	-	-	3.17(±2.51)	-	-	2.41(±1.32)
Confederate drinking odd	3.10(±1.14)	2.83(±1.26)	2.97(±1.07)	2.94(±1.19)	2.97(±1.06)	2.95(±0.82)
Confederate behave normally	3.86(±1.03)	4.10(±0.78)	3.98(±0.80)	3.69(±1.09)	4.16(±0.77)	3.92(±0.78)
Influence	2.52(±1.06)	2.31(±1.04)	2.41(±0.86)	2.66(±1.21)	2.47(±1.37)	2.41(±0.86)
Ad lib unit estimate	1.69(±1.54)	0.97(±0.82)	1.33(±2.56)	1.88(±1.13)	1.02(±0.65)	1.45(±0.59)
Priming unit estimate	3.97(±1.55)	0.03(±0.19)	-	3.94(±1.29)	0.13(±0.34)	-

Liking (*ad lib* drink) = Score on one item 'I liked the drink I was given during the comedy programme', normally drink (*ad lib* drink) = 'I would normally drink the drink I was given during the comedy programme, Confederate behave normally= 'Do you think the participant behaved normally in session x?' Confederate drinking odd 'would you say the amount the other participant drank, while watching TV was odd in session x?' Perception of social influence= 'Would you say the amount the other participant drank, while watching TV, influenced how much you drank in session x?' scores for all of these questions ranged from 1-5 anchored strongly disagree-strongly agree. *Ad lib* unit estimate= 'How many standard 25ml shots of Vodka do you think were contained in the drink you were given, while watching TV, in session x'. Priming unit estimate= 'Estimate how many standard 25ml shots of Vodka you consumed at the beginning of the study.' Both unit estimates scores ranged from 0-9+.

5.4.6 *Craving*

Overall mean DAQ scores were used to assess the effect of condition and drink type on craving. To assess this, an additional 2x2x3 mixed ANOVA was used. There was a main effect of drink F (1, 59) = 19.94, $p<.001$, $\eta_p^2=.25$, which was subsumed by a significant drink by time interaction, F (2, 118) = 11.82, $p<.001$, $\eta_p^2=.17$. LSD tests revealed craving increased following alcohol from baseline to post-drink ($p=.013$), from baseline to end of session ($p=.004$) but not from post-drink to end of session ($p=.140$). Following the control drink, craving significantly decreased from baseline to post-drink ($p=.021$) and from baseline to end of session ($p=.031$) but not from post-drink to end of session ($p=.866$). Comparisons between drink sessions revealed there to be no difference in craving at baseline ($p=.235$) but craving was greater in alcohol sessions at post-drink ($p<.001$) and at end of session ($p<.001$).

While there was no significant main effect of confederate condition on craving, F (1, 59) = 2.84, $p=.097$, $\eta_p^2=.05$. There was an unexpected significant drink x confederate condition interaction, F (1, 59) = 6.67, $p=.012$, $\eta_p^2=.10$. Comparisons between confederate conditions revealed this interaction to be driven by greater overall craving within the light-drinking, relative to the heavy-drinking, confederate condition during alcohol sessions ($p=.019$) but not during control drink sessions ($p=.538$). Moreover, comparisons within drink sessions revealed there to be greater craving within alcohol, relative to control sessions, within the light-drinking ($p<.001$), but not heavy-drinking ($p=.177$), confederate conditions. See table 7.

Taken together this suggests that an acute dose of alcohol increased alcohol-seeking behaviour while a control drink led to reductions in craving. Overall, craving was greater in the light-drinking condition relative to the heavy-drinking condition but only during alcohol sessions.

5.4.7 *Alcohol consumption*

A series of 2x2 mixed ANOVA's were conducted to assess the effect of condition (heavy vs light-drinking confederate) and drink type (alcohol vs control) on a number on alcohol-seeking measures. Concerning alcohol consumption (in ml's) during the *ad lib* session, there was no

significant main effect of drink, F (1, 59) = .375, p=.543, η_p^2=.01, and no drink x condition interaction, F (1, 59) = 1.52, p=.222, η_p^2=.03. There was, however, a significant main effect of confederate condition, F (1, 59) = 5.17, p=.027, η_p^2=.08, with participants exposed to a heavy-drinking confederate consuming significantly greater amounts of alcohol (168.83± 83.09) than those exposed to a light-drinking confederate (121.98± 77.22).

5.4.8 *Micro-drinking behaviours*

2x2 mixed ANOVA's were used to assess the effect of confederate condition and drink type on latency to first sip, number of sips and volume per sip. Regarding latency to first sip, there was no main effect of drink, F (1, 51) = .522, p=.473, η_p^2=.01, no drink x condition interaction, F (1, 51) = 1.67, p=.203, η_p^2=.03, and no between-subject effect of confederate condition, F (1, 51) = 1.04, p=.313, η_p^2=.02. Similarly, there was no effect of drink, F (1, 58) = .661, p=.419, η_p^2=.01 no drink x condition interaction, F (1, 58) = .265, p=.608, η_p^2=.01 and no effect of condition on number of sips, F (1, 58) = 3.44, p=.069, η_p^2=.06. Finally, there was no effect of drink, F(1,58)= 3.46, p=.068, η_p^2=.06, no interaction, F(1,58)= 2.61, p=.112, η_p^2=.04, and no effect of condition, F(1,58)= 1.19, p=.281, η_p^2=.02, on volume per sip.

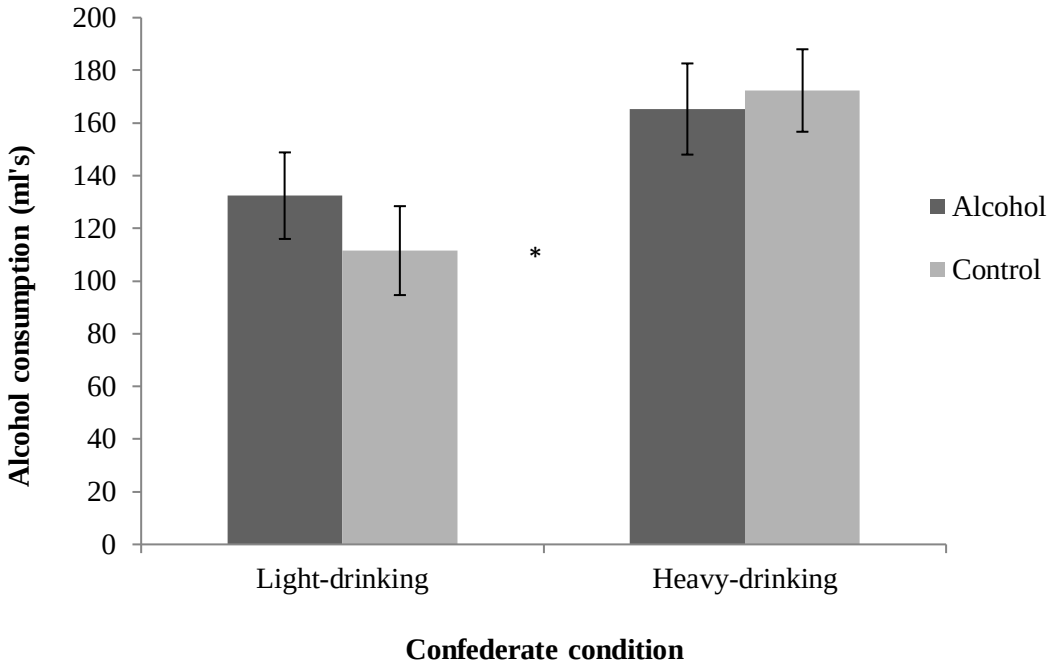

Figure 7. Mean alcohol consumed (in ml) split by confederate condition (light/heavy-drinking). Values are mean ± SEM. (*p=.027)

5.4.9 *Inhibitory control*

2x2 mixed ANOVA's were used to assess the effect of drink type on inhibitory control (SSRT's, go reaction times and inhibition errors). Confederate condition was also included in this analysis to assess for any baseline differences in inhibition. Regarding SSRT's, there was no main effect of drink type, F (1, 58) = 2.81, p=.099, η_p^2=.05, no drink x condition interaction, F (1, 58) = 1.07, p=.305, η_p^2=.02, and no effect of confederate condition, F (1, 58) = .245, p=.622, η_p^2=.004. There was a main effect of drink on go reaction times, F (1, 58) = 7.30, p=.009, η_p^2=.11, with slower responses following alcohol (249.12 ± 47.42) relative to control (232.60 ± 37.65). There was no significant interaction, F (1, 58) = .410, p=.524, η_p^2=.01, and no effect of confederate condition, F (1, 58) = .005, p=.924, η_p^2<.001. Finally, there was no effect of drink type, F (1, 58) = .007, p=.936, η_p^2<.001, no interaction, F (1, 58) = .576, p=.451, η_p^2=.01 and no effect of confederate condition, F (1, 58) = .707, p=.404, η_p^2=.01, on inhibition errors.

Correlational analyses also revealed there to be no significant associations with alcohol consumption, within alcohol or control sessions, for go reaction times (alcohol session: τ_b (61)= .039, p=.671; control session: τ_b (60)= .032, p=.732) inhibition errors (alcohol session: τ_b (61)= .108, p=.271; control session: τ_b (60)= .025, p=.069) and SSRT's (alcohol session: r (61)= .050, p=.720; control session: τ_b (60)= -.018, p=.849). As alcohol did not affect inhibition, regardless of confederate condition, and there was no association between measures of inhibition and subsequent alcohol consumption, the planned mediation analysis was not conducted.

Table 7. Study 3 descriptive statistics for craving, light-headedness and alcohol consumed in *ad lib* session split by confederate condition and drink (values mean ±SD)

	Sample						Light-drinking confederate						Heavy-drinking Confederate					
	Control			Alcohol			Control			Alcohol			Control			Alcohol		
	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session
Light-headed	11.62 (±17.74)	14.13 (±20.82)	13.31 (±18.47)	11.08 (±16.53)	40.41 (±27.94)	42.33 (±28.05)	12.86 (±19.30)	17.38 (±23.84)	14.10 (±19.25)	10.00 (±15.91)	42.00 (±28.67)	42.52 (±27.66)	10.50 (±16.42)	11.19 (±17.51)	12.59 (±18.01)	12.06 (±17.26)	38.97 (±27.63)	42.16 (±28.83)
DAQ	2.56 (±0.93)	2.40 (±0.92)	2.41 (±0.94)	2.66 (±1.01)	2.90 (±1.13)	3.00 (±1.24)	2.64 (±0.86)	2.49(±0.85)	2.46 (±0.83)	2.89 (±1.06)	3.25 (±1.05)	3.39 (±1.10)	2.48 (±1.00)	2.31 (±0.99)	2.36 (±1.04)	2.45 (±0.92)	2.59 (±1.12)	2.64 (±1.27)
Ad lib drinking (ml)	_		143.44 (±94.06)			149.67 (±94.25	-	-	111.55 (±90.90)	-	-	132.41 (±88.50)	-	-	172.34 (±88.58)	-	-	165.31 (±97.92)

Light-headed scores range from 0(not at all) to 100(extremely). DAQ denotes Desire for Alcohol Questionnaire mean scores range from 1(minimum) to 7 (maximum).

5.5 Discussion

The current study assessed the effect of an acute dose of alcohol, relative to a control drink, on imitation of heavy and light-drinking confederates. It was hypothesised that imitation of alcohol consumption would occur when participants had consumed a control drink but that this effect would not be present following an acute dose of alcohol. However, this hypothesis was not supported. Rather participants consumed more alcohol when exposed to a heavy-drinking confederate, relative to a light-drinking confederate, regardless of whether an acute dose of alcohol or the control drink had been consumed. Furthermore, the consumption of an acute dose of alcohol led to increased craving, while control drink consumption decreased craving, although the alcohol priming effect on craving did not translate to *ad lib* drinking. As with study 2, there was also no effect of alcohol on inhibitory control.

These findings support previous research which has suggested alcohol consumption to be influenced by the drinking behaviour of confederates (e.g. Quigley & Collins, 1999; Larsen, Engels et al, 2013; Larsen, Lichtwarck-Aschoff et al, 2013; Larsen et al, 2012; 2010) and, importantly, is the first study to show that volume of alcohol consumption, rather than choice, is influenced by the drinking behaviour of existing friends (Dallas et al, 2014). In addition, the current findings support the notion that acute doses of alcohol can increase craving for alcohol (see de Wit et al, 1993; Rose et al, 2013 for reviews). Despite this increase in craving, alcohol was not found to affect subsequent alcohol consumption. Taken together, this suggests imitation of peer drinking to be a more important determinant of alcohol consumption than the alcohol priming effect. This is particularly important as the majority of drinking occasions take place in the presence of others (Ally et al, 2016).

In the current study, inhibitory control was not impaired by alcohol. This contradicts previous findings which have found inhibitory control, as assessed using the SST, to be impaired by alcohol at similar doses to that which was administered in the current study (e.g. de Wit et al, 2000; Caswell et al, 2013; Gan et al., 2014; Mulvihill et al., 1997; Reynolds, Richards, & de Wit, 2006). However, the current findings are consistent with research (and study 2) which has found no effect of alcohol on inhibition and found evidence of a priming effect in the absence of these impairments (e.g.

Christiansen et al, 2013; Baines et al, 2019). This is the second study within this thesis which has failed to find an effect of alcohol on SST performance. This suggests that the reliability of the effect of alcohol on inhibition, as well as the suggestion that these impairments underlie alcohol priming (e.g. Field et al, 2010), to be exaggerated in the current literature.

However, there are some issues with the current study. Firstly, a mixed-design was used so that sufficient power could be achieved. As previously stated, it is possible that the confederate revealed the nature of their role to the participant during the interim between sessions. However, the novel awareness procedure (including the use of a bogus debrief), should have attenuated this issue and did allow identification of participants who were made aware of the role of the confederate. Indeed, the method of using participants' friends as confederates, and the novel awareness measure, is a particular strength of the current study and is a cost-effective method of assessing peer influence in the lab.

Another issue is that only mixed-sex dyads were used, with males always being the participant and the female the confederate. This approach was used to provide some standardisation in response to study 2's finding which showed males to consume more alcohol when in the presence of a confederate. It is therefore not known whether the current findings would translate for same-sex dyads or females exposed to male confederates. Finally, the current study only assessed alcohol consumption over a small period of time and using, relative to real-world drinking, a small dose (0.50g/kg) of alcohol as a prime. It is possible that the relationship between acute alcohol consumption and social influence may differ at higher doses and over an extended period of time. Future studies should, therefore, aim to test the effect of acute alcohol intoxication on imitation of alcohol consumption at higher doses.

It is also not clear, within this study, whether alcohol consumption increased due to exposure to a heavy-drinking confederate or decreased due to the light-drinking confederate. However, study 2 found alcohol consumption to be elevated in males exposed to a light-drinking confederate relative to when alone. Taken together, this suggests that the presence of a friend increases alcohol consumption but that this increase is greater if the friend is drinking heavily.

In summary, this study found participants consumed more alcohol when exposed to a heavy-drinking friend than when exposed to a light-drinking friend. This occurred regardless of whether the participant had previously consumed alcohol or a control drink and despite an alcohol-induced increase in craving. Alcohol, relative to a control drink, did not increase *ad lib* alcohol consumption. Finally, there was no effect of an alcohol prime on inhibitory control. These findings suggest imitation of alcohol consumption to be a more important determinant of drinking than the alcohol priming effect. As the current study used a sample of males who were exposed to a female confederate, future studies should continue to explore the effect of sex on imitation of alcohol consumption following an acute dose of alcohol and assess imitation at higher priming doses.

6 Chapter 6: Study's 4 and 5 The effect of beliefs about alcohol's acute effects on alcohol priming and alcohol-induced impairments of inhibitory control.

The previous three studies have investigated the effect of other people's alcohol consumption on personal alcohol consumption. Alongside this, the effect of alcohol on inhibition was also assessed. However, no effect of alcohol on inhibition has been found and there has been no clear effect of alcohol on subsequent consumption. However, the effect of alcohol on inhibition may be dependent on belief's regarding alcohol's acute effects. The next two studies aimed to investigate the role of alcohol-induced impairments of inhibitory control on the alcohol priming effect further. Specifically, these two studies both investigate the effect of acute alcohol consumption on alcohol priming and whether this effect is moderated by belief's regarding alcohol's acute effects on self-regulation.

This chapter reports the results from two studies which have been published (Appendix 16) as:

Knibb, G., Roberts, C.A., Robinson, E., Rose, A., & Christiansen, P. (2018). The effect of beliefs about alcohol's acute effects on alcohol priming and alcohol-induced impairments of inhibitory control. *PloS one*, 13(7), *e0201042.*

This chapter is altered from the published manuscript to be more cohesive with this thesis.

Study 4 was partly funded by an Alcohol Research UK small grant (SG13-14189).

6.1 Abstract

Several models argue that the alcohol priming effect is mediated by the effect of alcohol on inhibitory control. Alternatively, beliefs about how alcohol affects behavioural regulation may also underlie alcohol priming and alcohol-induced inhibitory impairments. Here two studies examine the extent to which the alcohol priming effect and inhibitory impairments are moderated by beliefs regarding the effects of alcohol on the ability to control behaviour. In study 4, following a priming drink (placebo or 0.50g/kg of alcohol), participants were provided with bogus feedback regarding their performance on a measure of inhibitory control (SST) suggesting that they had high or average self-control. However, the bogus feedback manipulation was not successful. In study 5, before an SST, participants were exposed to a neutral or experimental message suggesting acute doses of alcohol *reduce* the urge to drink and consumed a priming drink and this manipulation was successful. In both studies craving was assessed throughout and a bogus taste test which measured *ad lib* drinking was completed. Results suggest no effect of beliefs on craving or *ad lib* consumption within either study. However, within study 5, participants exposed to the experimental message displayed evidence of alcohol-induced impairments of inhibitory control, while those exposed to the neutral message did not. These findings do not suggest beliefs about the effects of alcohol moderate the alcohol priming effect but do suggest beliefs may, in part, underlie the effect of alcohol on inhibitory control.

6.2 Introduction

It has been suggested that the alcohol priming effect is mediated by alcohol-induced impairments in inhibitory control (Field et al., 2010). However, although previous research has demonstrated inhibitory control to be impaired following consumption of moderate (0.40-0.65g/kg) doses of alcohol (Abroms & Fillmore, 2004; Abroms, Gottlob, & Fillmore, 2006; de Wit et al., 2000; Marczinski, Abroms, Van Selst, & Fillmore, 2005); little research shows that such impairments mediate the alcohol priming effect.

Presently, only one study provides evidence that alcohol-induced impairments of inhibitory control correlate with subsequent alcohol consumption (Weafer & Fillmore, 2008). This study found that alcohol-induced impairments following a moderate (0.65g/kg) priming dose of alcohol were correlated with *ad lib* alcohol consumption measured in a subsequent testing session. Problematically, this does not offer convincing evidence that this impairment underlies the alcohol priming effect as participants did not consume a priming dose of alcohol in the same session as their alcohol-seeking was assessed. Critically, there is no evidence that alcohol-induced inhibitory impairments can account for *ad lib* alcohol consumption when measured in the same testing session (Christiansen et al., 2013; Fernie et al., 2012; Rose & Duka, 2007, 2008; Rose & Grunsell, 2008).

Furthermore, there is a growing evidence base suggesting that the alcohol priming effect cannot be wholly attributable to the pharmacological effects of alcohol on cognitive processes as anticipated (placebo) effects are also important (Christiansen, Jennings, et al., 2016; Christiansen et al., 2013; Christiansen et al., 2017; Marlatt et al., 1973). These anticipated effects are not limited to alcohol priming; placebo alcohol has also been found to impair inhibitory control (Christiansen, Jennings, et al., 2016), motor performance (Fillmore et al., 1994; Fillmore & Vogel-Sprott, 1994; Fillmore & Vogel-Sprott, 1995) and increased automatic approach tendencies (Christiansen et al., 2013). Furthermore, the anticipated effects of alcohol may be, at least in part, dependent on individual differences in alcohol-outcome expectancies. For example, impairments in inhibitory control and motor performance following placebo-alcohol are correlated with expectation of alcohol-induced cognitive impairment (Christiansen, Jennings, et al., 2016; Fillmore et al., 1994; Fillmore & Vogel-

Sprott, 1994; Fillmore & Vogel-Sprott, 1995). In addition, when participants were explicitly led to expect alcohol-induced impairment (but unknowingly consume placebo alcohol) their performance on a pursuit rotor task was improved relative to participants who were led to believe their performance would be enhanced, as participants in the former condition attempted to compensate for expected impairments (Fillmore et al., 1994). Such beliefs have also been shown to be important for performance on inhibitory control tasks such as the SST. For example, Fillmore and Blackburn (2002) found that when participants were led to expect impaired performance on an SST, their ability to inhibit responding improved following alcohol and placebo.

Beliefs about one's ability to control behaviour (regardless of actual ability) are likely to be important in explaining self-regulation. For example, participants led to believe that they possess high levels of 'willpower' or have 'self-control resources' available to them have been found to better regulate their behaviour than those led to believe they lack willpower/self-control (Clarkson, Hirt, Jia, & Alexander, 2010; Job, Dweck, & Walton, 2010). Critically, similar findings have also been demonstrated with regard to controlling substance intake.

Nordgren, van Harreveld, and van der Pligt (2009) provided smokers with bogus feedback, following a cognitive task, leading them to believe they had either high or low levels of 'impulse-control'; the authors report that those led to believe they possessed high impulse control were significantly more likely to smoke (notably this finding was actually non-significant, p=.06). However, Jones, Cole, Goudie, and Field (2012) led social drinkers to believe that they had either high or low levels of restraint prior to an *ad lib* alcohol consumption session. More alcohol was consumed by participants led to believe that their ability to control their behaviour was high. Problematically, neither of the aforementioned studies contained an average control group (i.e. a group told that their ability to control their behaviour was average) so it is unclear whether group differences in substance use were the product of the belief that ability to control behaviour was high or low.

Taken together, evidence suggests that manipulating beliefs about the ability to self-regulate is likely to influence *ad lib* alcohol consumption. It also suggests that the alcohol priming effect and

alcohol-induced inhibitory control impairments are, at least in part, the product of the belief that alcohol has been consumed and will impair self-regulation. The present research aims to assess the hypothesis that the alcohol priming effect, and alcohol-induced impairments of inhibitory control, is influenced by the belief that alcohol can impair behavioural control.

Two studies were conducted; both consisted of two experimental sessions with participants receiving a priming dose of alcohol (0.50g/kg) in one session and a placebo in the other. In both studies, participants completed measures of craving and subjective intoxication at three time points (baseline, post-drink, and end of session), an SST to assess inhibitory control and a bogus taste task at the end of each session. In the first study, participants were told that their performance on the SST was indicative of their ability to control their behaviour following alcohol. They were provided with bogus feedback following the task and were led to believe that they had high or average self-control similar to previous work (Jones et al., 2012; Nordgren et al., 2009). An 'average-control' condition was used so that the direction of the effect of high self-control beliefs could be properly elucidated.

In the second study, rather than implying that alcohol may lead to impaired self-regulation following alcohol (as with study 4), participants were explicitly told that consuming a small dose of alcohol *reduces* the urge to drink (experimental condition) or were provided with a neutral control message. Given that alcohol-related cues may impair inhibitory control and increase craving (Field & Jones, 2017) and bar-like environments may increase *ad lib* drinking (Lau-Barraco & Dunn, 2009; Moss et al., 2015) both studies were conducted in a semi-naturalistic bar laboratory.

Research has shown that if an individual believes that their self-regulation is poor they show poorer self-regulation (Clarkson et al., 2010; Job et al., 2010), but the direction of this effect on substance use has not been properly elucidated (Jones et al., 2012; Nordgren et al., 2009). Therefore, for the first study, it was hypothesised that the alcohol priming effect would be mitigated in participants led to believe that their ability to control their behaviour was high. So that participants within the high-control condition would consume less alcohol in the bogus taste task and report lower post-manipulation levels of craving than those within the average-control condition. For the second study, it was also predicted the alcohol priming effect would be mitigated among participants led to

believe that a small dose of alcohol would reduce their urge to drink (experimental condition) relative to the control condition. Finally, for study 5, it was hypothesised that alcohol-induced impairments of inhibitory control would be reduced within this group as demonstrated by improved performance on the SST relative to the control condition.

6.3 Study 4: Method

6.3.1 *Participants*

Eighty-one participants (44 male, 37 female) aged 18-49 (mean age 23.98 ± 6.49) were recruited via advertisements placed around the University of Liverpool or in return for course credit. The sample sizes for both studies were determined by a power calculation, using G*Power (Faul et al, 2007) to detect an effect of manipulated beliefs about self-regulation on *ad lib* alcohol consumption (η_p^2=.08; based on Jones et al, 2012). According to the power analysis, the target sample size for 80% power was *N*=70 more than this were recruited to account for potential removal of outliers. Participants were required to drink at least 10 UK units (1 UK unit= 8g alcohol) in an average week, be fluent English speakers, and like the taste of beer. Exclusion criteria included; past or present alcohol disorder, being on medication which may be affected by alcohol and current illness which may increase alcohol sensitivity. Females who were currently pregnant or breastfeeding were also excluded. The study was ethically approved by the University of Liverpool Research Ethics Committee and all participants in both studies provided written informed consent.

6.3.2 *Design*

This study used a mixed design with a within-subject factor of drink (alcohol/placebo) and a between-subject factor of condition (average-control group and high-control group). Participants attended the laboratory twice with at least 48 hours between sessions. During these sessions, they consumed a placebo or an alcoholic drink in a counterbalanced order. Participants were allocated to either experimental or control conditions using fixed block allocation. Drink content was single blinded.

6.3.3 *Materials*

6.3.3.1 Questionnaires

These studies used many of the same methods as study 2 and 3. This included; the AUDIT (Study 4 α= .75; Study 5 α=.66), TLFB, LDQ (Study 4 α= .82; Study 5 α=.82), SIS's (Study 4: α=.83, Study 5: α=.86), the DAQ (Study 4: α=.96, Study 5: α=.93) and a unit estimation scale.

6.3.3.2 Drinks preparation

The dose of alcohol participants received was calculated at 0.50g per kg of body weight. The alcohol drink contained vodka (Co-op Imperial Vodka, 37.5% alcohol by volume; ABV) which was mixed with chilled lemonade in the ratio of one-part vodka to three parts lemonade. The placebo drink consisted of lemonade (Co-op Sparkling Lemonade) of an identical total volume to the alcoholic drink. For both drinks an atomiser was used to spray vodka mist on the surface of the drink and the rim of the glass. This procedure and dose were used for both studies.

6.3.3.3 Manipulation Check

In order to assess whether the experimental manipulation affected participant's beliefs regarding their ability to control their drinking, item 3 ("I could easily limit how much I would drink if I drank now") and 14 ("If I started drinking now I would be able to stop") on the DAQ scale were used as a manipulation check. Both items ask participants to indicate the extent to which they believe they would be able to control their drinking after alcohol. A single variable was created by first reversing both items then summing the scores across these items and dividing by two (Pasche, Garner, Baldwin, & Sinclair, 2013).

6.3.3.4 Taste Test

Ad lib alcohol consumption was assessed using an adapted version of the bogus taste test procedure, a widely used and validated method for assessing alcohol intake (Jones et al., 2016). Participants were provided with numbered glasses each containing 225ml of beer. Participants were

instructed to rate the beers from 1 to 10 according to five different dimensions, identify the beers alcohol content, brand, and rank the drinks in order of preference. Participants were given 20 minutes to complete this task and were explicitly told to drink as much or as little of the drinks as they pleased. The drinks used in study 4 were; Skol (2.8% ABV) and Skol with 10ml of lemonade.

6.3.3.5 SST

An SST (Verbruggen, Logan, & Stevens, 2008b) was run using Inquisit 2.0 (Millisecond Software, Seattle, Washington, 2002), as with previous studies in this thesis this task was based on the STOP-IT program developed by Verbruggen et al (2008). SSRT was used as the primary measure of inhibitory control and was calculated using the integrated method. Go reaction times (reaction times on trials which require a response) and inhibition errors were also analysed.

6.3.4 *Procedure*

Testing took place in a semi-naturalistic bar laboratory at the University of Liverpool. The bar lab contains items associated with bars, including a stocked fridge, beer pumps, bar stools and seating similar to a typical British pub. At the beginning of both sessions, participants provided a breathalyser sample of 0.0mg/l (Lion Alcometer 500, Lion laboratories, Barry, UK) and were assigned to the experimental or control condition.

During the first session, participants were weighed and completed a battery of questionnaires (AUDIT, TLFB, LDQ, DAQ, and SIS). The second session began with the completion of the DAQ and SIS only. Participants were then administered the priming drinks (alcohol or placebo; order counterbalanced) which they were required to consume within 10 minutes. This was followed by a 10-minute absorption period during which time participants rested.

Following the absorption period, a second breathalyser sample was taken and the DAQ and SIS were completed. Participants were then informed that they would be taking part in a computer task which was designed to assess their ability to exert self-control following the consumption of alcohol. Following completion of the SST, participants were presented with a bogus feedback screen

which displayed a 'self-control index' score. For participants within the high-control condition, this score was 92.6%, while the score for those within the average-control condition was 51.2%. The experimenter visibly wrote down this score and provided further verbal feedback. Those within the high-control condition were told they were very good at controlling their behaviour following alcohol consumption and were within the top 10% of the population. Conversely, those within the average-control condition were told that they were average at controlling their behaviour following alcohol consumption and that most people scored similarly.

Participants then provided a third breathalyser sample and again completed the DAQ and SIS. At this point, participants were also asked to indicate how many units of alcohol they believed the priming drink contained on a 9-point scale (1-9+ units). The taste test was completed, and participants provided a final breathalyser sample. Following completion of the second session participants were debriefed and received compensation.

6.3.5 *Data reduction and analysis*

As with all other studies in this thesis, reaction time data was trimmed. Reaction times faster than 100ms, slower than 2000ms and more than three standard deviations above the mean were removed. As several participants inhibited responding significantly more or less than 50% of the time SSRT was calculated using the integration method (Verbruggen & Logan, 2009; Verbruggen et al., 2008b). Participants with negative SSRT's or those below 50ms were removed from SST analyses. Four participants were removed from SST analysis, and due to a technical issue, one participant's SST data was lost. Data for studies 4 and 5 are available on the Open Science Framework (https://osf.io/s5r3e/).

6.4 Study 4: Results

6.4.1 *Participant characteristics*

A series of independent sample t-tests revealed no differences between experimental conditions in age, units reported in the TLFB, scores on the LDQ and AUDIT scores (p's≥.301) A chi-square revealed no differences of gender between conditions, χ^2 (1) = 0.11, p=.459 (see table 8).

6.4.2 *Perceived alcohol content*

Perceived alcohol content (table 9) was analysed using a 2x2 mixed ANOVA with a within-subject factor of drink (alcohol, placebo) and a between-subject factor of condition (high-control/ average-control). There was a main effect of drink, F (1, 79) = 90.19, $p<.001$, $\eta_p^2=.53$, with participants estimating there to be significantly more units in the alcoholic drink than in the placebo drink. Perceived alcohol content did not differ between high and average-control conditions and there was no significant drink x condition interaction (p's$\geq.05$). A one-sample t-test with a test value of 0 found that participants perceived there to be significantly more than 0 units of alcohol in the placebo drink $t(80)= 9.43$, $p<.001$, $d=1.05$. This suggests that the placebo was successful and perceived alcohol content did not differ between levels of the between-subject factor.

Table 8. Study 4 Participant characteristics for experimental and control group (values mean ±SD)

Characteristic	High-control (n=41)	Average-control (n=40)	Sample (n=81)
Gender (male: female)	23 :18	21:19	44 : 38
Age (years)	23.10 (±4.92)	24.88 (±7.74)	23.98 (±6.49)
Alcohol consumption	45.67 (±28.65)	42.54 (±33.83)	44.12 (±31.16)
AUDIT	11.63 (±4.91)	13.76 (±8.47)	12.69 (±6.94)
LDQ	4.80 (±4.04)	4.85 (±3.79)	4.83 (±3.89)

Alcohol consumption= in UK units (1 unit= 8g alcohol), retrospectively recorded over two weeks. AUDIT= Alcohol Use Disorders Identification Test; scores range from 0(minimum) to 40(maximum). LDQ= Leeds dependence questionnaire, scores range from 0 (minimum) to 30 (maximum).

6.4.3 *Manipulation check*

To investigate whether the manipulation affected perceived ability to control drinking behaviour a composite score of DAQ item 3 and 14 was used, which pertain to perceived ability to control drinking. A 2 x 2 x 3 mixed ANOVA was used with condition (high-control, average-control) as a between-subject factor and drink (alcohol, placebo) and time (baseline, post-drink, end of session) as a within-subject factor. There was a main effect of drink, F (1, 79) = 9.74, p=.003, η_p^2= .11, with participants feeling less able to control their drinking during alcohol sessions relative to placebo sessions. There was also a main effect of time, F (1, 158) = 12.72, p<.001, η_p^2=.14, with participants reporting an increased ability to control their drinking from post-drink relative to baseline (p=.01) and from post-drink to end of session, p=.038. However, there were no other main effects of, or interactions with, experimental conditions, suggesting that the manipulation did not affect how well participants believed they could control their drinking (p's >.10).

6.4.4 *Breath alcohol readings (BrAC)*

All participants provided a breath alcohol reading of 0.00g/100 ml at the beginning of each session. Following alcohol mean BrAC was 0.31g/100ml (±0.10). Following the SST mean BrAC readings significantly decreased to 0.29(±0.08), t (79) = 2.65, p=.010, d=0.27, before significantly increasing, following the taste test, to 0.38(± 0.11), t (78) = 10.01, p<.001, d=0.99. Regarding the placebo session, participants did not consume any alcohol until the taste test, therefore BrAC readings were 0 at baseline and post-drink. Mean BrAC readings following the taste test, within the placebo condition, were 0.07(±.06).

6.4.5 *Subjective intoxication*

The light-headedness scale of the SIS was used as the primary measure of subjective intoxication (table 9). A 2 x 2 x 3 mixed ANOVA with condition (average-control, high-control) as a between-subject variable and drink (alcohol, placebo) and time (baseline, post-drink, and end of session) as within-subject variables revealed there to be a significant drink x time interaction, F (2, 158) = 75.95, p<.001, η_p^2=.49. Least significant difference (LSD) tests revealed that light-headedness

significantly increased following alcohol, $p<.001$, and placebo, $p<.001$. Light-headedness also increased post-drink to end of session within the alcohol session, $p=.015$, but not within the placebo session, $p=.800$. Light-headedness did not differ between alcohol and placebo sessions at baseline, $p=.122$, but was significantly greater following alcohol relative to placebo post-drink, $p<.001$, and at end of session, $p<.001$.

Secondary measures were also analysed significant effects only are reported here (all other *p's≥.055*). There was a main effect of time on irritableness, $F(2, 156) = 3.09$, $p=.048$, $\eta_p^2=.04$, as a result of a significant increase in irritableness from post-drink to end of session ($p=.012$). There was also a main effect of time on stimulation, $F(2, 158) = 18.62$, $p<.001$, $\eta_p^2=.19$, with stimulation increasing significantly from baseline to post-drink ($p<.001$) and from baseline to end of session ($p<.001$) but not from post-drink to end of session ($p=.267$). There was a main effect of drink, $F(1, 79) = 17.40$, $p<.001$, $\eta_p^2=.18$, and time, $F(2, 158) = 10.87$, $p<.001$, $\eta_p^2=.12$, on alertness. These main effects were driven by a drink x time interaction, $F(2, 158) = 5.75$, $p=.004$, $\eta_p^2=.07$, this was the result of greater alertness within placebo sessions relative to alcohol sessions at post-drink (*p<.001)* and end of session ($p<.001$) and significant decreases in awareness from baseline to post-drink ($p<.001$) and baseline to end of session ($p<.001$) within alcohol sessions only. Similarly, for relaxation there was a main effect of drink, $F(1, 79) = 4.02$, $p=.048$, $\eta_p^2=.05$, and time, $F(2, 158) = 6.95$, $p=.001$, $\eta_p^2=.08$, which was subsumed by a drink x time interaction, $F(2, 158) = 3.29$, $p=.040$, $\eta_p^2=.04$. This was the result of greater relaxation at post-drink ($p=.012$) and at end of session ($p=.035$) within alcohol sessions. Relaxation increased from baseline to post-drink ($p<.001$) and from baseline to end of session ($p=.015$) within alcohol sessions. For contentedness there was a main effect of time, $F(2, 158) = 7.87$, $p=.001$, $\eta_p^2=.09$, with contentedness increasing from baseline to post-drink ($p<.001$) and from baseline to end of session ($p=.010$).

6.4.6 *Craving*

A 2 x 2 x 3 mixed ANOVA was used to assess the effect of condition; drink and time on craving (mean DAQ scores; table 9). The results of this analysis yielded a significant main effect of

drink, F (1, 79) = 11.87, p=.001, η_p^2=.13, with craving significantly higher during alcohol sessions relative to placebo sessions. There was, however, no main effect of time, F (2, 158) = 1.44, p=.240, η_p^2=.02, condition, F (1, 79) = .339, p=.562, η_p^2<.01, and no significant drink x time interaction, F (2, 158) = 1.31, p=.273, η_p^2=.02. All other interactions were non-significant (p's≥.05).

6.4.7 *Inhibitory Control*

Three mixed ANOVA's were used to assess the effect of drink and condition, although there was no expected effect of condition, as participants were exposed to the experimental message following the task, on SST performance (SSRT, inhibition errors and go reaction times). There was no significant main effect of drink on SSRTs, F (1, 74) = 2.69, p=.105, η_p^2=.04, condition, F (1, 74) = .599, p=.441, η_p^2=.01, and no drink x condition interaction, F (1, 74) = 2.31, p=.133, η_p^2=.03. Similarly, go reaction times were unaffected by drink, F (1, 74) = 3.01, p=.087, η_p^2=.04, condition, F (1, 74) = .108, p=.744, η_p^2<.01 and the drink x condition interaction, F (1, 74) = .324, p=.571, η_p^2<.01.

There was no main effect of drink, F (1, 74) = 1.82, p=.182, η_p^2=.02, or condition, F (1, 74) = 3.09, p=.083, η_p^2=.04, on inhibition errors. However, there was an unexpected drink x condition interaction, F (1, 74) = 8.05, p=.006, η_p^2=.10. This was characterised by greater inhibition errors following alcohol (23.63±2.70) relative to placebo (22.38±1.96) within the average control condition, t (39) = 2.82, p=.008, d=0.53, but no difference between alcohol (22.06±2.28) and placebo (22.50±1.76) in the high control condition, t (35) = 1.14, p=.264, d=0.22.

This unexpected finding was explored further by running a univariate ANOVA to assess the effect of condition, session and order (which session alcohol and placebo were presented) on inhibition errors. A main effect of condition, F (1, 144) = 4.03, p=.047, η_p^2=.03 was found and was superseded by a significant condition x session x order interaction, F (1, 144) = 5.09, p=.026, η_p^2=.03. An LSD test revealed the three-way interaction to be the result of significantly greater inhibition errors when consuming alcohol (23.90 ± 1.97) relative to placebo (21.90 ± 1.58) but only when alcohol was consumed in the second session and only within the average-control condition, p=.004. There were no other significant differences, main effects or interactions (p's >.05).

This finding suggests that SST performance in the second session was affected by the bogus feedback provided to participants in the first session. Specifically, participants who were led to believe they had average self-control in the previous session performed worse in the second session when consuming alcohol.

Correlational analyses also revealed there to be no significant associations with alcohol consumption, within alcohol or placebo sessions, for go reaction times (alcohol session: r (76)= -.006, *p*=.956; placebo session: r (76)= .068, *p*=.561) inhibition errors (alcohol session: τ_b (76)= -.007, *p*=.935; placebo session: r (76)= .102, *p*=.379) and SSRT's (alcohol session: r (76)= -.163, *p*=.158; placebo session: τ_b (76)= -.041, *p*=.605). Although alcohol affected inhibitory errors (via an interaction with condition) there was no association of any inhibition measure and alcohol consumption. Therefore, the planned mediation analysis was not conducted.

6.4.8 *Taste Test*

A final mixed ANOVA was used to assess the effect of condition and drink on amount of beer consumed (in ml) during the taste test (table 9). There was a significant main effect of drink, F (1, 79) = 7.74, *p*=.007, η_p^2=.09, with participants consuming significantly more alcohol within the alcohol condition relative to the placebo condition. There was no significant drink x condition interaction, F (1, 79) = .12, *p*=.730, η_p^2<.01, and no main effect of condition, F (1, 79) = .116, *p*=.734, η_p^2<.01.

Table 9. Study 4 descriptive statistics for craving, light-headedness, unit estimation and alcohol consumed in the taste test (values mean ± SD)

	Sample						High-control						Average-control					
	Placebo			Alcohol			Placebo			Alcohol			Placebo			Alcohol		
	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session
Light-headed	9.99 (16.29)	18.91 (20.92)	18.53 (17.68)	9.99 (16.29)	41.74 (24.40)	46.70 (24.89)	.12(14.46)	17.05 (18.17)	18.38 (19.35)	10.93 (17.83)	39.24 (23.99)	45.02 (25.31)	.88 (17.76)	22.25 (18.82)	22.70 (17.99)	2.84 (.66)	2.90 (1.99)	49.43 (25.84)
DAQ	2.76(.68)	2.67 (.84)	2.61(.83)	2.94(.67)	3.00 (1.02)	2.91 (1.01)	2.78(.74)	2.65(.83)	2.65(.83)	3.03(.67)	3.09 (1.01)	2.96 (.87)	2.76 (.62)	2.69 (.81)	2.58(.84)	2.84 (.66)	2.90 (1.04)	2.86(1.15)
Unit est	_	_	1.90 (1.81)	_	_	4.14 (1.55)	_	_	1.85 (1.74)	_	_	3.73 (1.41)	_	_	1.90 (1.81)	_	_	4.14 (1.55)
Taste test (ml)	_	_	220.77 (131.44)	_	_	260.37 (129.02)	_	_	222.61 (143.15)	_	_	267.07 (135.88)	_	_	218.88 (120.06)	_	_	253.50 (122.94)

Light-headed scores range from 0(not at all) to 100(extremely). DAQ denotes Desire for Alcohol Questionnaire mean scores range from 1(minimum) to 7 (maximum). Unit est= Unit estimation, number of 25ml vodka measures participants believed the priming drink contained from 1 to 9+ (8g of alcohol= 1 UK unit).

6.5 Interim Discussion

In study 4, participants were provided with bogus feedback following an SST which suggested that their ability to control their behaviour was high or average. It was predicted that the alcohol priming effect would be mitigated when participants were led to believe that their ability to self-regulate was high following alcohol. The findings of study 4 do not support this hypothesis. Results suggest that an alcohol priming effect occurred as participants consumed more alcohol during the taste test following a priming dose of alcohol relative to placebo and that this occurred in the absence of alcohol-induced impairments in inhibitory control. However, while craving was higher overall during the alcohol session, craving did not further increase following the alcohol prime. Contrary to previous findings (Jones et al., 2012; Nordgren et al., 2009), there was no difference between participants led to believe they had high levels of self-control and those led to believe they had average self-control on any measure of alcohol seeking. It was also found that performance on the SST in the second session was affected by bogus feedback provided to them in the first session. Those led to believe their self-control was average had higher rates of inhibitory errors in the second session but only when alcohol was consumed. Study 5 was designed to test the effect of such beliefs on SST performance by manipulating beliefs prior to an SST.

In study 4 it was implicitly suggested that self-control following an acute dose of alcohol may affect subsequent drinking behaviour as participants were led to believe that their behavioural regulation was high or average following alcohol consumption. However, this manipulation did not affect how well participants believed they were able to control their drinking, suggesting that the manipulation was not successful. This manipulation may have been too subtle to affect beliefs about the effects of alcohol on control over drinking. Therefore study 5 addressed this by using a more explicit manipulation in which participants were directly told that consuming an acute dose of alcohol would reduce the urge to drink. In order to assess whether the alteration of these beliefs affected drinking outside of the lab, following the second session participants completed a two-week alcohol diary.

6.6 Study 5: Method

In study 5, participants were exposed to an experimental script (Appendix 17) which explicitly stated (see Clarkson et al., 2010; Job et al., 2010) that consuming a dose of alcohol would *reduce* the urge to drink or were provided with a neutral control message. Participants were exposed to this script prior to a priming drink and SST. It was hypothesised that the alcohol priming effect and alcohol-induced inhibitory impairments would be reduced following the experimental message.

6.6.1 *Participants*

Eighty-two participants (29 male, 53 female) aged 18-48 (*M*=26.30, *SD*= 8.01) were recruited via advertisements placed around the University of Liverpool or in return for course credit. Inclusion and exclusion criteria were identical to study 4.

6.6.2 *Materials*

In order to vary the taste tests which are used within the department the taste test within this study used three drinks Carlsberg (3.8% ABV), Becks Blue (<.05% ABV) and Fosters (4.0% ABV). All other materials were identical to the first study.

6.6.3 *Procedure*

The procedure for study 5 matched study 4. The only difference being that prior to the priming drink, the experimenter exposed participants to one of two messages under the pretence that they were informing the participant of the findings of the research programme so far. The experimental group were exposed to a message which suggested that consuming a small dose of alcohol would actually reduce the urge to drink:

Our research has found that consuming alcohol reduces the body's urge to drink as the body quickly becomes sated once it has received a small dose of alcohol, reducing the biological urge to drink. Furthermore, we have found that consuming large amounts of alcohol as part of an unplanned binge is a cultural phenomenon found in the UK and Ireland. Other European countries involved in our research program have not found that consuming alcohol leads to further alcohol consumption.

Meanwhile, the control group were provided with a control message:

Our research has been investigating the effects of alcohol on thought processes like memory, problem solving and attention. We have so far found that alcohol has a greater effect on some of these processes than others. This final experiment is testing the effects of alcohol on simple reaction times and taste perception.

Within the second session, participants were reminded of this information. The participants then completed the same procedure as study 4 (without bogus feedback following the SST) and were asked at the end of the second session to complete a two-week alcohol diary (Appendix 18) to assess whether the experimental manipulation was successful outside of the lab. Participants were fully debriefed when they returned with the diary.

6.6.4 *Data Reduction and analysis*

Data reduction and analysis were the same as for the first study. SST data from two participants in the control group were lost due to technical issues. An additional 6 participants presented SSRT's below 50ms and so were not included in SST analysis.

6.7 Study 5: Results

6.7.1 *Participant Characteristics*

Independent samples t-tests revealed no difference between the two script conditions for age, TLFB, AUDIT, and LDQ scores (all *p*'s≥.551). A chi-square revealed no gender differences between conditions, χ^2 (1) = .005, *p*=.942 (table 10).

6.7.2 *Perceived alcohol content*

There was a significant main effect of drink on the amount of alcohol perceived to be in the priming drink, F (1, 79) = 163.20, *p*<.001, η_p^2=.67, with participants estimating there to be significantly more units of alcohol in the alcoholic drink than in the placebo (table 11). There was no significant drink x script interaction and no main effect of script (*p's*≥.05). A one-sample t-test found

that participants estimated there to be significantly more than 0 units in the placebo drink, $t(79)=11.24$, $p<.001$, $d=1.26$.

Table 10. Study 5 Participant characteristics for experimental and control conditions (values mean ±SD)

Characteristic	Experimental (*n*=42)	Control (*n*=40)	Sample (*n*=82)
Gender (male: female)	14:27	14:26	29:53
Age (years)	26.66 (±8.16)	25.86 (±7.95)	26.30 (±8.01)
Alcohol consumption	45.43 (±18.91)	45.15 (±17.29)	45.29 (±18.02)
AUDIT	11.54 (±4.07)	12.13 (±4.75)	11.83 (±4.40)
LDQ	4.54 (±4.15)	5.08 (±4.00)	4.80 (±4.06)

Alcohol consumption= in UK units (1 unit= 8g alcohol), retrospectively recorded over two weeks. AUDIT= Alcohol Use Disorders Identification Test; scores range from 0(minimum) to 40(maximum). LDQ= Leeds dependence questionnaire, scores range from 0 (minimum) to 30 (maximum).

6.7.3 *Manipulation check*

There was a main effect of drink which was superseded by a significant drink x script interaction on the manipulation check, $F(1, 77) = 5.53$, $p=.021$, $\eta_p^2=.07$. Within the control condition, participants reported feeling less able to control their drinking during the alcohol session relative to the placebo session ($p<.001$). However, reported ability to control drinking did not differ between alcohol and placebo sessions within the experimental condition ($p=.462$). This suggests the experimental message was successful in reducing the belief that alcohol would lead to a loss of control over drinking.

6.7.4 *Breath alcohol readings (BrAC)*

Following alcohol mean BrAC reading was 0.30 g/100ml (± 0.15). Following completion of the SST, mean BrAC readings significantly decreased to 0.26 (±0.10), t (76) =2.84, p=.006, d=0.32, before increasing significantly to 0.36 (±0.15) following the taste task, t (72) =7.63, p<.001, d=0.78. Participants did not consume alcohol until the taste test within the placebo session. Following the taste test mean BrAC readings was 0.09 (±0.10).

6.7.5 *Subjective intoxication*

There was a significant drink x time interaction, F (2, 154) = 73.68, p<.001, η_p^2 =.489, with light-headedness increasing from baseline to post-drink, (p<.001), and from post-drink to end of session, (p=.001) following alcohol (table 11). Light-headedness also increased following placebo from baseline to post-drink (p<.001) and from baseline to end of session (p<.001), but not from post-drink to end of session (p=.372). There was an unexpected significant difference between alcohol and placebo conditions in baseline light-headedness (p=.001), with participant's reporting higher baseline light-headedness prior to placebo administration relative to alcohol. However, light-headedness was greater within the alcohol condition post-drink (p<.001), and at end of session (p<.001).

For secondary measures of subjective intoxication significant results are presented only (all other p's >.054). There was a main effect of time on irritableness, F (2, 154) = 9.98, p<.001, η_p^2 =.115, with irritability increase from post-drink to end of session (p<.001) and from baseline to end of session (p=.003). There was also a main effect of time on stimulation, F (2, 154) = 22.75, p<.001, η_p^2 =.23, which increased from baseline to post-drink (p<.001) and from baseline to end of session (p<.001). There was a main effect of drink, F (1, 77) = 4.90, p=.030, η_p^2 =.06, and time, F (2, 154) = 25.54, p<.001, η_p^2 =.25, on alertness. These effects were qualified by a significant drink x time interaction, F (2, 154) = 5.18, p=.007, η_p^2 =.06, which was the result of greater alertness within placebo sessions at end of session (p=.002). Alertness decreased within the alcohol session from baseline to post-drink (p<.001) and from baseline to end of session (p<.001). Alertness also decreased from baseline to post-drink within the placebo condition (p=.002). There was a main effect of drink, F

(1, 77) = 7.43, p=.008, η_p^2=.09, and time, F (2, 154) = 3.29, p=.040, η_p^2=.04 on relaxation. This was superseded by a drink x time interaction, F (2, 154) = 3.41, p=.035, η_p^2=.04. Relaxation was greater at post-drink (p=.009) and at end of session (p=.004) within alcohol sessions. Within alcohol sessions, relaxation increased from baseline to post-drink (p=.003). There was no main effects or interactions for subjective contentedness (all p's >.088).

6.7.6 *Craving*

There was a significant main effect of drink, F (1, 77) = 13.63, p<.001, η_p^2=.15, and time, F (2, 154) = 6.30, p=.002, η_p^2=.08, on craving (table 11). However, there was no drink x time interaction, F (2, 154) = 1.01, p=.366, η_p^2=.01, or main effect of script, F (1, 77) = .087, p=.769, η_p^2<.01. Overall, participants reported higher levels of craving within the alcohol condition than within the placebo condition. During both drink sessions, craving increased from baseline to post-drink, p=.009, and from baseline to end of session, p=.003, but not from post-drink to end of session, p=.943.

Table 11. Study 5 descriptive statistics for craving, light-headedness, unit estimation and alcohol consumed in the taste test (values mean ± SD)

	Sample						Experimental						Control					
	Placebo			Alcohol			Placebo			Alcohol			Placebo			Alcohol		
	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session	Baseline	Post-drink	End of session
Light-headed	9.95 (16.16)	19.62 (18.57)	20.49 (18.71)	4.61(8.19)	40.88 (22.51)	47.39 (25.48)	8.12(14.46)	17.05 (18.17)	18.38 (19.35)	4.14 (8.92)	41.90 (22.24)	45.36 (25.27)	11.88 (17.76)	22.25 (18.82)	22.70 (17.99)	5.07 (7.48)	39.90 (22.99)	49.43 (25.84)
DAQ	2.52(.83)	2.65 (.88)	2.63(.92)	2.63(.78)	2.87 (.91)	2.88 (.99)	2.57(.94)	2.60(.83)	2.67(.96)	2.56(.78)	2.78 (.92)	2.83 (1.01)	2.47 (.70)	2.70 (.95)	2.59(.90)	2.47 (.70)	2.70 (.95)	2.92(.90)
Unit est	_	_	1.40 (1.12)	_	_	4.06 (1.54)	_	_	1.29 (1.04)	_	_	4.17 (1.65)	_	_	1.53 (1.20)	_	_	3.95 (1.43)
Taste test (ml)	_	_	292.48 (182.33)	_	_	298.61 (179.47)	_	_	298.61 (183.09)	_	_	295.70 (178.20)	_	_	286.20 (183.66)	_	_	301.66 (182.96)

Light-headed scores range from 0(not at all) to 100(extremely). DAQ denotes Desire for Alcohol Questionnaire mean scores range from 1(minimum) to 7 (maximum). Unit est= Unit estimation, number of 25ml vodka measures participants believed the priming drink contained from 1 to 9+ (8g of alcohol= 1 UK unit).

6.7.7 *Inhibitory control*

There was no significant effect of drink on SSRT, F (1, 72) = 2.45, *p*=.122, η_p^2=.03. There was also no significant main effect of script, F (1, 72) = 3.07, p=.084, η_p^2=.04. However, there was a significant drink x script interaction, F (1, 72) =6.00, *p*=.017, η_p^2=.08. This interaction was the result of participants having greater SSRT's following alcohol (246.96± 108.58), relative to placebo (217.31±68.89), within the experimental condition, *t* (39) =2.65, *p*=.012, *d*=0.33, but there being no difference in SSRT's between alcohol (202.18 ±46.89) and placebo (208.71 ±35.58) session within the control condition, *t* (33) =.71, *p*=.480, *d*= 0.16 (see figure 8).

There was a significant main effect of drink on inhibition errors, F (1, 72) = 8.22, *p*=.005, η_p^2=.10, with higher error rates within the alcohol (23.85 ±3.29) relative to placebo condition (23.09±2.47). There was no significant drink x script interaction or main effect of script on inhibitory failures. There was also no significant main effect of drink, script and no significant drink x script interaction on go reaction times (*p*'s >.05). Overall, this indicates that the pharmacological effects of alcohol led to increased failures to inhibit responding. However, alcohol-induced impairments of SSRT were only present when participants were led to believe alcohol would reduce their urge to drink.

Correlational analyses also revealed there to be no significant associations with alcohol consumption, within alcohol or control sessions, for go reaction times (alcohol session: r (79)= .036, *p*=.756; placebo session: τ_b (73)= .058, *p*=.628) inhibition errors (alcohol session: τ_b (79)= -.002, *p*=.979; placebo session: τ_b (74)= .035, *p*=.687) and SSRT's (alcohol session: τ_b (79)<.000, *p*=.997; placebo session: τ_b (73)= -.007, *p*=.935).

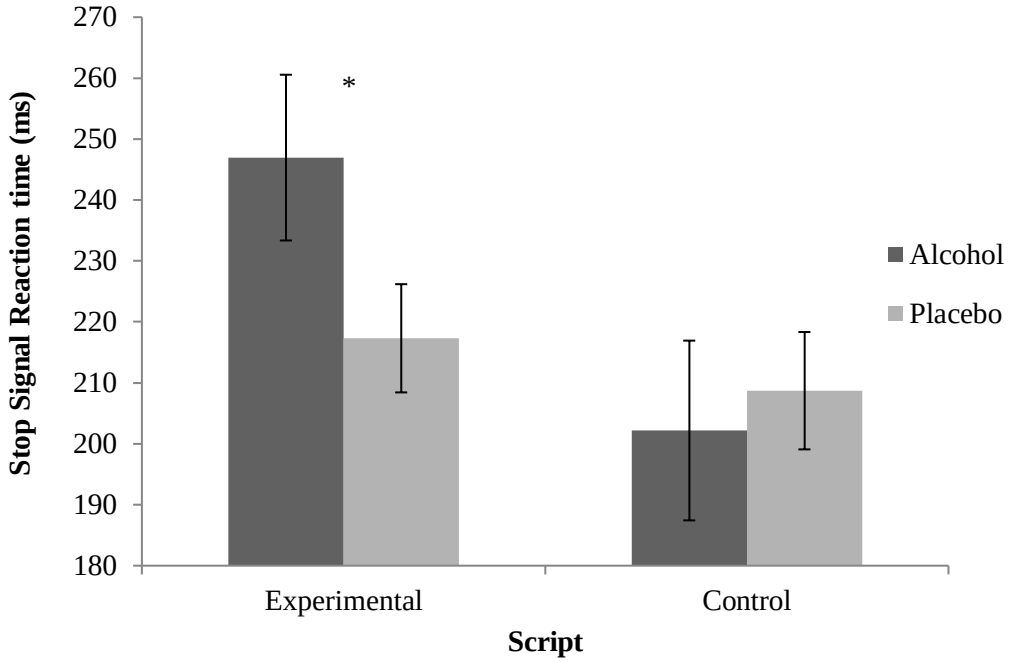

Figure 8. Mean integrated SSRT's (ms) following alcohol and placebo for both experimental and control condition. Values are mean ± SEM (*p=.012)

6.7.8 *Taste Test*

There was no significant main effect of drink, F (1, 78) = .066, p=.798, η_p^2<.001, on the amount of alcohol consumed in the bogus taste test. In addition, there was no significant drink x script interaction, F (1, 78) = .801, p=.373, η_p^2=.01, and no significant main effect of script, F (1, 78) = .002, p=.963, η_p^2<.001.

6.7.9 *Alcohol Diary*

Sixty-six participants returned the alcohol diaries (36 in the experimental condition, 31 in the control condition). A between-subjects t-test revealed there to be no significant differences between the control and experimental conditions on the amount of alcohol consumed 2 weeks prior to the second testing session, t (64) =0.63, p=.533, d=0.15.

6.8 Discussion

The current research aimed to manipulate beliefs about the effects of alcohol on behavioural regulation and assess the effect of such beliefs on the alcohol priming effect. In addition, study 5 also

explored the extent to which beliefs about the effects of alcohol can influence alcohol-induced impairments in inhibitory control. Study 4 provided participants with bogus feedback following an SST suggesting that they had high or average levels of self-control following alcohol. This study found no effect of this manipulation on alcohol consumption or craving; although it did reveal an alcohol priming effect with an alcohol-induced increase in *ad lib* consumption and higher levels of craving within alcohol sessions. It also suggested that performance on the SST was affected by bogus feedback they had received in the previous session.

In study 5, participants were either explicitly told that small doses of alcohol *reduce* the urge to drink or were provided with a control message. This occurred prior to receiving a priming drink and completing the SST. It was hypothesised that the alcohol priming effect and inhibitory control impairments would be reduced among those led to believe that a small dose of alcohol would reduce their urge to drink. As with the first study, the current findings do not support this hypothesis. While craving increased over time regardless of which drink was consumed, craving was higher in the alcohol session, relative to the placebo session and *ad lib* alcohol consumption did not differ between sessions. There was no effect of script on *ad lib* consumption or craving. While there was no difference between alcohol and placebo sessions for go reaction times, inhibition errors were greater following alcohol. Importantly, following exposure to the experimental script, there was evidence of alcohol-induced impairments of SSRT (greater SSRT following alcohol, relative to placebo) but no impairments were present following the control message.

Neither study 4 or 5 found beliefs regarding the effects of alcohol to affect alcohol-seeking. While this contradicts previous work which has suggested high perceived levels of behavioural regulation to be associated with increased substance use relative to low levels (Jones et al., 2012; Nordgren et al., 2009), it is important to note that participants in these studies were not intoxicated and the manipulation check suggests study 4's manipulation was unsuccessful. Alternatively, previous findings may have been driven by participants who led to believe that their ability to control their behaviour is low, reduce their substance use. Indeed, the current studies did not employ a low-control condition (study 4) or a condition suggesting alcohol will *increase* the urge to drink (study 5). In the

future, these conditions should be employed to properly disentangle the effects of perceived ability to self-regulate on subsequent drinking.

In study 5, participants who were informed alcohol would reduce the urge to drink had poorer inhibitory control following alcohol than control participants. This may occur as participants infer from the message that alcohol will not impair their ability to control their behaviour. Therefore, any compensatory effects which would usually occur, in an attempt to overcome alcohol's impairing effects, are not present. Indeed, Fillmore et al (Fillmore et al., 1994) found that participants led to expect alcohol-induced impairment on a pursuit rotor task performed better than participants led to expect improvement following alcohol. The authors suggest this is a result of participants attempting to compensate for the expected impairing effects of alcohol. The current results also support previous findings which suggest beliefs about the impairing effects of alcohol to lead to improvements on an SST, as assessed using inhibition errors and go reaction times (Fillmore & Blackburn, 2002). However, ours is the first to suggest that the expectation that alcohol will not impair self-regulation can lead to impaired SSRTs. Conversely, within study 5, inhibition errors were increased following alcohol relative to placebo regardless of belief. In study 4 those led to believe they had average self-control had higher rates of inhibitory errors in the second session when alcohol was consumed. This contrasts with study 5's finding that performance worsened when participants believed their ability to self-regulate would not be impaired by alcohol. Study 4 was, however, not designed to assess the effect of beliefs on SST performance. Furthermore, the manipulation in study 4 did not affect beliefs about the ability to control behaviour following alcohol but may have affected general beliefs about self-regulation. The effect of alcohol on inhibitory control may, therefore, be partly explained by individual differences in beliefs about the effects of alcohol (Christiansen, Jennings, et al., 2016). Future studies should take into account participant's beliefs about the effects of alcohol on their ability to control their behaviour when assessing the effect of alcohol on inhibitory control.

Study 4 replicated previous research, showing that initial alcohol consumption can prime further alcohol consumption (Christiansen et al., 2013; Christiansen et al., 2017; de Wit & Chutuape, 1993a; Hodgson, Rankin, & Stockwell, 1979; Ludwig et al., 1974; Marlatt et al., 1973). However, *ad*

lib beer consumption did not differ between alcohol and placebo sessions within study 5, although, craving was higher in the alcohol sessions. While study 4 found that more alcohol was consumed in the taste test following alcohol there was no evidence of inhibitory control impairments. In contrast, study 5 found evidence of inhibitory control impairments following an acute dose of alcohol but no evidence of an alcohol priming effect. Indeed, craving increased following both alcohol and placebo. This contradicts previous suggestions that such impairments mediate subsequent alcohol consumption (Field et al., 2010; Weafer & Fillmore, 2008) and supports research which has not found an association between inhibitory impairments and the alcohol priming effect (Christiansen et al., 2013; Fernie et al., 2012; Rose & Duka, 2007, 2008; Rose & Grunsell, 2008).

There are a number of limitations with the current studies. Firstly, the manipulation in study 4 did not affect participant's beliefs regarding their ability to control drinking and so was not successful. Participants were exposed to a 'self-control index' score following completion of the stop-signal task. Previous studies (Jones et al., 2012; Nordgren et al., 2009) have also included additional cognitive tasks, ostensibly to measures participants' behavioural regulation, and exposed participants to further information about their 'scores'. These components may increase the believability of the manipulation. Secondly, while craving was higher in alcohol sessions for both studies this was an overall difference and so may not have been increased following consumption of the priming drink. Furthermore, participants were not provided with water following the priming dose. It is, therefore, possible that alcohol residue in the mouth may have inflated BrAC readings. However, readings are similar to previous work which has used comparable doses (e.g. Erskine-Shaw, Monk, Qureshi, & Heim, 2017). Finally, both studies used different versions of the bogus taste test and so the amount of alcohol consumed between the two studies may not be directly comparable. While this is not ideal this was done in order to vary taste tests which are used in the department. Importantly, while the drinks differed, the form of the taste test remained the same and a number of different versions of the bogus taste test have been found to be valid (Jones et al., 2016).

In summary, neither of the two studies found that beliefs regarding alcohol's ability to control behaviour, following beverage consumption, moderated the alcohol priming effect. However, this

may be due to the absence of a low control group, for study 4, or a group led to believe an acute dose of alcohol will *increase* the urge to drink, in the case of study 5. This research adds to a growing body of research that suggests impairments in inhibitory control do not contribute to the alcohol priming effect. It is also the first to suggest that SSRTs may be impaired by the belief that alcohol will not lead to impaired self-regulation. Future studies should investigate the role of beliefs about the effects of alcohol on individual differences in alcohol-induced inhibitory control impairments and the potential effect of these beliefs on other widely used measures of inhibitory control.

7 Chapter 7: Study 6 Alcohol-induced impairments of inhibitory control and *ad lib* alcohol consumption: A secondary analysis.

The previous four studies of this thesis have administered priming doses of alcohol, assessed subsequent alcohol consumption and assessed inhibitory control using an SST. However, only one study has shown inhibitory control to be impaired following alcohol, and this occurred only when participants were exposed to an experimental script which aimed to manipulate beliefs regarding alcohol's acute effects. This is contrary to previous studies and theoretical frameworks concerning the effect of alcohol on inhibition and the alcohol priming effect. However, while all these studies were sufficiently powered to detect medium effect sizes it is possible that the effect of alcohol on inhibition is much smaller particularly given that only a moderate dose of alcohol (0.50g/kg) was used as a prime throughout. Therefore, the current study collated data across these four studies to provide increased power to investigate the effect of acute alcohol consumption on inhibition and the potential mediating effect of these alcohol-induced impairments on the alcohol priming effect.

7.1 Abstract

The majority of previous studies in this thesis have not demonstrated an effect of acute doses of alcohol on inhibitory control, nor an association between inhibitory control and *ad lib* alcohol consumption. Although this supports some previous findings, it is contrary to most previous research which suggests alcohol-induced impairments of inhibition to be present at doses ranging from 0.40g/kg-0.80g/kg and suggestions that these impairments underlie the alcohol priming effect. One explanation for this may be that the previous studies were underpowered to detect mediation. Therefore, this secondary analysis collates data from study's 2-5 of this thesis to increase power (*N*=326). All variables were standardised (z-scored) prior to analysis. Three mediation analyses were conducted to assess the indirect effect of acute alcohol consumption on *ad lib* alcohol consumption via, SSRTs, inhibition errors and go reaction times. There was found to be no mediating effect of any of these measures. There was, however, a direct effect of acute alcohol consumption on *ad lib* drinking. This analysis suggests that the alcohol priming effect is not mediated by alcohol-induced impairments of inhibitory control.

7.2 Introduction

Four of the previous studies have individually assessed the effect of an acute dose of alcohol on inhibitory control and the association between inhibitory control and *ad lib* alcohol consumption. Only one of these studies has found that alcohol to impair inhibitory control, and no study found that inhibitory control following alcohol consumption to be associated with subsequent drinking. Study 5 was the only one that found alcohol-induced impairments in inhibitory control; however, these were found only when participants were led to believe that acute doses of alcohol would not lead to subsequent consumption.

Although there is some research which has not found alcohol to impair performance on an SST (e.g. Loeber and Duka, 2009; Baines et al, 2019), it remains surprising that, across the four studies in this thesis, this was not demonstrated given the substantial amount of research which shows SST performance to be impaired at doses ranging from 0.40g/kg-0.8g/kg (e.g. Bartholow et al, 2018; Caswell et al, 2013; de Wit et al, 2000; Gan et al, 2014; Mulvihill et al, 1997; Reynolds et al, 2006). One possible explanation for the current null findings is that the previous studies in this thesis were not powered to detect an effect of alcohol on inhibition due to the relatively low dose of alcohol (0.50g/kg) that was employed throughout. Furthermore, the previous studies in this thesis have not found evidence of an indirect effect of acute alcohol consumption on *ad lib* drinking through impaired inhibitory control. This is contrary to previous suggestions that the alcohol priming effect is underwritten by alcohol-induced inhibitory impairments (Field et al, 2010; Weafer & Fillmore, 2008).

While the current studies were all sufficiently powered to detect medium effect sizes, if it is assumed that the effect of a 0.50g/kg dose of alcohol on inhibition and the association between inhibition and *ad lib* alcohol consumption is smaller than this, a much greater sample size is required. For example, using one recommendation (Fritz & Mackinnon, 2007), if the strength of the associations between these variables is small-medium then a sample size of at least 148 would be required to detect an indirect effect of alcohol on *ad lib* consumption (assuming 80% power, using bias-corrected bootstrapping).

This secondary analysis, therefore, aims to provide increased power to test the indirect effect of alcohol on ad lib consumption trough impairments in inhibition. To do this, data was standardized and collated from study's 2-5. Three mediation analyses were conducted with SSRTs, inhibition errors and go reaction times as mediators between priming dose (alcohol/no-alcohol) and *ad lib* alcohol consumption. An assumption of mediation analysis is that there is a significant IV-mediator and mediator-DV association. For illustrative purposes, mediation analyses were conducted regardless of these associations' significance. Given that the contradictory findings in the literature and the findings of the previous four studies it was predicted that there would be no significant mediation.

7.3 Method

7.3.1 *Participants*

Overall there were 199 males and 150 female participants, with one participant who did not record their sex, with an average age of 22.22 (±5.98). In all studies, participants were required to drink at least 10 units of alcohol per week. They were all required to like or drink occasionally the drink offered in the *ad lib* drinking session. Exclusion criteria included past or present alcohol disorder, being on medication which may be affected by alcohol and current illness which may increase alcohol sensitivity. Females who were currently pregnant or breastfeeding were also excluded.

7.3.2 *Data analysis and reduction*

Data were included from studies 2, 3, 4 and 5 of the current thesis. These studies were selected as all contained a measure of *ad lib* alcohol consumption, a priming drink, and the same SST. All studies also contained age, sex, AUDIT, TLFB, and LDQ which are controlled for in all analyses. All of these variables were transformed into z-scores within the original data allowing for comparisons across studies. As two of these studies (study's 2 and 3) had conditions in which there was a confederate present, who consumed either a light or heavy amount of alcohol, the presence of a light drinking confederate (light vs not light) and a heavy drinking confederate (heavy vs not heavy) was dummy coded. In addition, in two of the previous study's (4 and 5) beliefs regarding the effects

of alcohol were manipulated. A 'belief' variable was therefore created which indicated whether the belief manipulation aimed to lead to beliefs regarding unimpaired self-regulation following alcohol consumption (study 4; high-control group: study 5; experimental script) or did not (all other data points). Furthermore, studies either took place in the bar lab or the lounge lab; therefore, an additional 'environment' variable was created. All these categorical variables were added as covariates. Finally, a 'priming dose' variable was used to assess the effect of alcohol vs no-alcohol (placebo/control).

Participants were excluded using the same criteria as outlined in the respective study chapters. This included removing any participants who were aware of the main aims and those that presented negative SSRTs or SSRTs below 50ms. Three of these studies employed within-subject designs (study 3, study 4 and study 5). To ensure that the data remained independent, and to account for practice effects, only the first experimental sessions were used in the current analysis. In addition, participants with any missing data were not used in the current analysis. This data is available from the Open Science Framework (https://osf.io/s5r3e/).

7.4 Results

7.4.1 *Participant Characteristics*

Overall, data from 326 participants were analysed. Prior to all analyses, all variables were converted to z-scores within their original dataset. A multivariate analysis of variance (MANOVA) was used to assess differences in baseline characteristics (age, AUDIT, TLFB, LDQ) and each between-subject variable (table 12). There were no significant differences for any baseline characteristics between any of these variables (all p's≥.206).

Table 12 Sample characteristics. Values are mean ± SD.

Gender (male: female)	Age (years)	Alcohol consumption	AUDIT	LDQ
190:137	22.25(±5.99)	47.00(±29.40)	13.45(±6.06)	5.52(±4.02)

Alcohol consumption= in UK units (1 unit= 8g alcohol), retrospectively recorded over two weeks. AUDIT= Alcohol Use Disorders Identification Test; scores range from 0(minimum) to 40(maximum). LDQ= Leeds dependence questionnaire, scores range from 0 (minimum) to 30 (maximum).

7.4.2 *Indirect effect of acute alcohol consumption on ad lib drinking via SSRT*

In order to assess the indirect effect of an acute dose of alcohol on *ad lib* alcohol consumption via inhibitory control, mediation analyses with bias-corrected bootstrapping were conducted using the PROCESS macro for SPSS (Hayes, 2012). Within this model, age, sex, AUDIT, TLFB, LDQ, belief, presence of a confederate and environment were added as covariates.

Overall, there was a significant direct effect of priming dose on *ad lib* alcohol consumption (β=-.220, *p*=.033) with acute doses of alcohol being associated with greater consumption during *ad lib* sessions than placebo and control drinks. However, there was no association between priming dose and SSRT's (α path; β=-.031, *p*=.767), and no association between SSRT's and *ad lib* alcohol consumption (β path; β=-.023, *p*=.673). Therefore, there was no significant indirect effect of priming dose on *ad lib* alcohol consumption through SSRT's (*b*<.001, SE= .006, 95%, BCa CI -.009, .020).

The total effect model was significant, R^2= .179, F (9, 315) = 6.89, *p*<.001, with priming dose (β=-.219, *p*=.034), TLFB (β=.176, *p*=.003), LDQ (β=.151, *p*=.020) and sex, with males drinking more than females, (β=--313, *p*<.001) being significantly associated with *ad lib* consumption. In addition, the presence of a heavy-drinking confederate was associated with greater alcohol consumption (β=-.403, *p*=.041). However, AUDIT scores (β=-.025 *p*=.709), environment (β=--105, *p*=.521) age

(β=.044, *p*=.403), belief (β=-.008, *p*=.960) and presence of a light-drinking confederate (β=-.084, *p*=.578) were not significantly associated with *ad lib* drinking.

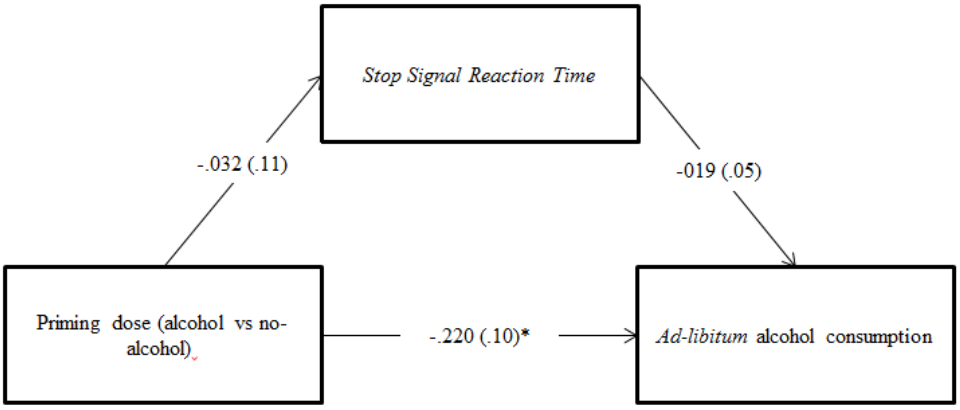

Figure 9. Mediation model of the indirect effect of priming dose on ad lib alcohol consumption via inhibitory control. Values are regression coefficients and standard errors (*p=.033).

7.4.3 *Indirect effect of acute alcohol consumption on ad lib drinking via inhibition errors and go reaction times*

Two additional mediation analyses were conducted with inhibition errors and go reaction times as mediators. The same pattern of results was found with a significant direct effect of priming dose on *ad lib* drinking when inhibition errors (β=-.220, *p*=.033) and go reaction times were (β=-.222, *p*=.035) entered as mediators. There was no association between priming dose and inhibition errors (α path; β=-.019, *p*=.865), and no association between inhibition errors and *ad lib* consumption (β path; β=-.066, *p*=.203). Indeed, the indirect effect was not significant (*b*=.001, SE= .010, 95%, BCa CI -.015, .026). Similarly, there was no association between priming dose and go reaction times (α path; β=-.084, *p*=.448), and no association between go reaction times and *ad lib* consumption (β path; β=-.023, *p*=.664). The indirect effect was not significant (*b*=.002, SE= .008, 95%, BCa CI -.008, .034). The findings of the total effects models produced the same pattern of results as the model obtained from the SSRT analysis.

7.5 Discussion

This secondary analysis aimed to test the indirect effect of acute alcohol consumption on *ad lib* consumption via alcohol-induced impairments of inhibitory control. Due to inconsistent findings in the current literature and those of the previous studies of this thesis, it was predicted that this mediation would not be significant. The current findings support this hypothesis. There was no indirect effect of acute alcohol consumption on *ad lib* alcohol consumption through SSRTs, inhibitory errors or go reaction times. In addition, there was no effect of alcohol consumption on any of these measures of inhibitory control. However, there was a significant direct effect of acute alcohol consumption on *ad lib drinking*, indicative of an alcohol priming effect.

This finding is contrary to previous research suggesting alcohol to impair performance on a SST (Bartholow et al, 2018; Caswell et al, 2013; de Wit et al, 2000; Gan et al, 2014; Mulvihill et al, 1997; Reynolds et al, 2006) and supports previous research which has found no effect of acute alcohol consumption on SST performance (Loeber and Duka, 2009; Baines et al, 2019). Importantly, alcohol consumption was found to be increased following an acute dose of alcohol and this was found in the absence of an effect of alcohol on inhibition and an association between inhibition and *ad lib* alcohol consumption.

These findings suggest that the current literature overstates the effect of alcohol on inhibition and that alcohol-induced impairments of inhibitory control do not underlie the alcohol priming effect (Field et al, 2010; Weafer & Fillmore, 2008). However, this analysis has a number of limitations. Firstly, *ad lib* alcohol consumption was standardized using z-scores; this was due to differences in the *ad lib* procedures, the amount of alcohol and type of alcohol on offer within each of the studies. Secondly, while this secondary analysis offers strong evidence that alcohol-induced impairments of inhibitory control do not underlie the alcohol priming effect, the effect of alcohol on inhibition was assessed only during the ascending limb of the blood alcohol curve. Indeed, recent research that has suggested SST performance to be impaired on the descending limb only (Bartholow et al, 2018), therefore, future studies should assess alcohol-induced SST performance and its association with the alcohol priming effect on the descending limb.

In conclusion, this secondary analysis provided increased power to assess the indirect effect of acute alcohol consumption on subsequent drinking through inhibitory control. This mediating effect was not significant for SSRTs, inhibition errors or go reaction times. These measures were also not related to *ad lib* alcohol consumption. However, there was evidence of an alcohol priming effect with greater *ad lib* consumption following alcohol relative to no-alcohol. These findings suggest that the alcohol priming effect is not underwritten by alcohol-induced impairments of inhibitory control.

8 Chapter 8 General Discussion

The primary aim of the current thesis was to investigate moderators of the alcohol priming effect. Two novel moderators of alcohol priming were assessed; beliefs regarding acute alcohol effects and social influence. In addition, this thesis aimed to further clarify the role of alcohol-induced impairments of inhibitory control in the alcohol priming effect. Within this chapter, the main findings are summarised and theoretical and practical implications are discussed. The methodological strengths and weaknesses of this thesis are then considered followed by directions for future research.

8.1 Results summary

The first study of this thesis (chapter 3) cross-sectionally investigated the associations between peer and personal heavy episodic drinking and whether this association was moderated by self-reported overall and affective drinking-induced disinhibition, urgency, and trait self-control. A positive association between peer and personal heavy episodic drinking was found, and this association was strongest amongst individuals high in urgency but low in affective drinking-induced disinhibition. Furthermore, individuals high in affective drinking-induced disinhibition but low in urgency did not differ from any other combination of these traits. This finding suggests that individuals high in urgency, but low in affective disinhibition may be more likely to drink similar to their friends. This may occur as they attribute valence onto social stimuli meaning that other people's drinking influences personal alcohol consumption. On the other hand, those with high affective disinhibition/low urgency may be more likely to attribute valence to alcohol-related cues and so are influenced by their peers to a lesser extent. This suggests that drinking induced disinhibition underlies subsequent alcohol use (the alcohol priming effect), but that this is also affected by social factors.

The second study of this thesis (chapter 4) aimed to assess the effect of other people's drinking behaviour on the alcohol priming effect more directly. Specifically, given the apparent strong effect of peers (Quigley & Collins, 1999), this study assessed whether the alcohol priming effect is mitigated by the presence of a light-drinking confederate. To do this, friendship dyads were recruited, one was allocated to be a confederate and the other the participant. A priming dose of alcohol or

placebo was administered and then the participant took part in an *ad lib* drinking session either in isolation or in the presence of the confederate who had been instructed to drink lightly. Overall, no effect of confederate was observed on *ad lib* alcohol consumption and, while the priming dose of alcohol increased craving, there was no effect of acute alcohol consumption on *ad lib* drinking. However, exploratory analysis of sex differences revealed that males consumed more alcohol and ordered more drinks when in the presence of a confederate than when alone. This suggested that male alcohol consumption is elevated when in the presence of others.

Building on the second study, study 3 (chapter 5) investigated the effect of alcohol on imitation of heavy and light-drinking confederate friends. Several methodological issues that were encountered within study 2 were addressed. This included the use of a control drink rather than a placebo and increased standardisation of the friendship dyads with mixed-sex pairs being used throughout, with the male allocated as the participant. Participants attended two sessions, consuming a priming dose of alcohol in one session and a control drink in the other. Following this, participants completed an *ad lib* drinking session in which their friend either drank heavily or lightly. Findings suggested an alcohol priming effect on craving but not on *ad lib* alcohol consumption. In addition, participants consumed more alcohol when exposed to the heavy-drinking confederate relative to the light-drinking confederate.

Study's 4 and 5 (chapter 6) investigated the effect of beliefs about alcohol's acute effects on alcohol priming and alcohol-induced impairments of inhibitory control. Specifically, Study 4 investigated the effect of self-control beliefs on alcohol consumption. To do this, participants consumed an acute dose of alcohol or a placebo before completing an SST which ostensibly assessed their self-control following alcohol consumption. They were then provided with bogus feedback suggesting they had either high or average self-control. According to the manipulation check, this was not successful and there was no subsequent effect of this manipulation was found on *ad lib* consumption. Within this study, craving was greater within alcohol relative to placebo sessions, regardless of time. Study 5 addressed this by providing a more explicit manipulation which involved exposing participants to a neutral or an experimental script which suggested that acute doses of

alcohol reduce the urge to drink. This occurred prior to the completion of an SST so that the effect of such beliefs on alcohol-induced inhibitory impairments could also be assessed. Results suggested no effect of beliefs on *ad lib* consumption, although craving was greater overall in alcohol sessions, but did find the experimental message to lead to alcohol-induced impairments of inhibitory control while the neutral message did not.

The final study of this thesis (study 6; chapter 7) aimed to further investigate whether alcohol-induced impairments of inhibition mediate the alcohol priming effect. Study's 2, 3, 4 and 5 all contained an SST following an acute dose of alcohol and an *ad lib* drinking session. The data from all of these studies were collated and standardised. This was done in order to provide increased power to detect mediation. While there was found to be a direct effect of acute alcohol consumption on *ad lib* drinking this effect was not mediated by alcohol-induced impairments of inhibitory control.

8.2 Theoretical and methodological implications

The studies in this thesis have demonstrated an alcohol priming effect on craving. Within all study's which assessed craving (study's 2-5), there was either an increase relative to baseline (Study's 2 and 3) or greater overall craving within alcohol sessions (study's 4 and 5). Identifying the effect of alcohol on craving was dependent on whether a placebo or a control drink was used. For example, on occasions when a placebo comparison was used, the increase in craving was found to be greater relative to placebo within study 2 but not within study's 4 and 5. When a control drink comparison was used (study 3) craving did increase to a greater extent following alcohol, while craving decreased following the control drink. These findings support previous research that has demonstrated alcohol to increase craving (Christiansen et al, 2013; Christiansen et al, 2016), but also research which has shown the anticipated effects of alcohol (manipulated through placebo administration) to increase craving (e.g. Christiansen et al, 2013; de Wit, 1996; de Wit & Chutuape, 1993; Hodgson et al, 1979; Ludwig et al, 1974). Indeed, the alcohol-control comparisons more accurately reflect real-world drinking as both the pharmacological and expectancy effects of alcohol are assessed.

There is also some, albeit inconsistent, evidence for an alcohol priming effect on *ad lib* alcohol consumption. Study 4 demonstrated a clear effect of acute alcohol consumption on *ad lib* drinking with alcohol consumption being greater, following alcohol relative to placebo. However, using the same design, this was not apparent for study 5. In addition, there was no alcohol priming effect found on the volume of alcohol consumed during an *ad lib* session within study 2 or 3, but when sex differences were considered, there was an effect of acute alcohol consumption on ordering additional drinks within study 2. Synthesising data from all these studies (study 6) revealed there to be an overall significant, albeit small, increase in alcohol consumed following acute alcohol consumption relative to no alcohol consumption (both placebo and control drink).

Overall, these findings support the notion that acute doses of alcohol can lead to increases in both craving for alcohol and *ad lib* alcohol consumption and this effect is dependent on whether a control drink or placebo is used as a comparison. Consistent with previous work which has demonstrated the anticipated effects of alcohol to increase craving and *ad lib* drinking, these findings suggest that both the pharmacological and anticipated effects of alcohol are important factors underlying alcohol priming. However, the effect of alcohol on *ad lib* consumption was inconsistent within this thesis and may be relatively small. The current literature may therefore currently exaggerate the effect of acute doses of alcohol on *ad lib* drinking.

The studies within this thesis also aimed to assess whether this alcohol priming effect is underwritten by alcohol-induced impairments of inhibitory control. However, I found no evidence that priming doses of alcohol leads to impaired inhibition. There was no effect of alcohol on SSRT, inhibition errors or go reaction times within study 2, 3 or 4. These measures were also found not to be correlated with *ad lib* alcohol consumption in any study. Due to this, the proposed mediation analyses were not conducted within any study, as there was no direct IV-mediator and/or mediator-DV associations; a necessary criterion for mediation. The synthesised analysis (study 6) found no effect of priming dose (alcohol vs no-alcohol) on SSRT, inhibition or go reaction times and no association with any of these measures and *ad lib* alcohol consumption. Therefore, there was also no indirect effect of priming dose on *ad lib* drinking via these measures.

Taken together, these findings strongly suggest there to be no effect of alcohol on inhibitory control, no association between alcohol-induced impairments of inhibitory control and no mediating effect of such impairments. This contrasts with widely cited findings and models of the alcohol priming effect that implicate inhibitory control (e.g. Field et al, 2010; Weafer & Fillmore, 2008). But is consistent with several studies which have failed to detect an effect of alcohol on inhibition and have not found inhibitory impairments to mediate the alcohol priming effect (Baines et al, 2019; Christiansen et al, 2013). The lack of an effect of acute alcohol consumption on inhibition is surprising given the amount of research which suggests that it is reliably impaired at the same and comparable doses at that used within this thesis (Bartholow et al, 2018; Caswell et al, 2013; de Wit et al, 2000; Gan et al, 2014; Mulvihill et al, 1997; Reynolds et al, 2006). This further suggests that the effect of alcohol on inhibition has been exaggerated within the literature.

The role of beliefs regarding the acute effects of alcohol was also assessed as a potential moderator of the alcohol priming effect. In studies 4 and 5 beliefs about the effects of alcohol on self-regulation were manipulated. This manipulation was seemingly not successful within study 4 but seemed to show some effect on an SST in a subsequent session. The manipulation was, however, successful within study 5. This study demonstrated that the belief that alcohol would not impair subsequent regulation led to alcohol-induced impairment of inhibitory control. Contrary to expectations, this did not affect alcohol consumption in or outside of the lab. In combination, these studies suggest that alcohol-induced impairments of inhibitory control occur only when individuals do not expect subsequent impairment. This may reflect mitigated compensatory effects i.e. those led to believe their self-regulation will be unimpaired may be less likely to attempt to overcome the effect of alcohol on inhibition, resulting in poorer performance. These findings are consistent with previous work which has demonstrated compensatory effects on a variety of tasks including the SST (Fillmore et al, 1994; Fillmore & Blackburn, 2002). It is currently not known to what extent these beliefs differ across samples. Inconsistent findings within the literature may, therefore, reflect differences in beliefs about the acute effects of alcohol across different samples and the extent to which participants compensate for these impairments. For example, it is possible that lighter drinkers compensate to a

greater degree than heavier drinkers, this would lead to improved performance on an SST relative to heavier drinkers and may explain previously noted associations between inhibitory impairments and alcohol consumption, following a priming dose (e.g. Weafer & Fillmore, 2008).

It is important to note that previous research has shown manipulation of beliefs to affect subsequent alcohol consumption. For example, Jones et al (2012) found participants led to believe they had high levels of restraint to drink more alcohol during a bogus taste test than those led to believe they had low levels of restraint. However, the studies in this thesis differ from this study in a number of ways which may explain these inconsistent findings. Firstly, Jones et al (2012) included only high and low conditions and did not include a control, neutral or average condition. It is, therefore, not possible to elucidate the direction of the effect within their study. The lack of an effect within the current studies may, therefore, be due to the lack of a condition wherein participants were led to believe their self-control was low. In addition, in the case of study 4, Jones et al (2012) included additional measures to increase the believability of the manipulation which were not included within the current studies. Finally, this previous study did not include an alcohol prime and the manipulation pertained to trait restraint rather than supposed changes due to alcohol's effects.

This thesis also investigated the potential role of other people's drinking on the alcohol priming effect. While previous research has demonstrated that individuals' alcohol consumption is affected by both descriptive norms (e.g. Jones-Webb et al., 1997; Larimer et al., 2004; Lee et al., 2012; Robinson et al., 2014b) as well as modelling of confederate (Larsen et al., 2009; Larsen, Engels, et al., 2010; Quigley & Collins, 1999; Robinson et al., 2016) and friend's (Dallas et al, 2014) alcohol consumption, the effect of acute alcohol consumption on these social influences has not been previously investigated. This is particularly important as both the alcohol priming effect (e.g. Field et al, 2010; Weafer and Fillmore, 2008) and social influences (e.g. Borsari & Carey, 2001; Borsari & Carey, 2003; Jones-Webb et al., 1997; Larimer et al., 2004; Lee et al., 2012; Robinson et al., 2014; Robinson et al., 2015) have been implicated as mechanisms which may underlie binge drinking. Furthermore, binge drinking most commonly occurs with others (Ally et al, 2016), yet the interaction between these two factors and their relative importance had not been investigated.

Study 1 (chapter 3) demonstrated that affective facets of drinking-induced disinhibition interact with urgency traits to affect the extent to which individuals drink similar to their peers. This finding suggests that the acute effects of alcohol can influence the effect of other's drinking on consumption. However, the findings of studies 2 and 3, which directly assessed the effect of acute alcohol consumption and peer drinking, offer conflicting evidence. Within these studies, a priming dose of alcohol increased craving for alcohol but did not lead to increased consumption. In addition, alcohol consumption was unaffected by the presence of a light-drinking peer relative to isolation (study 2), although when sex differences were assessed, males consumed more alcohol in the presence of this peer than when alone. In study 3 (again, using a male sample) alcohol consumption was elevated in the presence of a heavy-drinking peer relative to a light-drinking peer. These findings suggest that, at least for males, alcohol consumption is elevated when in the presence of a friend regardless of the amount that they consume, but consumption may increase further when exposed to a heavy-drinking peer. Importantly this effect remained consistent following consumption of alcohol, placebo (study 2) or a control drink (study 3) and was present despite alcohol-induced increases in craving. Taken together, these findings suggest that social factors are a more important determinant of drinking behaviour than the alcohol priming effect and alcohol-induced impairments of inhibitory control.

This finding supports previous research that has suggested other people's drinking exerts a strong effect on alcohol consumption and the current findings suggest this effect is maintained regardless of whether alcohol had been previously consumed or not. However, while imitation of other's drinking may be important when in the presence of others it is possible that the alcohol priming effect is more important for individuals when they are alone. In addition, while the findings of studies 2 and 3 seem to contradict that of study 1, it may be the case that acute alcohol consumption affects different types of social influences in distinct ways. For example, while acute doses of alcohol, and resultant disinhibition, may not affect direct imitation of alcohol consumption it is possible that the disinhibiting effects of acute alcohol consumption may moderate the influence of descriptive

norms on drinking. Indeed, the descriptive norm measures in study 1 referred to the number of total drinks consumed within a drinking session rather than imitation of alcohol consumption *per se*.

Within study 1, it was initially hypothesised that urgency would moderate the association between peer and personal drinking when drinking-induced disinhibition was high. However, the inverse was found to be the case, with urgency moderating this effect only when drinking-induced disinhibition was low. This contradictory finding adds to the current literature which has sought to investigate mechanisms underlying the association between urgency and alcohol consumption. For example, previous theoretical suggestions have posited that individuals high in urgency are more likely to employ an affect heuristic wherein decisions are based on their perceived valence (Stautz & Cooper, 2014a). Consistent with this, the findings of study 1 suggest that individuals high in urgency, but low in affective drinking-induced disinhibition, drink more like their friends as social cues become attributed with positive valence. However, for those who concurrently experience high levels of affective drinking-induced disinhibition the valence attributed to social cues is disrupted, perhaps by increased valence towards alcohol-related cues.

These studies investigating the effect of other's drinking have also contributed to the current literature through the development of novel methodological approaches. Within study's 2 and 3 a novel method of using pre-existing friendship dyads were used to assess the effect of confederate drinking. While this method was based on previous research (Dallas et al, 2014) it was adapted to allow the assessment of alcohol volume rather than simply choice. Currently, most confederate paradigms use research assistants, this methodology may, therefore, offer a simpler and more cost-effective method of assessing imitation of alcohol consumption. Indeed, study 4 demonstrates the construct validity of this method. Secondly, study 4 used a novel debriefing procedure which involved presenting a fake debrief sheet to participants prior to questioning them regarding their awareness of the study's aims. This method may prove useful for future research for which deceit is particularly crucial.

Taken together, current models of alcohol priming require considerable adjustment in light of these findings. Future models should take into account the inconsistent effects of alcohol on inhibition

and the lack of evidence that such impairments mediate the alcohol priming effect. Furthermore, although there is some evidence to suggest that drinking-induced disinhibition, along with urgency, moderate social influences, and these findings suggest that the alcohol priming effect may not be an important determinant of hazardous drinking behaviours such as binge drinking.

8.3 Clinical implications and interventions

As well as these theoretical implications, the findings of this thesis also present some clinical implications. These studies have demonstrated that acute doses of alcohol can increase craving and, to a lesser extent, *ad lib* alcohol consumption among social drinkers. This suggests that current advice to consume less than 14 units of alcohol per week (HSCIC, 2016) may prove difficult for people to adhere to. Indeed, if individuals intend to consume 14 units of alcohol per week over one or two drinking sessions, the alcohol priming effect may mean that controlled drinking becomes difficult, so they would be likely to violate intentions to drink moderately. Increasing awareness of the alcohol priming effect alongside the delivery of such information may be useful. This may allow individuals to develop effective strategies to maintain their drinking at their desired level. For example, someone who has consumed some alcohol units already in the week, but wishes to drink, may instead opt to abstain as they are aware that they may become 'primed' and exceed the government (or their self-imposed) guidelines.

Secondly, there is a growing literature which has investigated whether inhibitory control can be improved via training. This inhibitory control training (ICT) attempts to either improve individual's capacity to successfully inhibit responses or to associate alcohol-related stimuli with inhibition, with the assumption that improved inhibition will lead to reductions in alcohol consumption (or other unhealthy behaviours). Currently, there are mixed-findings regarding the effectiveness of ICT with some work suggesting small effects of training (Allom, Mullan, & Hagger, 2016) and others reporting null findings (Jones et al., 2018). Indeed, the findings from this thesis suggest that such interventions are unlikely to be effective, as inhibitory control was not related to alcohol consumption following consumption of an initial drink. Therefore, even if such interventions prevent initiation of drinking, following an initial drink they are unlikely to be effective.

Alcohol consumption was influenced by other's drinking even following acute alcohol consumption. This suggests that interventions which reduce imitation are likely to be effective. Some previous interventions have targeted perceived peer drinking. These interventions aim to correct exaggerated perceptions of other's drinking which theoretically should decrease personal drinking (Prestwich et al., 2016). However, there are inconsistent findings with this approach, with the most hazardous drinkers often unaffected by such interventions (Reid & Carey, 2015). Other research has attempted to mitigate direct imitation of alcohol consumption by increasing individual's self-affirmation, but this has not been found to be effective (Reid, Field, Jones, DiLemma, & Robinson). Consistent with previous research, study 1 found urgency to underlie the association between self-reported peer and personal drinking. (Stautz & Cooper, 2014b) In addition, urgency has been shown to underlie susceptibility to social influences (Stautz and Cooper, 2014a). Therefore; urgency may be a valid target for intervention. Indeed, a number of interventions such as dialectical behaviour therapy (DBT; Robins & Chapman, 2004) and integrated cognitive-affective therapy (ICAT; Wonderlich et al., 2014) have been developed to address high levels of urgency, and focus on developing skills to manage high levels of affect. Such interventions may prove effective in mitigating the effect of social influences on alcohol consumption. Importantly, as studies 2 and 3 found social influence to be unaffected by a priming dose, it is likely that effective interventions which target the effect of imitation and norms on alcohol consumption are unlikely to be mitigated by acute alcohol intoxication.

8.4 Limitations

The findings in this thesis must be considered in light of several methodological limitations. Firstly, only immediate alcohol consumption was assessed throughout this thesis, the impact of priming over a longer drinking session is therefore not known. Indeed, the alcohol priming effect may be particularly important in driving the decision to continue drinking rather than the volume of alcohol which is consumed. *Ad lib* alcohol consumption paradigms may, therefore, not be the best method to assess priming. This is a particularly salient point in concern with the bogus taste test, for which continued alcohol consumption is required. Indeed, all *ad lib* procedures took place over a

small amount of time (10-30 minutes) which does not accurately reflect 'real-world' drinking occasions. Furthermore, the priming effect may be a more important determinant of continued alcohol consumption among individuals who are not motivated to drink. As these studies had a preponderance of student participants, who often drink heavily (Davoren et al., 2016) and for whom there may be little motivation to restrain drinking, it is possible that the full extent of the alcohol priming effect was masked within these studies. For example, study's 2 and 3 found no evidence of a priming effect on *ad lib* alcohol consumption using a predominately student sample but did find social influences to be important. This strong peer effect, across three studies, may also have been bolstered by the student sample, for which social approval may be particularly important.

All of these studies used the same dose of alcohol (0.50g/kg) this was done in order to maintain consistency across studies and also because the effect of alcohol on inhibition and *ad lib* drinking has been previously demonstrated at these, and lower, doses. Using this moderate dose of alcohol also allowed the administration of further alcohol during *ad lib* sessions while remaining within limits imposed by the ethics committee. However, a 0.50g/kg dose is relatively lower than doses of alcohol that may be consumed in the 'real-world'.

A further limitation is the latency at which the SST was administered across studies. As discussed, acute alcohol effects are biphasic with stimulant effects present on the ascending limb of the blood alcohol curve and sedative effects occurring during the descending limb (Earleywine & Erblich, 1996). Throughout this thesis, the SST was administered on the ascending limb of this blood alcohol curve. However, research published during the course of the thesis suggests that the effect of alcohol consumption on SST performance may only be apparent on the descending limb (Bartholow et al, 2018). In addition, no study included a baseline SST to account for individual differences in performance. However, this decision was made in order to limit the influence of practice effects on this task, which the SST may be particularly sensitive to (Huizenga, van der Molen, Bexkens, Bos, & van den Wildenberg, 2012).

It is also not known to what extent the current findings can be generalised to other measures of inhibitory control. Indeed, many studies which have found an effect of alcohol on inhibition have

used alternative tasks such as the cued go/no-go (GNG) task (Weafer & Fillmore, 2008). Importantly, GNG and SST's may measure distinct components of inhibition, with SST performance reflecting action cancellation and the GNG (and related) tasks assessing action restraint (Littman & Takács, 2017). These tasks have also been linked to activation in different neural networks (Swick et al, 2011) and the release of different neurotransmitters (Eagle et al, 2008; 2009). However, there are a number of issues with using a GNG task. They are often prone to ceiling effects (Barch, Braver, Carter, Poldrack, & Robbins, 2009) and are a less direct measure of inhibitory control than SSTs, from which SSRT can be computed. There is also research suggesting the SST to be a more sensitive measure of impaired inhibition among non-dependent drinkers than the GNG task (Smith et al, 2014). In addition, although the evidence base for the cued GNG task appears convincing there has been a failure to replicate these findings beyond a single research group. I would argue, therefore, that the use of an SST is preferable to using a GNG task, or its variants.

Recent theoretical frameworks of inhibitory control have posited that it consists of a number of different subcomponents (Verbruggen, McLaren, & Chambers, 2014). For example, SSRTs are also the result of effective signal detection, and individuals also have the capacity to plan their responses and implement strategies to enhance their performance - referred to as 'proactive control'. The studies within this thesis did not assess these underlying mechanisms of inhibition. Indeed, it is possible that alcohol affects these processes to varying degrees. It is also likely that the compensatory effects observed within study 5 are the result of changes in proactive rather than reactive control (SSRTs, but it is not possible to examine this using the SST which was employed. It is, however, important to note that recent research has demonstrated alcohol (0.60g/kg) to have no effect on signal detection, proactive or reactive control (Baines et al, 2019).

In addition, Field et al (2010) suggested that inhibitory control exerts a top-down control on automatic responses to alcohol cues. It could be suggested that disinhibition does not directly affect alcohol-seeking but exerts an indirect effect only via automatic responses. However, these automatic responses were not assessed within this thesis. However, this is unlikely to be the case given that no effect of acute alcohol consumption was found on inhibition.

All experimental studies were conducted in semi-naturalistic laboratories, either a bar or lounge lab. While this increased ecological validity is a strength of the current thesis, these environments may be associated with alcohol-consumption the bar-lab contain alcohol-related cues. It is, therefore, possible that these environments exerted an effect on *ad lib* alcohol consumption and inhibition. Indeed, previous research suggests that such environments can lead to impaired inhibitory control (Field & Jones, 2017) and increased *ad lib* alcohol consumption (Moss et al., 2015), although there have been contradictory findings (Christiansen et al, 2017). Rather than a lack of an effect of acute alcohol consumption, the studies within this thesis may instead demonstrate no additive effect of acute consumption on inhibition beyond environmental influences.

There were also a number of limitations for the studies which investigated social influences. The main issue from these studies concerns the observed sex differences. Within study 2, contrary to expectations, male's alcohol consumption increased when exposed to a light-drinking confederate. However, as it was not possible to balance the gender compositions of the dyads, it is unknown whether this is suggestive of a sex difference or exposure to an opposite-sex confederate. Due to this finding, study 3 used a male sample of participants and female confederates only. While this provided increased standardisation it also limits the generalisability of these findings. In addition, within study 3 it is not clear whether *ad lib* alcohol consumption increased as a result of exposure to a heavy-drinking confederate or decreased as a result of the light-drinking confederate. However, study 2 demonstrated increased alcohol consumption in the presence of a light-drinking friend. Indeed, previous research has shown alcohol to affect subjective response to alcohol within social contexts relative to when alone (de Wit & Sayette, 2018). This may underlie the increased alcohol consumption in the presence of a light-drinking confederate within this study. However, this cannot be tested as measures of subjective intoxication were administered prior to the *ad lib* drinking session. Taken together these findings suggest that alcohol consumption is elevated in the presence of a friend but that this increase is greater when the friend drinks heavily.

8.5 Future research

The current findings provide an important contribution to the existing literature concerning mechanisms underlying and interacting with the alcohol priming effect. In particular, they suggest that alcohol-induced impairments of inhibitory control do not underlie the alcohol priming effect. What is clear is that the effect of alcohol on inhibition, and its association with the alcohol priming effect, has been overstated. A meta-analysis assessing the overall effect of alcohol on a range of inhibitory control measures, as well as assessment of potential publication bias and p-curve analysis to assess the extent of 'p-hacking' (Simonsohn, Simmons, & Nelson, 2015) would provide further clarification of the state of the literature. In addition, there remains only one study (Weafer & Fillmore, 2008) that has established a clear association between inhibitory deficits and alcohol consumption. A pre-registered replication of this study may also yield further clarification. Failure to find an effect of alcohol on inhibition using these methods would provide support for abandoning the notion that alcohol leads to impairments of inhibitory control.

However, recent research suggests the effect of alcohol on inhibition may occur only during the descending limb of the blood alcohol curve (Bartholow et al, 2018). These studies could, therefore, be replicated using an SST implemented during this phase. In addition, it is currently not known which component of inhibition is impaired during the descending limb. Future research should, therefore, investigate the effect of acute alcohol consumption on signal detection, proactive and reactive control during the descending limb relative to the ascending limb and whether these impairments mediate alcohol priming.

Considering the current findings, it is possible that beliefs about alcohol's acute effects underlie the effect of alcohol on inhibitory control. Further research should aim to assess whether these beliefs also underlie other measures of inhibitory control and which components (i.e. proactive control) are affected by these beliefs. In addition, individual differences in these beliefs may explain inconsistencies in the literature. A study investigating whether alcohol outcome expectancies regarding cognitive control moderate the effect of alcohol on inhibitory control would provide further clarity.

Two studies in this thesis (studies 2 and 3) were the first to directly assess the effect of social influences on alcohol consumption following acute alcohol administration. Further research should build on these findings by using dyads adequately balanced by sex. Indeed, previous research has found the sex composition of confederate dyads to exert no differential effect on alcohol consumption (Larsen et al, 2010). However, as demonstrated in study 2, following acute alcohol consumption this effect may differ. The mechanisms underlying these potentially alcohol-induced sex differences could also be explored. Furthermore, while study 2 and 3 taken together suggest that individuals increase their alcohol consumption in the presence of a heavy-drinking confederate the direction of this effect is not yet clear. Subsequent research could, therefore, employ a light-drinking, heavy-drinking and isolation condition within the same study so that the direction of this effect can be properly elucidated.

8.6 Concluding comments

This thesis has investigated mediators and moderators of the alcohol priming effect. The overarching aim of this thesis was to assess whether alcohol-induced impairments of inhibitory control mediate alcohol priming. In addition, the role of two novel moderators, beliefs about the effects of acute alcohol consumption and social influences, were assessed. Overall, inhibitory control was found not to be affected by acute alcohol consumption and not to mediate the alcohol priming effect. However, the belief that alcohol would not impair self-regulation was found to moderate the effect of alcohol on inhibition. Further studies investigated the interaction between acute alcohol consumption and social influences on the alcohol priming effect. Findings suggest that the alcohol priming effect exerts little effect on drinking when in the presence of others and that alcohol consumption is increased when exposed to a heavy-drinking peer regardless of whether an acute dose of alcohol is consumed or not. The association between self-reported peer and personal alcohol consumption was, however, moderated by urgency and only when affective-drinking induced disinhibition was low. Taken together, these findings suggest that the importance of the alcohol priming effect as a determinant of hazardous drinking behaviours, such as binge drinking, is minimal. The alcohol priming literature and theoretical models require considerable adjustment in light of these findings.

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10 Appendices

10.1 Appendix 1: Alcohol use disorders identification test (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

10.2 Appendix 2: Timeline follow back (TLFB)

Timeline Followback

To help me evaluate your drinking I need to get an idea of your alcohol consumption in the past fourteen 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?

The list below shows the number of units of alcohol in common drinks:-

- A pint of ordinary strength lager (Carling Black Label, Fosters) - 2 units
- A pint of strong lager (Stella Artois, Kronenbourg 1664) - 3 units
- A pint of ordinary bitter (John Smith's, Boddingtons) - 2 units
- A pint of best bitter (Fuller's ESB, Young's Special) - 3 units
- A pint of ordinary strength cider (Woodpecker) - 2 units
- A pint of strong cider (Dry Blackthorn, Strongbow) - 3 units
- A 175ml glass of red or white wine - around 2 units
- A 750ml bottle of red or white wine – around 9 units
- A pub measure of spirits - 1 unit
- An alcopop (eg Smirnoff Ice, Bacardi Breezer, WKD, Reef) - around 1.5 units

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. Please double check that you have filled in the number of units for all fourteen days.

Last week:

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

Previous week:

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday

10.3 Appendix 3: Leeds Dependence Questionnaire (LDQ)

Leeds Dependence Questionnaire - LDQ

Here are some questions about the importance of alcohol or other drugs in your life. Think about the main substance you have been using over the **last 4 weeks** and tick the closest answer to how you see yourself

	Never 0	Sometimes 1	Often 2	Nearly Always 3
Do you find yourself thinking about when you will next be able to have another drink or take more drugs?	☐	☐	☐	☐
Is drinking or taking drugs more important than anything else you might do during the day?	☐	☐	☐	☐
Do you feel that your need for drink or drugs is too strong to control?	☐	☐	☐	☐
Do you plan your days around getting and taking drink or drugs?	☐	☐	☐	☐
Do you drink or take drugs in a particular way in order to increase the effect it gives you?	☐	☐	☐	☐
Do you drink or take drugs morning, afternoon and evening?	☐	☐	☐	☐
Do you feel you have to carry on drinking or taking drugs once you have started?	☐	☐	☐	☐
Is getting an effect more important than the particular drink or drug you use?	☐	☐	☐	☐
Do you want to take more drink or drugs when the effects start to wear off?	☐	☐	☐	☐
Do you find it difficult to cope with life without drink or drugs?	☐	☐	☐	☐

10.4 Appendix 4: Drinking induced disinhibition scale (DIDS)

Drinking-Induced Disinhibition Scale (DIDS)

Please indicate on the scale below how likely you are to experience each of the following occurrences either while drinking or as a direct results of consuming alcohol. As a point of reference, items rated as '5' or '6' should be things you often experience during the course of drinking or as a consequence of alcohol consumption, items rated '3' or '4' should happen from time to time and those marked with a '1' or '2' should be things you either never or have very rarely experienced in conjunction with drinking and have little or no intention of experiencing again.

How likely are you to experience each of these occurrences either while drinking or as a direct results of consuming alcohol? Please rate each occurrence according to a typical drinking experience or you.

Acting more friendly or outgoing around others than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Expressing more disappointment in yourself or others than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Engaging in casual, consensual sex with someone who you are not dating

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Expressing more optimism than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely

Expressing stronger feelings of sadness than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Engaging in consensual sex acts that you would be less likely to take part in when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Greater feelings of personal freedom than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Feeling more depressed than when not drinking

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

Hooking up with someone who you are not dating

1 2 3 4 5 6

(Highly Unlikely) (Highly Likely)

10.5 Appendix 5: Desire for alcohol questionnaire (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

10.6 Appendix 6: Subjective intoxication scales (SIS)

Subjective effects 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

_____

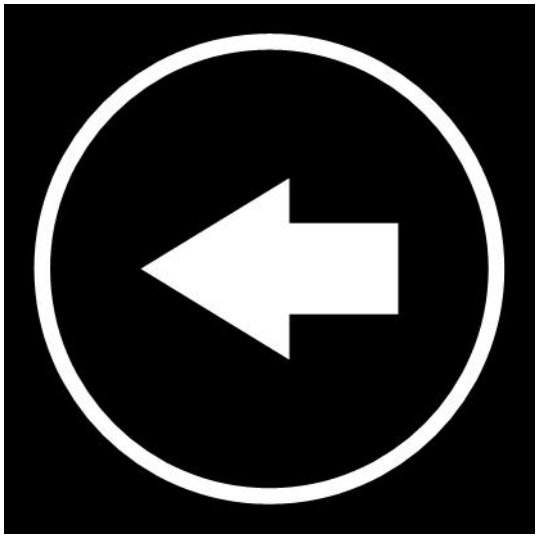
Not at all Slightly Moderately Quite a lot Extremely

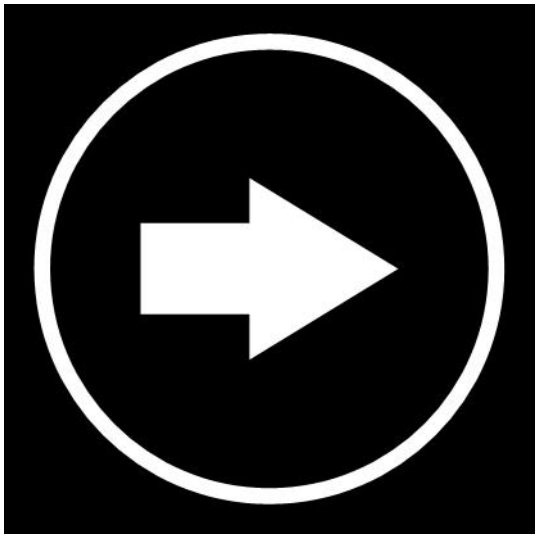
Contented

_____

Not at all Slightly Moderately Quite a lot Extremely

10.7 Appendix 7: Stop signal task stimuli





10.8 Appendix 8 : SUPPS-P

SUPPS-P

Below are a number of statements that describe ways in which people act and think. For each statement, please indicate how much you agree or disagree with the statement. If you Agree Strongly circle 1, if you Agree Somewhat circle 2, if you Disagree somewhat circle 3, and if you Disagree Strongly circle 4. Be sure to indicate your agreement or disagreement for every statement below. Also, there are questions on the following pages.

		Agree	Agree some	Disagree some	Disagree Strongly
1	I generally like to see things through to the end	1	2	3	4
2	My thinking is usually careful and purposeful.	1	2	3	4
3	When I am in a great mood, I tend to get into situations that could cause me problems.	1	2	3	4
4	Unfinished tasks really bother me.	1	2	3	4
5	I like to stop and think things over before I do them.	1	2	3	4
6	When I feel bad, I will often do things I later regret in order to make myself feel better now.	1	2	3	4
7	Once I get going on something I hate to stop.	1	2	3	4
8	Sometimes when I feel bad, I can’t seem to stop what I am doing even though it is making me feel worse.	1	2	3	4
9	I quite enjoy taking risks	1	2	3	4
10	I tend to lose control when I am in a great mood.	1	2	3	4
11	I finish what I start.	1	2	3	4
12	I tend to value and follow a rational, ‘sensible’ approach to things.	1	2	3	4
13	When I am upset I often act without thinking.	1	2	3	4

14	I welcome new and exciting experiences and sensations, even if they are a little frightening and unconventional.	1	2	3	4
15	When I feel rejected, I will often say things I later regret.	1	2	3	4
16	I would like to learn to fly an airplane	1	2	3	4
17	Others are shocked or worried about the things I do when I am feeling very excited.	1	2	3	4
18	I would enjoy the sensation of skiing very fast down a high mountain slope.	1	2	3	4
19	I usually think carefully before doing anything.	1	2	3	4
20		1	2	3	4

10.9 Appendix 9: Awareness (study 3) participant specific

What do believe the aims of the study to be?

10.10 Appendix 10: Friend questions (study 2)

How do you and the other participant know each other?

How long have you known each other?

Do you believe the other participant to be your friend? Please circle response.

Strongly Disagree Disagree Unsure Agree Strongly Agree

10.11 Appendix 11: Unit estimate- Priming drink

Estimate how many standard 25ml shots of Vodka you consumed at the beginning of the study

0_____1_____2_____3_____4_____5_____6_____7_____8_____9+

10.12 Appendix 12: Awareness (study 2)

What do believe the aims of the study to be?

Do you think the other participant behaved normally?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If you disagree, why not?

Did you notice the drinking of the other participant during the comedy programme?

Yes No

Would you say the amount the other participant drank, during the comedy programme, was odd?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Would you say the amount that other participant drank, during the comedy programme, influenced how much you drank?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If so, why?

If not, why not?

Would you normally drink the drink you were given while watching the comedy programme?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Did you like the drink you were given while you were watching the comedy programme?

Strongly Disagree Disagree Unsure Agree Strongly Agree

10.13 Appendix 13: Waiver

I have been informed by the experimenter that I am intoxicated and should remain in the laboratory but am willing to leave at my own risk

Name_____

Date_____

Signature_____

10.14 Appendix 14: Awareness (study 3) participant specific

What do believe the aims of the study to be?

Do you think the other participant behaved normally?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If you disagree, why not?

Did you notice the drinking of the other participant during the comedy programme?

Yes No

Would you say the amount the other participant drank, during the comedy programme, was odd?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Would you say the amount that other participant drank, during the comedy programme, influenced how much you drank?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If so, why?

If not, why not?

Would you normally drink the drink you were given while watching the comedy programme?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Did you like the drink you were given while you were watching the comedy programme?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Would you normally drink the drink you were given while watching TV?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Did you like the drink you were given while you were watching TV?

Strongly Disagree Disagree Unsure Agree Strongly Agree

How many standard 25ml shots of Vodka do you think were contained in the drink you were given, while watching TV, in session 1?

0_____1_____2_____3_____4_____5_____6_____7_____8_____9+

How many standard 25ml shots of Vodka do you think were contained in the drink you were given, while watching TV, in session 2?

0_____1_____2_____3_____4_____5_____6_____7_____8_____9+

Do you think the other participant behaved normally in session1?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If you disagree, why not?

Do you think the other participant behaved normally in session 2?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If you disagree, why not?

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PARTICIPANT DEBRIEFING INFORMATION

Study Title: **Effect of alcohol and personality on ability to withhold information**

Thank you for participating in this study

What was the study about?

The aim of this study was to investigate the ability of individuals to withhold information from friends. We were also interested to see if the extent to which people are affected by alcohol affects the ability to withhold information. We also examined the relationship between ability to withhold information and other personality traits.

In the first session your friend was told to perform an action which would make sense given the context of the experiment. We hypothesised that those people affected more by alcohol would reveal this information to you during the gap between that first session and this one.

We hope that the findings of this research will further our understanding of the role alcohol plays in withholding information and social interaction.

What if I want advice about drinking, or help with reducing my drinking?

We are not qualified to offer advice ourselves, but if you are concerned about your drinking, and would like help giving up, we advise you to seek information and advice from your Doctor, by calling Drinkline on 0800 917 82 82, or from one of the following websites:

www.drinkaware.co.uk

www.nhs.uk/Change4Life/Pages/drink-less-alcohol

Who can I contact if I have further questions?

If you have any questions then please contact the principle investigator:

Dr Paul Christiansen

2.25, Eleanor Rathbone Building

University of Liverpool,

Liverpool, L69 7ZA,

UK

e: prc@liverpool.ac.uk

Tel: 0151 794 695

What do you think your friend was asked to do/told?

Why do you think this?

Has your friend revealed any information to you about this study since the last session?

Yes No

If yes, what did they reveal?

Has anyone else, perhaps someone who also knows your friend, revealed any information to you about this study since the last session?

Yes No

If yes, what did they reveal?

Has the participant said something which has made you in any way suspicious about their role in the study?

Yes No

If yes, what did they say?

Is there anything else to report which may be relevant?

Did you notice the drinking of the other participant while watching TV in session 1?

Yes No

Did you notice the drinking of the other participant while watching TV in session 2?

Yes No

Would you say the amount that the other participant drank, while watching TV was odd in session 1?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Would you say the amount that the other participant drank, while watching TV was odd in session 2?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Would you say the amount that the other participant drank, while watching TV, influenced how much you drank in session 1?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If so, why?

If not, why not?

Would you say the amount that the other participant drank, while watching TV, influenced how much you drank in session 2?

Strongly Disagree Disagree Unsure Agree Strongly Agree

If so, why?

If not, why not?

10.15 Appendix 15: Awareness (study 3) confederate specific

What do believe the aims of the study to be?

Would you normally drink the drink you were given while watching TV?

Strongly Disagree Disagree Unsure Agree Strongly Agree

Did you like the drink you were given while you were watching TV?

Strongly Disagree Disagree Unsure Agree Strongly Agree

How many standard 25ml shots of Vodka do you think were contained in the drink you were given, while watching TV, in session 1?

0_____1_____2_____3_____4_____5_____6_____7_____8_____9+

How many standard 25ml shots of Vodka do you think were contained in the drink you were given, while watching TV, in session 2?

0_____1_____2_____3_____4_____5_____6_____7_____8_____9+

Have you revealed any information about your role in the study to your friend since the last session?

Yes No

If yes, what did you reveal?

Have you revealed any information about your role in the study to someone who also knows your friend?

Yes No

If yes, what did you reveal?

Have you said something, either intentionally or by mistake, which may have made your friend in any way suspicious about your role in the study?

Yes No

If yes, what did you say?

Anything else to report which you think may have results in your friend knowing about what you were told to do?

10.16 Appendix 16: Publication (study 4 and 5)

Knibb, G., Roberts, C. A., Robinson, E., Rose, A., & Christiansen, P. (2018). The effect of beliefs about alcohol's acute effects on alcohol priming and alcohol-induced impairments of inhibitory control. *PloS one*, *13*(7), e0201042

10.17 Appendix 17: Experimental and neutral scripts (study 5)

Experimental:

Our research has found that consuming alcohol reduces the body's urge to drink as the body quickly becomes sated once it has received a small dose of alcohol, reducing the biological urge to drink. Furthermore, we have found that consuming large amounts of alcohol as part of an unplanned binge is a cultural phenomenon found in the UK and Ireland. Other European countries involved in our research program have not found that consuming alcohol leads to further alcohol consumption.

Neutral:

Our research has been investigating the effects of alcohol on thought processes like memory, problem solving and attention. We have so far found that alcohol has a greater effect on some of these processes than others. This final experiment is testing the effects of alcohol on simple reaction times and taste perception.

10.18 Appendix 18: Two-week alcohol diary (study 5)

Participant Number_____

Assessment dates_____

Week 1	Consumed	Units
Day 1		
Day 2		
Day 3		
Day 4		

Day 5		
Day 6		
Day 7		

Week 2	Consumed	Units
Day 1		
Day 2		

Day 3		
Day 4		
Day 5		
Day 6		
Day 7		