UNIVERSITY OF LIVERPOOL

Inhibitory control in heavy drinking: Improving our understanding to optimize behavioural treatments

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Sam Burton (October, 2021).

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Abstract

Fluctuations in inhibitory control are thought to induce states of disinhibition, playing a key role in Alcohol Use Disorders. However, current conceptualisations of inhibitory control in heavy drinkers are *over-simplistic*, including both measures of inhibitory control and influential factors. This thesis aimed to investigate the relationship between environmental cues and reward on inhibitory control in non-dependent heavy drinkers. Specifically, whether exposure to environmental (appetitive-cue exposure) and psychological (extrinsic rewards) triggers can lead to short-term impairments or improvements in inhibitory control. Lastly, this thesis discusses how both environmental and extrinsic rewards may work together to effect inhibitory control and how further research is required. Theories relating to the above are discussed in chapter one, and general methods used throughout the experimental studies in this thesis are described in chapter two.

In chapter three, a meta-analysis suggests that inhibitory control is a transient state that fluctuates in response to reward. Chapter four then sought to examine the effect of reward and environment (cues and context) on inhibitory control and if this was predictive of alcohol use across two studies. There was no consistent effect of reward or environment on inhibitory control, and inhibitory control could not predict alcohol consumption. In chapter five an ecological momentary assessment study showed no significant fluctuations in inhibitory control in a real-world environment, in response to environmental or task-based cues. Chapter six sought to examine if inhibitory control and working memory (executive functioning) could predict alcohol use in adolescents and if inhibitory control performance was sensitive to reward. There was limited evidence for executive functioning being related to alcohol use, or reward causing a significant impairment in inhibitory control.

The overall results of this thesis suggest that inhibitory control is not a transient state that fluctuates in response to environmental and motivational stimuli. Further analysis of reactive control, proactive control and trigger failures (in respect of reward) found no consistent effects of environment or reward on inhibitory control in more detailed analysis. Taken together as a whole, findings contradict theories that suggest inhibitory control is a key factor in the initiation and manifestation of alcohol addiction.

Dissemination

I submit this thesis in partial fulfilment of the conditions for a PhD by published papers. The experimental chapters (chapter three to chapter six) take the form of journal article manuscripts in accordance with the guidelines for the University of Liverpool. These have either been published before submission (chapter three, chapter six), or are prepared for publication (chapter four and chapter five). As required, specific details of each article submission (including the contribution of authors) are given at the beginning of each chapter.

Declaration

This thesis is the result of my own work. No portion of this has been submitted either wholly or partly in support of any other degree or qualification at this or any other University.

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Hel thank you for the unwavering support and being my rock when I needed it most, and even the times I didn't know I needed it. I know you find my work boring, so I promise to never bore you with it again and when you get to the bottom of this page you can stop reading, I promise.

Baloo, you're a dog you'll never read this, you were the sounding board when I needed to talk about an idea or a sentence, so you basically helped write this.

Charlotte, chase your dreams as I have been allowed to chase mine. You'll be told you're not good enough or have set backs, ignore all of it. If in doubt finish it with sheer spite & stubbornness to prove a point (my case in point with this entire thesis).

Dedicated to Charlotte, I started this for me & finished this for you. Love always Dad x

Chapter 1

General Introduction

Alcohol use, prevalence and associated problems

Alcohol is widely and frequently consumed across much of the world. An estimated 43% of the adult population internationally and 88.2% in Western Europe reported consuming alcohol in the 12 month period preceding measurement ton. According to the latest available data from the World Health Organisation (WHO, 2018), individuals over the age of 15 years old consume 6.4 units of pure alcohol per year internationally, with only smoking and obesity being larger risk factors for mortality and disability. Internationally, the harmful use of alcohol accounts for 5.3% of all (~3 million) deaths and 5.1% of disability adjusted life years (DALYs).

Within the U.K., the National Health Service data suggest that 31% of men and 16% of women over 18 years old drink in excess of the recommended guidelines (14 UK units per week; 1 UK unit= 25ml of a standard spirit = 8 grams of pure alcohol; NHS, 2016l) (NHS, 2016). According to the Adult Psychiatric Morbidity Survey (2014), 16.6% of the UK's population drink at 'hazardous' levels, as defined by the Alcohol Use Disorders Identification Test (AUDIT) (Saunders, Aasland, Babor, De la Fuente, & Grant, 1993).

Chronic alcohol consumption is thought to have a causal influence on multiple non-communicable diseases and injuries, and contributes to the development of over 200 others (Rehm et al., 2009). It can lead to liver disease (Osna, Donohue Jr, & Kharbanda, 2017), cardiovascular problems (Holmes et al., 2014), alcohol poisoning and has been linked to 3.6% of all known cancers (Bagnardi et al., 2015; Boffetta & Hashibe, 2006) . Increases in alcohol consumption can lead to an increased risk for mental illness, such as depression (Boden & Fergusson, 2011). A dose-response relationship has been observed for an array of alcohol-attributable disease and injuries. For instance, increased volume of consumption of alcohol is associated with a greater risk of alcohol-attributable cancer (Nelson et al., 2013). Similarly, heavy episodic drinking (also known as 'binge drinking') is predictive of an increased risk of alcohol-related injury compared to lighter consumption (Antai, Lopez, Antai, & Anthony, 2014). Acute alcohol intoxication can cause a variety of symptoms from amnesia ('blackouts') to respiratory depression (Vonghia et al., 2008). Acute alcohol use, particularly at higher doses, is associated with an increased likelihood of attempting suicide (Borges et al., 2017). Even with acute alcohol use, research has demonstrated a clear dose-response relationship between alcohol use and related harms (Cherpitel, Ye, Bond, Borges, & Monteiro, 2015).

As such, overall misuse of alcohol accounts for 4.2% of reduction in viable years of life, this being the impact of health problems as measured by morbidity and cost, with only smoking in relation to adverse health effects being more detrimental to health (Degenhardt et al., 2018; Drummond et al., 2004). The accumulation of evidence for dose-response relationships recently led the UK Government to revise alcohol consumption guidance (2016). Specifically, lower-risk drinking guidelines were revised so that individual's were advised to not exceed 14 units of alcohol per week for both males and females (compared to previous estimates of 21 units for males). It was further clarified that these units should be consumed over a minimum of 3 days to reduce the risk of alcohol related accidental harms, such as injuries or sickness (Department of Health and Social Care, 2016).

Alcohol consumption not only poses a risk to the individual, but also to wider society. Heavy consumers of alcohol demonstrated impairments in cognitive processes after consumption, including reaction times and attention, which has potentially detrimental societal impacts (Gunn, Mackus, Griffin, Munafò, & Adams, 2018). For example, in driving simulations moderate amounts of alcohol have been shown to impair driving ability (Starkey & Charlton, 2014), and this is a major risk factor in most crashes (Fell, 2014; Racioppi, Eriksson, Tingvall, Villaveces, & Organization, 2004). It is estimated that 1 month a year (average hangover length of 2.7 days) is experienced under a 'hangover' (Vester, 2006), with direct costs to government and industry alike. A recent survey showed that in North West England and Scotland, 78.8% and 51.4% of respondents respectively experienced alcohol-related harm, such as road-traffic accidents or physical harm (Gell, Ally, Buykx, Hope, & Meier, 2015). In England and Wales there were nearly 1 million alcohol-related violent crimes in 2010-11 (Chaplin, Flatley, & Smith, 2011) with an estimated 1.1 million alcohol-related hospitals admissions in England during 2015-16 (NHS, 2016). Though frequency of drinking prevalence has dropped in the U.K. from 65% to 58% (ONS, 2017), hospitals admission rates for alcohol-related harms are 19% higher in the past ten years (NHS, 2019), suggesting people who regularly consume alcohol are drinking in greater quantities. Such drinking behaviour costs the National Health Service £3.5 billion annually, with a cost of £21 billion to society as a whole (HSCIC, 2014).

Alcohol use disorder

Statistics cited so far highlight harmful drinking at both a national and international level. Individuals who regularly drink in excess of the UK guidelines (14 units a week) are proposed to have an increased risk of developing alcohol dependence (ONS, 2017). The Diagnostic and Statistical Manual of Mental Disorders V (DSM-V) revised the classification for diagnoses of substance (alcohol) use disorders in 2013 (Reichenberg, 2013), merging abuse and dependence into one specific disorder: Alcohol Use Disorder. Symptoms range from mild to severe, severity of the diagnosis is dependent upon the number of symptoms met: 2-3, 4-5, and 6 or more symptoms are considered mild, moderate and severe respectively. The presence of at least two of the eleven following symptoms in a 12-month period are required for a diagnosis:

1) "Drinks more than intended, or for longer than intended"
2) "Efforts to control or cut back on drinking have been unsuccessful"
3) "Large amounts of time are spent obtaining, using or recovering from alcohol"
4) "Cravings (the presence of a strong desire to drink)"

5) "Recurrent use resulting in problems at work, home or school"
6) "Continued use despite recurrent social or interpersonal problems resulting from drinking"
7) "Curtailing important activities in favour of alcohol use"
8) "Alcohol use despite potentially hazardous outcomes (drinking and driving, for example)"
9) "Continued alcohol use despite knowledge that alcohol use is causing or exacerbating a persistent physical or psychological problem"
10) "Tolerance or a need for increased amounts of alcohol"
11) "Withdrawal symptoms"

The argument for a single disorder is supported by research demonstrating that the criteria for abuse and dependence load onto one factor (Hasin, Muthuen, Wisnicki, & Grant, 1994; Krueger et al., 2004) or two highly correlated factors (Harford & Muthen, 2001; Proudfoot, Baillie, & Teesson, 2006). Craving was added to substance use disorder criteria due to craving being a diagnosis and treatment target (O'Brien, 2005; Tiffany & Wray, 2012). Inclusion of craving has been shown to improve discriminatory power in comparison to previous measures (Mewton, Slade, McBride, Grove, & Teesson, 2011).

To summarise, regular heavy drinking puts individuals and wider society at higher risk of multiple health-related and other negative consequences. Identifying psychological factors that lead to heavy drinking and potentially alcohol use disorder may help reduce the risk of recreational alcohol use progressing to hazardous levels, minimising the burden on the NHS from alcohol-related costs, as well as both individual and population-level harms. To investigate factors that may contribute to this transition, the research presented in this thesis recruited adult individuals who consumed more than 14 units per week but had no previous or current diagnosis of alcohol use disorder. By investigating individuals who are not clinically diagnosed with alcohol use disorder, interventions can be designed to reduce the likelihood of transition.

Modelling heavy drinking: a loss of self-control and dysfunctional motivation

Contemporary models of addiction suggest that addiction to a substance is the result of a 'brain disease' or a product of deep-learning (Lewis, 2017; Volkow, Koob, & McLellan, 2016), however the brain disease model is extremely challenged by the academic community (Heather et al., 2018). Theories of addiction tend to constitute biological, social or psychosocial processes. Models often deal with these factors as an individual process or a combination thereof. Such processes can be used to describe and explain addiction in terms of substance-related stimuli, individual's predisposed risk, motivations, environment, recovery and relapse (West & Brown, 2013). While neurobiological theories of addiction propose that the substance (mis)use is the result of two systems interacting; broadly described as an activation (or approach) system and an inhibition system. Theories of addiction tend to capture certain elements of the phenomenon, such as the brain disease models lack of emphasis on psychosocial factors (Afzal, 2020), but do not approach addiction in a holistic manner. To better understand addiction, one must look at the underlying processes that contribute to its development.

A widely hypothesised precursor to excessive alcohol use and potential development of alcohol use disorder is a lack of self-control. Self-control is defined as resisting temptations and the urge to act impulsively (Diamond, 2013). A second non-mutually exclusive psychological factor of addiction is thought to be a biased motivational system, in which an abnormal amount of priority is given to reward driven behaviour(s), manifesting as a loss of behavioural control (Volkow et al., 2010). Individuals with substance use disorders have been reported to characterise their behaviour as 'a loss of control', or use associated language, when discussing their addiction (Everitt, 2014;. Levy, 2014). Furthermore, a 'loss of control' clearly fits within the second DSM-V criteria for an alcohol use disorder (e.g. "Efforts to control or cut back drinking have been unsuccessful"), and overlaps with other criteria (e.g. "Continued alcohol use despite knowledge that alcohol use is causing or exacerbating a persistent physical or psychological problem").

Questionnaire measures of self-control are designed to tap into one's ability to inhibit a dominant response, in both behaviour and thought (Tangney, Boone, & Baumeister, 2018). Tangney and colleagues have demonstrated that Self-Control Scale scores were related to better psychological adjustment and reduced problematic food and alcohol consumption, with a meta-analysis reporting similar findings (De Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012). However, recent work suggests that self-report measures are better suited to measure domain-general aspects, such as trait level self-control, rather than specific behavioural measures, such as inhibition (Wennerhold & Friese, 2020). In addition, questionnaire-based measures of self-control are inherently subjective, with respondents having to predict or recall a given behaviour (i.e. "I spend too much money or getting up of a morning is hard for me"), allowing for biases and inaccuracies (Reynolds, Richards, & de Wit, 2006). In support of this, experimental and questionnaire-based measures of self-control are shown to not be associated (Enkavi & Poldrack, 2020; Mazza et al., 2020), and convergent validity between task measures of inhibition and questionnaire measures of self-control is poor (Saunders, Milyavskaya, Etz, Randles, & Inzlicht, 2018), with the two loading onto distinct and unrelated constructs (Eisenberg et al., 2019; Reynolds, Ortengren, Richards, & De Wit, 2006). Objective measures of inhibitory control may therefore better characterise the '*loss of control*' as defined in models of addiction, as an inability to withhold or delay preponent responses, due to the un-reliance on retrospective recall of individuals (Petersen, Hoyniak, McQuillan, Bates, & Staples, 2016).

The following sections will discuss theories of inhibitory control and how these fit in to theories of addiction. 'Loss of control', in terms of addictive behaviours, is characterised as automatic processes that drive behaviour irrespective of our conscious control (Wiers, Rinck, Kordts, Houben, & Strack, 2010). This will be discussed in respect to addictive stimuli, individual predisposed risk, and environmental factors and motivations, with a particularly focus on how symptoms of addiction, such as reduced self-control, are dependent upon one's environment (Field, Heather, & Wiers, 2019).

Disinhibition / Response Inhibition measures of inhibitory control in laboratory environments

(Dis)inhibition (also termed inhibitory control or response inhibition) has been identified as the measurable equivalent of 'loss of control' (Leeman, Corbin, & Fromme, 2009). It is defined as "the (in)ability to stop, change or delay an inappropriate response in

the current environment ". In respect to alcohol use disorder, it can be conceptualised as the inability to withhold from drinking when walking past a bar when attempting to cut down or when already intoxicated. Importantly, deficits in inhibitory control are not exclusive to substance use disorders and have been shown to be present in numerous psychiatric conditions, such as ADHD, Schizophrenia, OCD, and Parkinsons (Coutinho, Reis, da Silva, Miranda, & Malloy-Diniz, 2018; Obeso et al., 2011; Richardson, 2008). Furthermore, comorbidity between alcohol use and psychiatric disorders is well documented (Brière, Rohde, Seeley, Klein, & Lewinsohn, 2014; Neupane & Bramness, 2013; Petrakis, Gonzalez, Rosenheck, & Krystal, 2002).

Measuring Disinhibition: Behavioural Tasks

Stop-Signal Task (SST)

The stop-signal task (SST) (Logan & Cowan, 1984) is a widely accepted and established measure of response inhibition. The task involves participants categorising stimuli into predefined categories in 'go trials' as quickly as possible, to build up a pre-potent motor response for the majority of trials. Stimuli can be relevant or irrelevant cues (Verbruggen & Logan, 2009) or arbitrary ('x' or 'o' on a standard discrimination task). On a minority of trials, a stop signal is presented either in the form of an auditory tone or a visual signal; known as 'stop trials'. Participants are instructed to withhold their categorisation response when the stop-signal occurs, and are asked to respond as quickly as possible to prevent compensatory strategies on stop trials and skewing the data (Sylwan, 2004). Go and stop trials are normally presented in a ratio of 3:1 to provide a dominant discrimination response. Stop trial frequency can be varied to alter and examine response conflict, difficulty of inhibition (Ramautar, Kok, & Ridderinkhof, 2004) and response strategies (Verbruggen & Logan, 2009). An individual's ability to inhibit the dominant 'go' response on 'stop' trials is taken as a measure of inhibitory control.

Stop-signals are presented after the 'go' stimulus, and the delay between the go stimulus and the stop signal is called the stop-signal delay (SSD). Hypothetically, longer SSDs mean it is more difficult to prevent a categorisation response ('go' response) as the likelihood of initiating a response has already begun, whereas shorter SSDs make inhibiting easier, as it is less likely that a go response has been initiated (Verbruggen, McLaren, & Chambers, 2014). A variety of settings for the SSD have been developed to assess the full extent of inhibitory processes. The original task used fixed SSDs (Logan & Cowan, 1984), alternative versions use a tracking algorithm (Logan, Schachar, & Tannock, 1997) which adjusts SSDs based upon inhibitory performance within the task, or SSDs based on mean 'go' trial reaction times (Carter et al., 2003). Specifically, Carter and colleagues set SSDs based on the mean 'go' reaction time, specifically as a proportion of it (20%, 40%, 60% or 80%). Logan and colleagues used a method in which SSDs are adjusted based on whether the individual makes a correct stop on stop trials or not, for example if they stop the SSD is increased by 50ms and vice versa if incorrect.

The task is conceptualised as a race between going and stopping, which are seen as two independent processes (Band, van der Molen, & Logan, 2003). Whichever response is

executed first is the observed behaviour, for example if the 'go' response is completed before the 'stop' response, the individual will respond with the 'go' behaviour. In standard conditions, participants are asked to categorise the stimuli as quickly as possible but to withhold their response should they hear or see the stop-signal. This creates a speed/accuracy trade off or a response conflict, so they cannot go too fast without reducing inhibition, or focus on inhibition at the cost of go categorisation (Leotti & Wager, 2010). To account for these issues, one is able to calculate a Stop-Signal Reaction Time (SSRT) which is taken as a measure of inhibitory control.

SSRT is the epoch from the commencement of the stopping process (stop-signal presentation) to the end, which cannot be measured as one cannot directly observe people inhibiting. SSRTs are calculated using the probability of successful inhibition and 'go' reaction times, taking into account participant's motivational biases and response conflict (Leotti & Wager, 2010). One's ability to inhibit is influenced by an array of factors, such as stop-signal delay and 'go' reaction time, therefore SSRT are a key measure of inhibitory control.

SSRTs allow for a reliable and valid measurement of inhibitory control that is sensitive to changes in the human condition. They allow for the control of individual differences in 'go' reactions times and task difficulty, providing a robust index of inhibitory control (Band et al., 2003), and have been observed to be a reliable measure of inhibitory control for a variety of psychopathologies compared to controls (Lijffijt, Kenemans, Verbaten, & van Engeland, 2005; Lipszyc & Schachar, 2010). Neural activation in regions known to be involved in inhibitory control during fMRI scans have been shown to be related to SSRTs (Congdon et al., 2010). SSRTs have also been shown to be sensitive to pharmacological manipulations (Rubia, Halari, Mohammad, Taylor, & Brammer, 2011; Tannock, Schachar, & Logan, 1995), showing application in understanding neurobiological functions, such as inhibitory control in heavy drinkers. Inhibitory control estimation methods, can reliably estimate SSRTs across the life span suggesting a high reliability for SSRTs as a measure of inhibitory control (Congdon et al., 2012; Williams, Ponesse, Schachar, Logan, & Tannock, 1999).

For inhibitory control to be effective, it is suggested that not only reactive control is required but also proactive control processes (Criaud, Wardak, Ben Hamed, Ballanger, & Boulinguez, 2012). Verbruggen, McLaren, and Chambers (2014) note how the majority of the literature focuses on reactive inhibitory control, despite human ability to plan and modify our behaviour proactively. The Dual Mechanisms of Control (DMC) framework (Braver, 2012) argues for the operationalisation of inhibitory control as reactive (retrieving contextual information that is purely required for the immediate instance) and proactive control (actively maintaining contextual information to prepare a response). Aron (2011) suggests that 'proactive' control is a more appropriate model of inhibition in substance-use behaviour, with some research suggesting it is the 'default' form of inhibition (Criaud et al., 2012). Indeed, the notion that 'proactive' control is the default form of inhibition is logical, as individuals proactively adjust their substance-use behaviour over time to control cravings, rather than relying on reactive control as a late correction mechanism (Braver, 2012; Braver, Paxton, Locke, & Barch, 2009). Yet within the current research base, there is little evidence that clarifies the relationship between proactive control and substance misuse.

Through Stop-Signal paradigms, efforts have been made to allow individual measurements of reactive and proactive control. In these tasks, participants are asked to

respond as quickly as possible rather than waiting for the appearance of a stop signal (Logan, Cowan, & Davis, 1984a). In blocks when inhibition is required, participants are observed proactively slowing their responses in preparation for inhibition compared to non-inhibition blocks (Verbruggen, Stevens, & Chambers, 2014). Further research has demonstrated similar proactive adjustments in both healthy (Elchlepp, Lavric, Chambers, & Verbruggen, 2016; Verbruggen & Logan, 2009b), and alcohol-dependent populations, who display poorer proactive control, in comparison to controls (Hu, Ide, Zhang, Sinha, & Chiang-shan, 2015).

Alternatively, the stop-signal cue has been used to indicate stop-signal probability (Brevers et al., 2012; Verbruggen & Logan, 2009b). In such paradigms the proportion of inhibition errors and SSRTs are taken as measures of proactive and reactive control respectively (Castro-Meneses, Johnson, & Sowman, 2015). Castro-Meneses et al. (2015) suggest that proactive control is operationalised as a preparation to stop in anticipation of a stop-signal, which in turn is associated with faster SSRTs, suggesting proactive control has a downstream effect on reactive control. The authors propose this downstream effect is a result of the reactive and proactive inhibition systems using the same network, pre-activating the reactive system and facilitating reactive inhibition.

The Go/No-go task

To assess inhibitory control (i.e. the ability to withhold a pre-potent response), a basic Go/No-go task (GNG) involves presentation of either a go cue or no-go cue, and participants are asked to respond to go cues and to provide withhold their response to no-go cues (Gordon & Caramazza, 1982). As in the stop-signal task, the majority of trials are go trial, to reinforce the go response. The number or proportion of errors on no-go trials, termed commission errors, is taken as the measure of response inhibition (Simmonds, Pekar, & Mostofsky, 2008).

A common variation of the standard paradigm, in which complex cues are presented as the stimuli such as alcohol or food, can be used to examine cognitive processes other than inhibitory control. Such paradigms, in which a given cue is indicative of a need to respond to a further stimulus, i.e. possibility of receiving a reward or probability of having to inhibit, can influence top-down processes involved in response preparation (Ahmadian, Cagnoni, & Ascari, 2013; Grane et al., 2016; Stuss, Miller, & Cummings, 2007). Cues may also be presented as stimuli, such as pictures or words, to examine the effects of substance-related stimuli on inhibitory control (Ames et al., 2014; Kreusch, Vilenne, & Quertemont, 2013). In substance-use populations, GNG is able to assess attentional biases towards substance-related stimuli (Field & Cox, 2008).

Variations of the GNG have sought to increase participant motivation (Griffith-Lendering, Huijbregts, Vollebergh, & Swaab, 2012; Kohls, Herpertz-Dahlmann, & Konrad, 2009), thereby attempting to distinguish the effects of inhibitory load and participant motivation (Demurie, Roeyers, Wiersema, & Sonuga-Barke, 2016). The passive avoidance task (Newman & Kosson, 1986) is a GNG variation which incorporates reward or punishment to increase participant motivation, and has been used to examine substance-use behaviour (Christiansen, Cole, Goudie, & Field, 2012; White et al., 2016). Yet, as GNG variations become more complex (i.e. multiple go stimuli or reward-indicative cues) working memory is placed under increased demand, in turn influencing inhibitory control (Simmonds

et al., 2008). This makes it hard to discern the true effect of experimental manipulations on inhibitory control.

Longitudinal research has demonstrated that atypical neurological responses during inhibitory control (i.e. GNG performance) is predictive of substance-use and dependency symptoms (Mahmood et al., 2013), as well as alcohol use (Norman et al., 2011) and alcohol-related problem outcomes (e.g. blackouts) (Wetherill, Castro, Squeglia, & Tapert, 2013). Performance on the GNG has been correlated with inhibitory control deficits in abstinent alcohol-dependent individuals (Dom, D'haene, Hulstijn, & Sabbe, 2006), young drinkers with poor working memory (Finn, Justus, Mazas, & Steinmetz, 1999) and general executive functioning in chronic heavy drinkers (Bowden, Crews, Bates, Fals-Stewart, & Ambrose, 2001). GNG is sensitive to the psychological and pharmacological effects of alcohol in substance-use populations, with inhibitory control deficits (Noël et al., 2007; Noël et al., 2005; Weafer & Fillmore, 2012) and heightened salience towards alcohol stimuli observed (Rose & Duka, 2008).

Less widely used tasks to measure inhibitory control are the Flanker (Eriksen & Eriksen, 1974), the Stroop (Stroop, 1935), the Antisaccade (Hallett, 1978), and the Simon tasks (Simon, 1969). These paradigms share task instructions to not respond to given stimuli and are underpinned by similar cognitive resources as the Stop-signal and Go/No-go tasks (Schachar et al., 2007). Evidence suggests that a global inhibition network is activated independent of the task used, yet conceptually-distinct regions are subsequently activated for distinct inhibitory aspects of a task (Dambacher et al., 2014). Although not all tasks load onto the same factor of inhibitory control, primarily effortful and automatic inhibition, some variance is shared between them (Howard, Johnson, & Pascual-Leone, 2014). In the wider field of inhibitory control research, experimental measures allow us to operationalise inhibitory control and provide an unbiased measure of the 'loss of control' observed in addictive behaviours. Hence the research presented in this thesis used experimental measures of inhibitory control in heavy drinkers to provide an unbiased measure of inhibitory performance in an attempt to characterise the 'loss of control' that has been observed in situations involving alcohol.

Development of inhibitory control and associated neuronal components

Development of inhibitory control and executive functioning can be explained as a shift from set stimulus-action associations towards a more conceptual rule-like system (Munakata, Snyder, & Chatham, 2012). Verbruggen et al. (2014) state that children build upon stimulus-action associations to form abstract representations that dictate behaviour in later life. Individuals exhibit delayed prefrontal development, due to the need to understand social and linguistic cues, that are required to acquire inhibitory control based on abstract concepts as an adult (Thompson-Schill, Ramscar, & Chrysikou, 2009). The following section will discuss how inhibitory control develops throughout the lifespan and its associated neuronal components.

Inhibitory control, and executive functions in general, are proposed to develop steadily throughout an individual's life span, even during adulthood (Best & Miller, 2010; Carlson, 2005; Garon, Bryson, & Smith, 2008). Multiple studies describe a rapid improvement in inhibitory control throughout childhood to adolescence (Brocki & Bohlin,

2004; Cragg & Nation, 2008; Garon et al., 2008). Results from behavioural studies suggest that inhibitory control and executive functioning continues to develop up until age 21, with evidence of proactive control slowing to avoid errors suggesting metacognitive development (Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005; Huizinga, Dolan, & van der Molen, 2006). In older adults (age 30+) it is suggested that inhibitory control begins to regress, yet results are inconsistent as they show this may reflect poorer responding to preponent signals, rather than global deficits in inhibitory control (Rey-Mermet & Gade, 2018; Williams et al., 1999). Differences observed across the life course, particularly following early childhood, are suggested to be refinement of cognitive processes for complex tasks that require the input of different cognitive modalities (Best & Miller, 2010; Petersen et al., 2016).

Maturation of underlying neuronal processes dictate the development of inhibitory control throughout the life course (Munakata et al., 2011). Using imaging techniques (Tamm, Menon, & Reiss, 2002; Tompson et al., 2018), it has been demonstrated that response inhibition and Pre-Frontal Cortext (PFC) activation are correlated. In behavioural studies using Go/No-Go tasks has shown that adults show a greater localised response in the prefrontal cortex when compared to children (Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Johnstone et al., 2007; Rubia et al., 2006). Increased localisation in prefrontal regions in adults is a result of synaptic pruning decreasing short-range connectivity to increase efficiency (Fair et al., 2007). On the other hand, some studies have found contradictory findings, such as increased activation in prefrontal regions in children compared to adults (Casey et al., 1997; Hwang, Velanova, & Luna, 2010; Lamm, Zelazo, & Lewis, 2006) potentially due to increased short-range connectivity.

The precise location of the regions involved with the development of inhibitory control and executive functioning are difficult to identify, due to the sub-processes involved and their contribution to said processes (Aron, Fletcher, Bullmore, Sahakian, & Robbins 2003). Aron and colleagues compared healthy control and individuals with a lesion to the right Inferior Frontal Cortex (rIFC), and found impaired inhibition compared to controls. Findings implicating the role of the rIFC in inhibition are supported by neuroimaging studies and using a variety of measures of inhibitory control (Hampshire, Chamberlain, Monti, Duncan, & Owen, 2010; Katya Rubia, Smith, Taylor, & Brammer, 2007). However, inhibition is not considered a unitary process (Miyake et al., 2000). Depending on the type of inhibition processes – such as conflict detection – attention, motor movements, and memory can be recruited (Booth et al., 2003; Casey, Giedd, & Thomas, 2000), yet the rIFC is argued to be the locus of inhibition (Aron, Robbins, & Poldrack, 2014).

Inhibitory control as a component of impulsivity and executive functions.

In addiction research the concept of impulsivity is well studied as an analogous measure of self-control, given that it is an antipode of self-control (i.e. if someone can withhold the impulsive choice to consume alcohol they have good self-control)(Mirabella, 2021; Garavan, 2011; Reed & Naudé, 2020). Given the well-known issues with self-report measures of self-control mentioned previously, models have begun to shift focus to experimental (objective) measures. Particularly, models of addiction propose that elevated impulsivity has an important role in addiction, as both an antecedent and consequence of substance misuse (De Wit, 2009; Weafer, Mitchell, & de Wit, 2014). Impulsivity has multiple definitions, with inhibitory control a widely accepted facet (Leshem & Yefet, 2019),

with an array of measurements, approaches and theories. Early theories of addiction describe impulsivity as rash and unplanned behaviours, irrespective of consequences. However, Dawe, Gullo, and Loxton (2004) argued that acquisition and use of substances involves pre-meditated goal-directed behaviour, and argue it is influenced by another factor, 'reward sensitivity'. More recently, impulsivity has been viewed as a multi-faceted construct, serving as an umbrella term for various behaviours, such as delay discounting, risk-taking and inhibitory control (Weafer & Fillmore, 2016).

Impulsivity can be further dissected into both 'state' and 'trait' measures. Trait measures of impulsivity tend to be questionnaire-based, such as The Barratt Impulsivity Scale (BIS; (Patton, Stanford, & Barratt, 1995), and conceptualises impulsivity as being stable over long periods of time. These measures of impulsivity have been shown to be associated with alcohol use disorders (e.g. (Martínez-Loredo et al., 2015), and are a risk factor for future hazardous drinking (Christiansen et al., 2012; Gordon Fernie et al., 2013). State measures are thought to be transient and to fluctuate. For example, intoxication as a result of alcohol can cause state fluctuation in components of impulsivity (de Wit, 2009). Supporting the notion of impulsivity being multifaceted, Christiansen et al. (2012) and Reynolds et al. (2006) demonstrated that SST and GNG loaded on the same factor (inhibitory control), whereas other behavioural measures loaded to another factor (impulsive decision-making or delay discounting). Christiansen et al. (2012) also found a third component, representing trait impulsivity, with all three components predicting hazardous drinking. Multiple sources suggest behavioural and self-report measures of impulsivity are distinct from one another, which suggests a multifaceted structure (Eisenberg et al., 2019; White et al., 1994) as is promoted by other prominent theories of addiction (De Wit, 2009; Dick et al., 2010; Olmstead, 2006).

Multiple theories have been debated as to how individuals carry out and adapt goal-directed behaviour; cognitive systems involved in these behaviours are termed Executive Functions (EF) (Verbruggen, McLaren, et al., 2014). The term EF refers to an array of higher-order cognitive functions, including planning behaviour, inhibition and goal-directed decision making, which enable self-regulation and feed into more complex behaviours (Miyake et al., 2000). These processes are viewed as working in unity (Miyake & Friedman, 2012), with a proposed common underlying ability or process, likely controlled by the pre-frontal cortex. The structure attributed with the role of maintaining task-related information and goals has been coined the 'central executive', and is proposed to utilise Working Memory (Baddeley, 1996). Finn (2002) put forward the notion of Working Memory differences, which contribute to and moderate impulsivity and related behavioural issues, such as alcohol misuse and abuse (Ellingson, Fleming, Vergés, Bartholow, & Sher, 2014; Finn, Gunn, & Gerst, 2014; Hatz, McCarty, Bartholow, & McCarthy, 2018).

Importantly, Bickel, Jarmolowicz, Mueller, Gatchalian, and McClure (2012) propose that components of EF and impulsivity are antipodes of each other. They argue that behavioural (dis)inhibition – the (in)ability to restrain an already initiated behaviour – is associated with impulsive and norm-violating behaviour (Bogg & Finn, 2010), and is the antipode of behavioural inhibition. Behavioural inhibition describes three processes: inhibition of a prepotent response, withholding an ongoing response to delay the decision to respond, and inference control (Barkley, 1997), with Bickel arguing that behavioural disinhibition is implicit in the second process. Therefore, good performance on the SST could be inferred as reduced impulsivity and efficient EF, and poor SST performance would represent heightened impulsivity and inefficient EF. Therefore, components of EF and

impulsivity operate at opposite ends of a continuum (Bickel et al., 2012). In support, individuals with dysfunctional impulsivity have been shown to have slower SSRTS compared to individuals with lower impulsivity (Castro-Meneses et al., 2015). Yet Bickel and colleagues discuss the lack of overlapping research between the constructs, and some literature reports null findings (Wu et al., 2013; Zhang et al., 2015).

Inhibitory control in substance use

A hallmark of substance-use models is the dysregulation of inhibitory control in individuals who use substances, which may contribute to or exacerbate detrimental substance-seeking behaviours (Goldstein & Volkow, 2011; Kalivas & Volkow, 2005). Neuroimaging studies have consistently reported abnormalities in brain functions during response inhibition in currently addicted individuals (Luijten et al., 2014; Smith, Mattick, Jamadar, & Iredale, 2014). Previous research supports the aforementioned predictions in both dependent and non-dependent populations.

In comparison to healthy controls, individuals with alcohol dependency have been shown to have poorer inhibitory control, mainly using Stop-Signal and/or Go/No-go tasks (Goudriaan, Oosterlaan, De Beurs, & Van Den Brink, 2006; Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009; Zago-Gomes & Nakamura-Palacios, 2009). Evidence from meta-analyses have shown alcohol dependent and heavy drinkers have impaired inhibitory control compared to controls, and such impairments are observed across both SST and GNG tasks (Smith et al., 2014). Alcohol-dependent populations tended to show larger effect sizes than heavy drinkers, suggesting a larger deficit in inhibitory control in comparison to controls. Deficits in inhibitory control has been related to cigarette dependency (Billieux et al., 2010), as well as cocaine (Czermainski, Willhelm, Santos, Pachado, & de Almeida, 2017; Fillmore & Rush, 2002) and methamphetamine use (Monterosso, Aron, Cordova, Xu, & London, 2005). In contrast, other research has demonstrated no difference between healthy controls and those with a current (van der Plas, Crone, Van Den Wildenberg, Tranel, & Bechara, 2009) or previous (Taylor et al., 2016) diagnosis of alcohol dependency, suggesting the relationship may not be robust.

Longitudinal research suggests that deficits in inhibitory control contribute to the development of alcohol dependence (Rubio, Jiménez, Rodríguez-Jiménez, et al., 2008), comorbid substance use (Nigg et al., 2006) and treatment success (Rupp et al., 2016). In one such study, Czapla et al. (2016) showed that inhibitory control was a significant predictor of relapse in individuals who were alcohol-dependent compared to controls at six-month follow up assessments. Specifically, individuals with the highest impairment of inhibitory control and multiple previous detoxifications were the strongest predictor of relapse. Deficits in inhibitory control may play a causal role in the development of alcohol dependency, as deficits have been observed prior to alcohol use (Ersche et al., 2012; Moeller, Bederson, Alia-Klein, & Goldstein, 2016). In substance dependent populations, high levels of impulsivity exist prior to drug use (Argyriou, Um, Carron, & Cyders, 2018). However, evidence suggests that substance use may mask pre-existing characteristics due to their effect on brain structures and long-term functioning (Verdejo-García, Lawrence, & Clark, 2008).

Longitudinal research has also shown that at a 4-year follow up, SST performance can predict transition from being a heavy drinker to having an alcohol-use disorder (Rubio,

Jiménez, Rodríguez-Jiménez, et al., 2008), along with other substance use disorders (Monterosso et al., 2005; M. Moreno et al., 2012). SST performance is less consistently associated with binge drinking, alcohol consumption (Bø, Billieux, Gjerde, Eilertsen, & Landrø, 2017; G. Fernie, Cole, Goudie, & Field, 2010), or hangover days (Paz, Keim, & Rosselli, 2016). Yet, in longitudinal research, the predictive validity of inhibition on future alcohol-use is questionable (Byrne & Worthy, 2019; Jurk, Mennigen, Goschke, & Smolka, 2018). SST is sensitive to those with a diagnosis of alcohol-use disorder (Ferrett, Carey, Thomas, Tapert, & Fein, 2010) and to individuals with a family history of alcohol-related problems (Acheson, Richard, Mathias, & Dougherty, 2011). In summary measures of inhibitory control deficits are able to predict current alcohol use and transition to a diagnosed condition, yet their predictive validity for future alcohol use is questionable.

With regards to non-dependent drinkers, a substantial amount of evidence suggests that impairments in inhibitory control are associated with alcohol use in non-dependent drinkers (Christiansen et al., 2012; Houston et al., 2014; P. Murphy & Garavan, 2011). Cross-sectional findings show that inhibitory control impairments are associated with binge drinking (Carbia, López-Caneda, Corral, & Cadaveira, 2018), number of intoxication and hangover days (Paz et al., 2016) and *ad libitum* alcohol consumption in a laboratory environment (Field & Jones, 2017). Longitudinally, individual differences in inhibitory control predict future alcohol and drug use (Gordon Fernie et al., 2013; Peeters, Oldehinkel, & Vollebergh, 2017) and are associated with higher AUDIT scores (Hu, Zhang, Chao, Krystal, & Li, 2016).

The association between inhibitory control deficits and substance-use is less consistent in healthy controls compared to heavy drinkers (Smith et al., 2014). Heavy drinkers generally had poorer inhibitory control, suggesting that heaviness of use is associated with inhibitory control performance. Yet in one case, heavy drinkers demonstrated greater inhibitory control than controls for a GNG task (Ames et al., 2014). Authors noted, however, that heavy drinking populations are harder to categorise due to a lack of a definitive cut-off for heavy drinking, and findings are drawn from correlations warranting a more causal assessment. Multiple studies have been unable to find an association between inhibitory control and alcohol consumption (Fernie et al., 2010; Weafer, Milich, & Fillmore, 2011), with recent work suggesting an inverse relationship in the general population, with weekly alcohol use found to be associated with better inhibitory control (Bø & Landrø, 2017). Research suggests that impairments in inhibitory control may be restricted to certain populations or a given developmental period, since the majority of research focuses on heavy-drinking university students or young adults. In addition, other studies have shown there is little evidence of inhibitory control deficits in heavy drinkers (Franken, Luijten, van der Veen, & Van Strien, 2017; Nederkoorn, Baltus, Guerrieri, & Wiers, 2009), or binge drinkers (Czapla et al., 2015; Moreno et al., 2012).

Inhibitory control as both a risk factor and consequence of substance use

The research discussed above is largely associative (cross sectional) and does not capture temporal variables that can have a downstream effect on substance-related behaviours. Therefore, the evidence for a relationship between inhibitoryu control and substance use does not meet criteria for causality. It is difficult to empirically investigate causality with regards to substance-use behaviours, given the ethical implications.

Longitudinal designs provide one solution, as we can see how various variables interact and influence substance-use behaviour and neuropsychological functions.

Recent reviews in this area provide two potential explanations for the relationship between inhibitory control and alcohol use (Jones, Christiansen, Nederkoorn, Houben, & Field, 2013; Perry & Carroll, 2008). The first explanation is that the prefrontal cortex is exposed to neurotoxic effects as a results of chronic substance use, which may impair inhibitory control. The Incentive Sensitization theory (Robinson & Berridge, 1993) argues that through recurrent use of a substance, abnormalities form in the brain reward-related systems that underpin motivated behaviour. These abnormalities increase the salience of drug-related stimuli which, even in periods of abstinence, can increase future substance-seeking behaviour (Robinson & Berridge, 2008), evoking a reward response similar to that experienced at first consumption (Di Chiara, 2002). Following on from this, the 'Impaired Response Inhibition and Salience Attribution Syndrome of Drug Addiction Model' (I-RISA) suggests that the reinforcement of substance-seeking is primarily based in the frontal cortex, which is involved in periods of increased craving, intoxication and withdrawal (Goldstein & Volkow, 2002). This is a result of recurrent exposure to a given substance and the associated cues, which increases salience and alters the brain systems and chemistry that control behaviour. Both binging and relapse behaviours stem from alterations in brain systems from recurrent substance use and alterations in chemical pathways that include Dopamine, Serotonin and GABA (Banerjee, 2014).

In support, chronic substance-use has been shown to damage brain structure and negatively impact brain function, in turn influencing (dis)inhibition. Animal studies have shown that in adolescent and adult rats placed on a four day ethanol binge paradigm, significant damage to frontal cortical regions occurs, particularly in the adolescent rats (Crews, Braun, Hoplight, Switzer III, & Knapp, 2000). Nixon and Crews (2002) found similar results, indicating that both acute and chronic ethanol binges can damage frontal cortical regions. Such neurotoxic effects are not limited to ethanol, with similar patterns seen for cocaine and methamphetamine (Kuczenski et al., 2007). However, it is worth noting that the veracity of animal models of addiction is under dispute, especially given to the multifaceted nature of addiction that requires the incorporation of environmental, psychological and sociological factors (Field & Kersbergen, 2020).

Nevertheless, in human neurobiological models, similar patterns of results have been found to animal models. Substances cause dysfunctions to neurotransmitters, dopamine and serotonin (Koob & Volkow, 2010), leading to both increase salience of rewarding stimuli (Robinson & Berridge, 1993) and changes to specific cortical regions and their associated functions (Klenowski, 2018). In individuals who are alcohol-dependent, research has demonstrated reductions in frontal lobe volumes (Kubota et al., 2001; Pfefferbaum, Sullivan, Mathalon, & Lim, 1997), pre-frontal cortex (Crews et al., 2004) and decreased grey matter (van Holst, de Ruiter, van den Brink, Veltman, & Goudriaan, 2012) as a result of chronic alcohol-use in comparison to healthy controls.

A significant new body of evidence suggests that brain atrophy is somewhat reversible following a prolonged period of abstinence (Bartsch et al., 2007; Cardenas, Studholme, Gazdzinski, Durazzo, & Meyerhoff, 2007; Gazdzinski, Durazzo, & Meyerhoff, 2005). Despite the re-growth of previously atrophied brain regions, from recurrent alcohol consumption, brain volume is still reduced compared to healthy controls (Mann et al., 2005), and it is unclear whether a 'full recovery' is possible long term (Zahr & Pfefferbaum, 2017).

Therefore, through chronic substance use frontal cortical regions can sustain damage which may lead to impaired inhibitory control. Such impairments may contribute to subsequent substance use behaviour.

The second explanation is that during adolescence, impaired inhibitory control is a risk factor for developing substance-use, and consequently a substance use disorder. To investigate this explanation, one must examine if pre-existing impairments of inhibitory control exist before substance (mis)use develops, and if they can predict such behaviours. One such study (Peeters et al., 2017) reported that response inhibition at age 11 was able to predict future risk for alcohol and cannabis consumption during adolescence. Similar findings have been observed and several other studies in both in individuals with substance use disorders and their healthy siblings, suggesting a predisposition to substance addictions as a result of brain irregularities (Ersche et al., 2012; Whelan et al., 2012; Whelan et al., 2014). Other studies have shown that impairments in early life present as a risk factor for future alcohol problems (Mahmood et al., 2013; Nigg et al., 2006; Wetherill et al., 2013; Wong et al., 2006). For example, Squeglia, Jacobus, Nguyen-Louie, and Tapert (2014), reported that inhibitory control deficits, prior to initiation of substance use (aged 12-14 years old), were associated with increased alcohol use (number of drinking days and drinks per occasion) and marijuana use during late adolescence (aged 17-18 years old). Similarly, comparable results have been reported in adults in addition to developmental evidence in children. Rubio, Jiménez, Rodríguez-Jiménez, et al. (2008) used a stop-signal task to examine inhibitory control of adults in a primary care environment. Impaired inhibition predicted the likelihood of transition from heavy drinking to alcohol use disorder at four-year follow-up. Another study, using a Go/No-Go task in alcohol dependent patients, demonstrated that poor response inhibition was a risk factor for treatment adherence and relapse (Rupp et al., 2016). Certainly, there is evidence that poor inhibitory control is a risk factor for alcohol misuse in later life.

However, some research argues that impairments in inhibitory control, and the associated frontal cortical region differences, may both precede and occur following chronic and excessive substance use (Morein-Zamir & Robbins, 2015). Throughout adolescence inhibitory control is still developing, placing individuals at risk for alcohol misuse, which may in turn interfere with cognitive development. Furthermore, there exists a body of evidence that suggests the longitudinal link between inhibitory control and alcohol use may be weak to non-existent (Boelema et al., 2015; Caswell, Celio, Morgan, & Duka, 2016; I. H. Franken et al., 2017; Peeters et al., 2014). Researchers have further suggested that the relationship between the two is in fact explained by a third common factor, such as sex, environment, or reward reactivity (Perry & Carroll, 2008), thus more longitudinal studies with a broader examination of variables are required to understand the relationship between inhibitory control and alcohol use (López-Caneda, Rodríguez Holguín, Cadaveira, Corral, & Doallo, 2013).

Interim summary

To summarise, evidence shows that deficits in inhibitory control can be both a risk factor for developing a substance-use disorder and the result of one. Deficits in inhibitory control have been observed in dependent and non-dependent populations to varying degrees, with the latter being less consistent. The mechanism by which inhibitory control fluctuates in response to stimuli is not well understood, and it is argued that current models are too

simplistic (Verbruggen, McLaren, et al., 2014) and do not focus on the processes or factors that underpin these behaviours (Berkman, 2018). Therefore, the aim of this thesis is to provide a more comprehensive account of inhibitory control and how it may act as a short-term risk factor in response to external stimuli. Particularly, this thesis aimed to assess the effect of environment and extrinsic reward on inhibitory control, and how the two may interact.

Fluctuations in inhibitory control

Previous research has recognised inhibitory control as a risk factor for alcohol use disorders, yet such studies do not explain whether an individual's ability to inhibit is stable over time. Empirical studies demonstrate that inhibitory control is subject to short-term fluctuations within individuals (De Wit, 2009; Jones et al., 2013), suggesting an individual's ability to exert inhibition is fluid. Fluctuations can occur in response to environmental, psychological and physiological triggers (De Wit, 2009; Inzlicht & Berkman, 2015; Jones et al., 2013). Examples of environmental cues are substance-cue exposure and rewards. De Wit (2009) proposed that such fluctations present a particular risk to abstainers, as experiencing a short-lapse in inhibitory control may lead to relapse of substance use. However, theories of disinhibition are based on a simplistic view of inhibitory control, i.e. inhibition is reactive stopping. As a result, a second key aim of this thesis was to explore if some of the environmental and psychological mechanisms suggested by previous research (Fleming & Bartholow, 2014; Jones et al., 2013; Weafer & Fillmore, 2012), in particular how alcohol-cue exposure and reward lead to short-term fluctuations in inhibitory control, and if inhibitory performance is related to an individual's alcohol consumption.

Motivation: how value-based choices influence health behaviours in a mechanistic model

Dual process theories (Evans, 2008; Wiers et al., 2007) have been used to explain inhibitory control behaviour in both the general population and adolescents involved in substance use behaviours, and are not dissimilar to neurobiological theories. Such models propose that excessive alcohol use is the result of two competing systems -: an appetitive and an executive system (Dawe et al., 2004; Goldstein & Volkow, 2002). The appetitive system becomes sensitized following repeated exposure to addictive substances driving automatic drug seeking (Robinson & Berridge, 1993). Approach behaviours can be observed in both implicit and explicit behaviours, and have a bidirectional effect. For example, cues can motivate individuals to elicit approach behaviours, whereas approaching and consummatory behaviour can increase the salience of such cues (Chein & Schneider, 2005; Deutsch, Gawronski, & Strack, 2006; Fleming & Bartholow, 2014). In support of this, individuals have been shown to exhibit greater motivation to consume alcohol and hold more positive expectancies if they already consume large amounts of alcohol (Hamonniere & Varescon, 2018; Jones, Corbin, & Fromme, 2001). Individuals with increased sensitization of appetitive systems can show increased attentional-bias (Field & Cox, 2008) and approach bias (Christiansen et al., 2012) for substance related stimuli.

A fundamental component of automatic behavioural control is that it develops slowly over time through regular stimuli-reinforcement associations, with stimuli outside of conscious awareness being subject to reinforcement (Garavan et al., 2000; Pessiglione et al., 2008; Volkow et al., 2006). However, evidence from neuroimaging suggests that goal maintenance is regularly updated in relation to reward responses available (D'Ardenne et al., 2012). To reach goals, individuals may have to use cognitive strategies other than effortful inhibition (Fujita, 2011), such as strategies to avoid situations for inhibition failures (Duckworth, Gendler, & Gross, 2016) . As such, it is argued that dual-process models over-rely on a dichotomy of two systems, when in reality information is continuously processed in parallel. As such, they do not explain the fine-grain mechanistic nature of inhibitory control (Keren & Schul, 2009; McClure & Bickel, 2014).

Berkman, Hutcherson, Livingston, Kahn, and Inzlicht (2017) put forward a model of inhibitory control in which an individual's decisions are viewed as a series of competing value-based choices. Inhibitory control is based around a focal goal, yet behaviour is dictated by gains (e.g. money, social approval), costs (e.g. effort, opportunity costs), transforming objects into subjective value in predictable ways (e.g. discounting delayed rewards, penalizing effort), and enacting the most value option. In respect to health behaviours, it accounts for the dynamic relationship between both cognitive and personality factors, and subsequent health behaviour (Berkman, 2018). Behavioural economics state that the value (or demand) for a substance is determined by the benefit-to-cost ratio compared to the ratio for other activities an individual may engage in (Bickel, Johnson, Koffarnus, MacKillop, & Murphy, 2014; Murphy & Dennhardt, 2016). Distortion of the valuation process through reinforcement of substance-related expectancies and outcomes leads to the development and persistence of substance misuse and inhibitory deficits(Bickel et al., 2014; Lamb & Ginsburg, 2018). Substance users are shown to place increased value on substance use (Hogarth & Hardy, 2018) and to exhibit a diminished reward response to non-substance rewards (Meshesha, Pickover, Teeters, & Murphy, 2017).

Alcohol-cue exposure

Exposure to substance-related cues (for example, the smell or sight of beer) have been well established to induce craving, influence physiological responses (such as increased heart rate or salivation (Liu et al., 2021; Perry, Zbukvic, Kim, & Lawrence, 2014; Pomerleau, Fertig, Baker, & Cooney, 1983; Zhao et al., 2012), and behavioural responses (such as increased substance use in substance users (Carter & Tiffany, 1999; Veilleux & Skinner, 2015)). This behavioural response is coined 'cue reactivity' and is proposed to be an underlying factor involved in the transition to substance dependence (Drobes, 2002; Stein, Fey, Koenig, Oehy, & Moggi, 2018) and relapse (Goldstein & Volkow, 2002; Stacy & Wiers, 2010). Exposure to alcohol-related cues has been shown to increase alcohol seeking in samples without alcohol use disorder (Christiansen, Townsend, Knibb, & Field, 2017; Heinze, Wölfling, & Grüsser, 2007; MacKillop & Lisman, 2007). However, there are discrepancies. For example, Thomas, Drobes, and Deas (2005) showed that adolescents with substance dependence demonstrated increased salivation to substance-related cues compared to controls, but heart rate did not differ between the groups.

Associative learning is generally accepted as the underpinning mechanism that evokes the above responses to substance-related cues (Field & Jones, 2017). Incentive-sensitization

theory argues that associations are built between substance-related cues and the substance's positive effects (Robinson and Berridge 1993). As a result, substance-related cues gain increased salience to the user, promoting substance-seeking and consumption. Supporting this theory, evidence has suggested increased amounts of dopamine are released following exposure to substance-related cues (Boileau et al., 2007; Koob & Volkow, 2010). Also, experiments which use Ecological Momentary Assessment (EMA) protocols have demonstrated that exposure to substance-related cues increases craving and substance use in naturalistic environments (Fatseas et al., 2015; Serre, Fatseas, Swendsen, & Auriacombe, 2015). However, in contradiction to such findings, lower dopamine release or receptors have been shown in those diagnosed with addiction (Martinez et al., 2004; Martinez et al., 2007). Differences observed in dopamine levels in response to alcohol may be due to populations sampled, methodological differences, and differing alcohol dosages administered, making it difficult to draw conclusions (Ma & Zhu, 2014).

The situational specificity hypothesis (Wall, Hinson, McKee, & Goldstein, 2001; Monk & Heim, 2013) postulates that drinking behaviour is determined by drinking cues and environments. The use of EMA and semi-naturalistic methods allow for the direct testing of the cue-reactivity paradigm, which is context dependent (Qureshi, Monk, Pennington, Li, & Leatherbarrow, 2017; Ramirez, Monti, & Colwill, 2015). Alcohol-related cognitions have been found to fluctuate, using indirect measures, as a result of natural or laboratory-manipulated contextual cues (Monk, Sunley, Qureshi, & Heim, 2016; Read & Curtin, 2007). This research converges to suggest that environmental cues may serve as risk factors, in which alcohol-related stimuli present as a risk factor for binge-drinking episodes (Ryan, Kreiner, Chapman, & Stark-Wroblewski, 2010) and potentially facilitate relapse following abstinence (Robinson & Berridge, 2008; Weerts, Goodwin, Kaminski, & Hienz, 2006).

Researchers disagree on other psychological mechanisms that underpin this relationship (Field & Jones, 2017), although there is evidence indicating that inhibitory control is involved in the mechanism. Papachristou, Nederkoorn, Havermans, van der Horst, and Jansen (2012) reported that the relationship between alcohol-cue exposure and craving in heavy drinkers was moderated by inhibitory control. Specifically, those with poorer response inhibition reported increased craving in comparison to those with better response inhibition following exposure to alcohol-cues. However, this moderation was not observed in light drinkers. In an extension of this line of thinking, Field and Jones (2017) found that increases in disinhibition and craving partially mediated the effect of alcohol-cue exposure on the amount of alcohol consumed on a bogus taste-test in non-dependent drinkers.

However, findings are not consistent; in dependent populations, studies have shown that alcohol-cue exposure impairs inhibitory control (Gauggel et al., 2010; Noël et al., 2007), however some research suggests no difference in inhibitory control as a result of alcohol cue-exposure (Mainz et al., 2012). In both problem and non-problem samples, research shows that embedded alcohol-cues into Stop-Signal and Go/No-go tasks has demonstrated that inhibitory control experience short-term deficits (Kreusch et al., 2013; Muraven & Shmueli, 2006; Petit, Kornreich, Noël, Verbanck, & Campanella, 2012). However, other research has failed to replicate these findings. For example, when exposed to alcohol cues - both of images (Nederkoorn et al., 2009) and physical alcohol cues (Jones, Rose, Cole, & Field, 2013) - no impairments of inhibitory control was observed in non-dependent drinkers.

The effect of alcohol-cue exposure has been the subject of a recent meta-analysis (Jones et al., 2018) which found that the effect was small (Standardised mean difference= -

0.21, 95%CI=-0.32, -0.11) yet robust across Stop-Signal, Anti-Saccade and Stroop tasks. Therefore, it is plausible that increased alcohol-seeking following alcohol-cue exposure is a result of fluctuations in inhibitory control. Such fluctuations may prevent self-regulation of behaviour in the form of response to alcohol, and lead to substance-seeking behaviour (De Wit, 2009; Jones et al., 2013). This effect is presumed to be intensified when an individual is under the influence of alcohol (Adams, Ataya, Attwood, & Munafò, 2013; Weafer & Fillmore, 2015). An explanation for this is because of the increased salience of these cues during intoxication (Field, Wiers, Christiansen, Fillmore, & Verster, 2010). However, Duka and Townshend (2004) only found increased attentional bias to alcohol-cues at low alcohol dose (0.3g/kg). Despite these inconsistencies, deficits experienced in the presence of alcohol cues are exacerbated under intoxication, driving a "loss of control" over drinking (Weafer & Fillmore, 2015).

The research described above focuses on 'reactive' inhibitory control, which may explain discrepancies in the literature. Alcohol-cue exposure has the ability to induce cognitive biases, which in turn can influence proactive slowing and reactive stopping (Stacy & Wiers, 2010). Sharma (2017) demonstrated that following alcohol-cue exposure on a modified Stroop task, heavy drinkers (compared to light drinkers) displayed detrimental task performance. The performance of heavy drinkers suggests they are employing reactive control as a late correction mechanism (Braver, 2012), while light drinkers filter out context of prior cues through proactive control. Similar findings have been found for individuals with Cannabis Use Disorder (Brevers et al., 2018) compared to healthy control. In contrast, users did display enhanced proactive control to cannabis cues, yet this is attributed to their motivation to reduce their use, or to stop using cannabis entirely. Therefore, additional research should investigate the effect of alcohol-related cues on both proactive and reactive control, and if inhibitory control performance following alcohol-cue exposure can account for future alcohol-seeking behaviour.

Reward and inhibitory control

The transient effects of motivation on inhibitory control are well studied in relation to substance and non-substance stimuli as motivational factors (Hogarth & Field, 2020). However, various theoretical approaches account for substance use in respect to relative rewards available (Everitt & Robbins, 2016; Robinson & Berridge, 1993; Tiffany, 1990; Wise & Koob, 2014). In particular, neurocognitive theories account for the impairment of inhibitory control (as discussed previously), but also impaired foresight of future rewards (Bickel et al., 2017). Concurrent choice tasks are used to assess the value of the substance to the value of an alternative reward, such as money, with research suggesting that as the non-substance reward is increased, substance choice decreases (Cassidy, Tidey, Kahler, Wray, & Colby, 2015).

Given that substance reward value decreases when non-substance reward increases, the prospect of an alternative non-substance reward may increase inhibition towards substance-related stimuli, Inhibitory control is usually measured in environments devoid of extrinsic rewards (Hanne Schevernels et al., 2015). However, research suggests that reward induces increased proactive control in a top-down fashion (Chelazzi, Perlato, Santandrea, & Della Libera, 2013; Pessoa & Engelmann, 2010). In non-dependent samples, the prospect of reward can improve both proactive and reactive inhibitory control (Greenhouse & Wessel,

2013; Rosell-Negre et al., 2016; Boehler, Schevernels, Hopf, Stoppel, & Krebs, 2014; Wilbertz et al., 2014)). Schevernels et al. (2015) demonstrated that when there is a prospect of reward inhibitory control is faciliated (via proactive and reactive control); using a modified Go/No-go task in which Event-Related Potentials (ERPs) were examined. They found increased stop-signal attention under reward-related trials, suggesting that participants were actively preparing for a stop-signal. However, findings are not consistent (Demurie et al., 2016; Paschke et al., 2015; Shanahan, Pennington, & Willcutt, 2008), with some reporting that the prospect of reward is detrimental to inhibitory performance (Marini, van den Berg, & Woldorff, 2015; Yamaguchi & Nishimura, 2019). Discrepancies in findings may be the result of methodological differences particularly with the reward used, varying between points and monetary rewards, that may exert differing effects on inhibitory control.

Little research has examined the effect of reward on inhibitory control in substance-use populations, despite research suggesting hypersensitive reward systems (Joyner et al., 2019; Volkow et al., 2010). Chung et al. (2011), using a reward anti-saccade task, found enhanced inhibitory performance in the presence of reward in individuals with substance-use disorder. The authors argue that the inhibitory response can be enhanced with reward. However, findings are not consistent in samples of individuals with substance use problems. Charles-Walsh, Upton, and Hester (2016) examined inhibitory control in opiate-dependent participants using an incentivised Go/No-go task, and found no effect of reward on inhibitory control. The authors noted that participants were in various stages of withdrawal, and drug abstinence is known to affect inhibitory control (Charles-Walsh, Furlong, Munro, & Hester, 2014; Fu et al., 2008). Additional research is required to clarify the effect of reward on inhibitory control, both in standard laboratories but also in the presence of alcohol stimuli acting as potential distractors. Clarifying the effect of extrinsic rewards may be able to increase the probability of successful inhibition in alcohol rich-environments by increasing the value of the inhibition behaviour (Monterosso, Piray, & Luo, 2012).

Summary of Aims and Hypotheses

The overall aim of this thesis was to investigate the effect of environment and rewards on inhibitory control to develop a more complex model of inhibitory control in heavy drinkers. Specifically, to investigate whether the prospect of an extrinsic reward can improve inhibitory control, in controlled lab and real-world environments. In three of the studies presented in this thesis, heavy drinkers were recruited, with the fourth and final study recruiting an adolescent sample (aged 16-18 years old). In all studies, individuals with a current or previous diagnosis of substance use disorder were excluded. This was in part due to ethical constraints, but also to allow examination of individuals who are at risk of developing a substance use disorder.

Chapter three presents the findings of a meta-analysis assessing the relationship between inhibitory control and reward. The research presented in this thesis aimed to examine how robust the effect of reward on inhibitory control, and if it was moderated by the following factors, task type, reward type, clinical diagnosis, or age.

Chapter four sought to examine the effect of reward on inhibitory control in a heavy drinking sample (i.e. reporting alcohol consumption in excess of 14 units a week) across differing

environments and inhibitory loads. Reward was examined across varying magnitudes and probabilities to examine sensitivity of participants to the prospect of reward.

Chapter five was an Ecological Momentary Assessment (EMA) study, using a naturalistic design to examine fluctuations in inhibitory control and alcohol use in the real-world. This research aimed to examine whether certain locations or cues would induce momentary fluctuations in inhibitory control and impact subsequent alcohol use. A baseline session was administered in order to control for exogenous variables in EMA sessions, and to enable cross-validation with the laboratory findings.

Finally, chapter six sought to examine executive functioning, inhibitory control and working memory in adolescents. An adolescent sample was recruited to extend the findings from previous work, in particular to examine if there were specific predictors that can identify those who may transition to heavy drinking.

Chapter 2

General Methods

Baseline measurements were taken at the beginning of each study. The majority of the measures were consistently used across all the studies, and so the format of each and the rationale for their use, are described here in detail once (with any adjustments described in in the relevant chapter), as well as their respective psychometric properties.

Two Week Timeline Follow-Back (TLFB)

The TLFB (Sobell & Sobell, 1992) assesses an individual's alcohol consumption for the previous fortnight in U.K. units. Participants self-report their alcohol consumption on a day-to-day basis. Participants are provided with a guide for the number of units in the most common alcoholic drinks and were allowed to consult their mobile phones and/or diaries to aid their recall. The TLFB allows for retrospective assessment of the quantity and frequency of alcohol consumption. The paper-and-pencil version has been found to be as reliable as interview methods when assessing alcohol consumption behaviour (Hoeppner, Stout, Jackson, & Barnett, 2010). The TLFB can be administered over a long period of time, 30 days (Henges & Marczinski, 2012) or up to 12 months (Sobell & Sobell, 1992), yet these suffer low completion and accuracy rates in social drinkers (Hoeppener et al., 2010). Hoeppener et al. (2010) suggests that shorter recall (two-week version) is both more accurate and is a sufficient timeframe to capture an individual's typical drinking behaviour, in comparison to 30-day drinking diaries. Test-retest reliability of shorter TLFBs (sub 4 weeks) on the whole is quite high in dependent and non-dependent samples, with reported correlation coefficients between .75 to .90 (Cohen & Vinson, 1995; M. B. Sobell, Sobell, Klajner, Pavan, & Basian, 1986). Test-retest reliability of shorter TLFBs (less than 4-weeks) is overall strong in dependent and non-dependent samples, with reported correlation coefficients between .75 to .90 (Cohen & Vinson, 1995; Sobell et al., 1986).

Alcohol Use Disorders Identification Test (AUDIT)

The AUDIT (Saunders et al., 1993) was administered to assess hazardous drinking behaviour. The AUDIT is a ten-item scale (e.g. "*How often do you have a drink containing alcohol?*"), with each item scored from 0-4, giving a total possible score of 40. WHO guidelines state that scores ≥ 8 are indicative of hazardous or harmful use, with a risk for dependence. A score ≥ 20 is indicative of alcohol dependence and further investigation is advised. The AUDIT has been standardized internationally, allowing it to be used cross-culturally (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001). It has been found to have a

high degree of internal consistency, ranging from .75 to .97 (Reinert & Allen, 2007), and high test-retest reliability (Dybek et al., 2006). Allen, Reinert, and Volk (2001) discuss how the sensitivity of the AUDIT is comparable to and exceeds other alcohol screening measures, making it an effective measure of problem drinking and associated risk.

Barratt Impulsivity Scale (BIS)

The BIS (Patton et al., 1995) is a self-report measure of trait impulsivity. It is comprised of 30 questions, scored from 1 to 4 ('rarely', 'occasionally', 'often' and 'always'). The BIS has three subscales: Attention, Non-Planning and Motor Impulsiveness. Each subscale has a total score, and an overall BIS score can be calculated by adding all three subscales together, yielding a total possible score of 120. The BIS has been shown to be internally consistent in a variety of populations, including undergraduates (α =.82), substance abuse patients (α =.79) and general psychiatric patients (α =.83). The BIS has been demonstrated to have robust criterion validity and test-retest reliability (α = .83; r_s = .83) (Patton et al., 1995; Stanford et al., 2009). However, in relation to other behavioural measures of impulsivity there are discrepancies (McCarthy et al., 2016), and internal consistency has been found to be α =.79 (Orozco-Cabal, Rodríguez, Herin, Gempeler, & Uribe, 2010; Jessica Weafer, Baggott, & de Wit, 2013), suggesting that behavioural and self-report measures of impulsivity may measure different constructs.

Temptation and Restraint Inventory (TRI)

The TRI (Collins & Lapp, 1992) is a 15-item scale that assesses cognitive restraint, and motivation to reduce drinking. TRI loads on to two subscales, Cognitive Emotion Preoccupation (e.g. "*At times, do you find yourself unable to stop thinking about drinking?*"; CEP) and Cognitive and Behavioural Control (e.g. "*Does seeing other people drink remind you of your efforts to control your alcohol consumption?;* CBC). These subscales were found to be moderately correlated to each other, yet they represent distinct constructs. The CEP measures how unsuccessful an individual is at regulating their drinking, while the CBC measures success of regulating. Both factors have been shown to predict weekly drinking (Collins, Koutsky, & Izzo, 2000). The whole TRI has a robust internal validity of α =.87, with α =.85 and α =.83 for the CEP and CBC subscales (MacKillop, Lisman, & Weinstein, 2006).

Chocolate use questionnaire

To measure implicit wanting of chocolate, an appetitive stimulus used in some studies, a scale was developed based on Tibboel et al. (2011). Chocolate use was assessed to control for a generalised increase in attentional bias for rewarding stimuli, resulting from

exposure to the prospect of a reward (Jones et al., 2012). This scale was designed to assess how many standard bars of chocolate an individual consumes in a week and has stored at home. Craving of chocolate was assessed using five statements, examining their liking, urge to eat, their wanting of chocolate, and if they could stop eating chocolate, which were answered using Likert scales ranging from 0 (never) to 7 (always). Based on answers provided, an index of chocolate consumption, chocolate stored, and chocolate craving was created, with a total possible score of 35.

Brief Self-Control Scale (BSCS)

The Brief Self-Control Scale (Tangney et al., 2018) was used to assess general trait self-control. It consists of 13 items, each with a 5-point Likert scale from 'not at all' (0) to 'very much' (5), yielding a maximum score of 65. The BSCS has been shown to successfully predict behavioural outcomes across multiple studies and populations (Baay, de Ridder, Eccles, van der Lippe, & van Aken, 2014; de Ridder, Lensvelt-Mulders, Finkenauer, Stok, & Baumeister, 2012). Ferrari, Stevens, and Jason (2009) proposed a two factor structure, comprised of general self-discipline and impulse control, which is supported by Maloney, Grawitch, & Barber (2012). The BSCS has been shown to be reliable and valid (Nebioglu, Konuk, Akbaba, & Eroglu, 2012).

Desire for Alcohol Questionnaire (DAQ)

The DAQ (Love, James, & Willner, 1998) was administered to assess cravings for alcohol, and has been shown to be sensitive to moment-to-moment changes in alcohol craving (Courtney et al., 2013). The abbreviated DAQ contains 14 items each on a 7-point Likert scale from 'strongly disagree' to 'strongly agree'. DAQ consists of three subscales that assess, strong desires/intentions to drink, negative and positive reinforcement, and ability to control drinking (Kramer et al., 2010). However, the factor structure has been found to be inconsistent (Pasche, Garner, Baldwin, & Sinclair, 2013). Courtney et al. (2013) report partial correlations, ranging from .43 -.50, between DAQ scores and alcohol craving during alcohol administration. In addition to this, Courtney et al suggest the DAQ Cronbach alpha scores are adequate in respect to reliability, ranging from .70 to .86. All three subscales are correlated with the alcohol symptom scale (.25-.64) in both clinical and non-clinical samples (Kramer et al., 2010).

Thirst VAS

. To account of differences in thirst, a 200mm VAS was administered, and scores on this were controlled for in subsequent analyses.The thirst VAS was a 200mm Visual Analogue Scale, rating "How thirsty do you feel?", from "Not at all" to "Extremely". Scores could range from 0mm to 200mm.

Funnelled debriefing (Field, Christiansen, Cole, & Goudie, 2007)

A funnelled debriefing was also used in all studies to assess demand characteristics. It consisted of the following open-ended questions: "What was the purpose of this experiment?", "The computer task was designed to….?", and "The purpose of the taste test was to…?". Participants were asked to write a few short sentences for each. This was used as sensitivity analysis as to whether a participant guessed the true purpose of a study.

Bogus taste test/ *ad-libitum* alcohol session

To assess an individual's alcohol-seeking behaviour an *ad-libitum* consumption session was used, disguised to the participants as a taste test. Across studies the procedure was consistent for the taste test. The taste-test paradigm was first administered to non-abstinent alcoholics, who were given 15 minutes to taste and rate an alcoholic and non-alcoholic beverage on adjectives (Marlatt, Demming, & Reid, 1973). Field and Eastwood (2005) refined the taste test for use in social drinkers, by reducing the amount of alcohol available and adjectives to rate up to four (e.g. pleasantness, taste strength, bitterness, gassiness).

At the beginning of each *ad-libitum* session, participants were given up to three units of alcohol across two different drinks, equal fluid amounts, and an equivalent amount of a soft drink (cola). The type of alcohol given corresponded to their preferred chosen alcohol drink (beer, cider, white wine, red wine, rose wine) as shown in the behavioural tasks. Alcohol was personalised to increase the internal reliability of the taste test (Christiansen, Mansfield, Duckworth, Field, & Jones, 2015a). Use of personalised alcohol stimuli increase the ecological validity, by providing alcohol they are more likely to consume in their everyday environment (Jones, Button, et al., 2016). Participants were explicitly told they could drink as much or as little as they wanted to. They were asked to rate both beverages on a scale of 0 (not at all) to 10 (extremely) on a set of four adjectives. The same four adjectives were presented for both beverages. The dependent measure was the amount of units consumed. Alcohol consumption score was calculated by alcohol consumed divided alcohol given multiplied by three. This measure was used rather than the amount in millilitres, to standardise the amount consumed to units

Leeman and colleagues demonstrated that alcohol consumption in laboratory-based studies is representative of naturalistic drinking behaviour (Leeman et al., 2009; Leeman et al., 2013)). However, little work has been done to assess the construct validity and reliability of the taste test. Jones, et al. (2016) provide evidence for construct validity as a measure for alcohol consumption in the lab. However, individual differences in AUDIT scores were not related to taste test consumption, suggesting the test may not be representative of hazardous drinking behaviour. Construct validity is potentially confounded by participants having poor recall or deliberately underreporting their typical consumption (Leeman et al., 2013; Monk, Heim, Qureshi, & Price, 2015). However, drinking diaries have been demonstrated to produce reliable and accurate data (Hoeppner et al., 2010) and drinking behaviour is generally consistent over time (Rueger, Trela, Palmeri, & King, 2012).

Chapter 3

A meta-analytic investigation of the effect of reward on inhibitory control

This chapter presents a meta-analysis of the effect of reward on inhibitory control, which has been published in *The Quarterly Journal of Experimental Psychology* (2021). The data and analysis script is freely available on the Open Science Framework (link presented in main text). The format of the original article has been modified to match the work presented in the thesis, however the content remains the same as in the published article. With regards to contributions, I designed the study which was approved by Andrew Jones. I ran searches, screening, data extraction, and wrote the manuscript. Graeme Knibb assisted with article screening and data extraction. Before the original submission, and in response to reviewer's comments, both co-authors provided feedback on the manuscript.

The aim of this study was to clarify the effect of reward on inhibitory control. I also aimed to investigate whether the effect of reward was moderated by: reward type, clinical diagnosis, or inhibitory control task. Lastly, I aimed to examine whether reward exerted specific effects on both reactive and proactive control respectively.

Abstract

Background: Contemporary theories predict that Inhibitory Control (IC) can be improved when rewards are available for successfully inhibiting. In non-clinical samples empirical research has demonstrated some support, however 'null' findings have also been published.

Objective: The aim of this meta-analysis was to clarify the magnitude of the effect of reward on inhibitory control, and identify potential moderators.

Methods: Seventy-three articles (contributing k = 80 studies) were identified from Pubmed, PsychInfo and Scopus, published between 1997 – 2020, using a systematic search strategy. A random effects meta-analysis was performed on effect sizes generated from inhibitory control tasks which included rewarded and non-rewarded inhibition trials. Moderator analyses were conducted on clinical samples (vs 'healthy controls'), task type (Go/No-Go vs Stop Signal vs Flanker vs Simon vs Stroop vs Anti-Saccade), reward type (monetary vs points vs other), and age (adults vs children).

Results: The prospect of reward for successful inhibition significantly improved inhibitory control (SMD=0.429 (95% CI= 0.288, 0.570), I^2=96.7%), compared to no reward conditions/groups. This finding was robust against influential cases and outliers. The significant effect was present across all inhibitory control tasks. There was no evidence the effect was moderated by type of reward, age or clinical samples. Moderator analyses did not resolve considerable heterogeneity.

Conclusions: Findings suggest that inhibitory control is a transient state that fluctuates in response to motivations driven by reward. Future research might examine the potential of improving inhibitory control through rewards as a behavioural intervention.

Introduction

Inhibitory control is defined as 'the (in)ability to change, suppress or delay a response that is no longer required under the current circumstances' (Logan et al., 1984a), and is thought to be a core component of executive functioning and impulsive responding (Bickel et al., 2012). Inhibitory control (also termed 'response inhibition') can be both either reactive and/or proactive (Braver, Gray, & Burgess, 2007). Reactive control refers to the act of stopping a response as a 'late correction' mechanism, whereas proactive control is the pre-planned behavioural alterations (e.g. response slowing) in anticipation of subsequent inhibition (Aron, 2011).

Computerised tasks have been developed for the assessment and operationalisation of inhibitory control in laboratory settings, with the most common being the 'Stop Signal' and 'Go/No-Go' tasks. Whilst these tasks measure slightly different forms of reactive inhibitory control (action cancellation vs. action restraint, see Eagle, Bari, and Robbins (2008), their component parts are similar. Both establish prepotent / dominant motor responses through promoting speeded reaction times to usually arbitrary cues. On a majority of trials, usually 75% or greater (Young, Sutherland, & McCoy, 2018), these responses are uninterrupted and thus prepotent or dominant responding is reinforced. However, on a minority of trials a 'stop signal' or 'no-go' cue is presented, prompting participants to withhold their prepotent motor response to the arbitrary cue. The inability to inhibit the prepotent response following presentation of the 'stop signal' or 'no-go' cue can be measured using commission errors (i.e. making a motor response to the arbitrary cue), or Stop Signal Reaction Time (SSRT: the unobserved latency of inhibition – see (Band et al., 2003). Other tasks such as the Stroop (Stroop, 1935) and Flanker tasks (Eriksen & Eriksen, 1974), measure the ability to override responses to congruent stimuli but are used less frequently in the literature (Diamond, 2013).

The development of these computerised tasks has led to a proliferation of studies examining IC across numerous psychological characteristics and behavioural outcomes. For example, estimates suggest that 80-90% of self-regulation attempts require some form of inhibition (Baumeister, 2014; Wilhelm Hofmann, Schmeichel, & Baddeley, 2012), highlighting a key role in behavioural adaptation and human survival (Verbruggen et al., 2014). Previous research has demonstrated that effective IC is associated with increased happiness and wellbeing (Hofmann, Luhmann, Fisher, Vohs, & Baumeister, 2014), intelligence (Polderman et al., 2009), and psychosocial functioning (Anzman-Frasca, Francis, & Birch, 2015). Whilst poorer inhibitory control is associated with numerous maladaptive behaviours and outcomes such as alcohol dependence (Rubio, Jiménez, Rodríguez-Jiménez, et al., 2008), incidence of overweight/obesity (Blanco-Gómez et al., 2015), poor educational attainment (Caspi et al., 2016) and crime (Vazsonyi, Mikuška, & Kelley, 2017).

The majority of published research considers inhibitory control as a trait-like variable, stable within individuals over long-periods. However, more recently research suggests there are both internal and external factors which might cause transient changes in stopping responses (Jones et al., 2013; Keren & Schul, 2009), which might better predict individual differences. For example, Berkman et al. (2017) propose that inhibitory control is a value-based process, and represents a trade-off between short and long-term rewards (Duckworth et al., 2016). This process involves assigning a momentary value for given behaviours, gains (e.g., money, or social approval) and costs (e.g., effort, and opportunity costs), to determine whether inhibition is required. Research has sought to enhance the "gains" valuation through the prospect of extrinsic or intrinsic rewards (e.g. extrinsic reward may be the prospect of money while intrinsic reward may be the desire to lose weight) (Duckworth, Milkman, & Laibson, 2018). This suggests the role of motivation is key in the expression of IC processes (Poulton, Mackenzie, Harrington, Borg, & Hester, 2016).

A number of studies have examined the role of motivation (through the prospect of obtaining rewards) on general cognitive performance, including reaction times, working memory, and task switching (Jimura, Locke, & Braver, 2010; Umemoto & Holroyd, 2015) all of which may have a downstream influence on inhibitory processes (Miyake & Friedman, 2012; Snyder, Miyake, & Hankin, 2015). Indeed, recent work has examined whether direct rewards for successful inhibition can improve inhibitory control. For example, Boehler et al. (2014) used a modified Stop Signal task in which the colour or the Stop Signal indicated whether inhibition would be rewarded or not. They demonstrated that on reward-related stop trials inhibition (measured using Stop Signal Reaction Time: the unobserved latency to inhibit behaviour) was greater than on reward-unrelated trials (see similar findings, (Chiew, Stanek, & Adcock, 2016; Geier & Luna, 2012; Ma et al., 2016; Schevernels, Bombeke, Krebs, & Boehler, 2016). In a modified Go/No-Go task (the Monetary Incentive Delay task; (Demurie et al., 2016) participants were provided information at the beginning of each trial as to the magnitude of monetary rewards available ('No reward', 'Medium Reward', 'High Reward'). Social, as well as monetary rewards, which consisted of positive feedback (e.g., 'You're a champion' for high rewards) were also available. In this case, the effect of rewards did not influence inhibition performance (see similar findings, (Michałowski, Koziejowski, Droździel, Harciarek, & Wypych, 2017; Paschke et al., 2015; Schevernels et al., 2015; Shanahan et al., 2008). Furthermore, some studies have reported the presence of reward being *detrimental* to IC (Marini et al., 2015; R. S. Williams, Kudus, Dyson, & Spaniol, 2018; Yamaguchi & Nishimura, 2019), possibly due to a break in attentional focus caused by reward stimuli (Wang, Li, Zhou, & Theeuwes, 2018). Finally, studies have examined whether the presence or magnitude of reward interacts with clinical diagnoses (e.g., Attention Deficit Hyperactivity Disorder, Substance Use Disorder), however these effects are also equivocal (Charles-Walsh et al., 2016; Chung et al., 2011; Rosell-Negre et al., 2016).

Given the considerable amount of research in the area and the inconsistent pattern of findings across individual studies, our aim was to conduct a meta-analysis on the effects of reward on IC in order to clarify the magnitude of the effect. We also aimed to examine potential moderators of the effect, including; type of task used (Stop Signal, Go/No-Go, Anti-saccade, Flanker, Simon, Stroop), type of reward (monetary, points, or other), clinical samples vs non-clinical samples, and age (adults, children), in an attempt to explain potential heterogeneity of published findings. We hypothesised that the presence of rewards during inhibitory control tasks would improve subsequent inhibitory control. We did not make any directional hypotheses in regard to moderators. This meta-analysis was pre-registered on the Open Science Framework (see – https://osf.io/5hbqu/) following the development of our systematic search terms, but prior to formal searches being carried out.

Method

Search Strategy

We searched three electronic databases: Scopus, Pubmed and PsycInfo in September 2018. Searches were updated in December 2020. The following search terms were used 1) *response inhibition* OR *inhibitory control* OR *disinhibition* OR 2) *stop signal* OR *stroop* OR *go/no** OR *flanker* OR *antisaccade OR simon task*, as well as 3) *reward* OR *incentive**. Searches were limited to human participants, published in English, and between years 1978-2020. The reference list of each identified paper was examined for any eligible articles not identified through our search strategy, and this led to the addition of one further article (Asci, Braem, Park, Boehler, & Krebs, 2019).

Eligibility Criteria

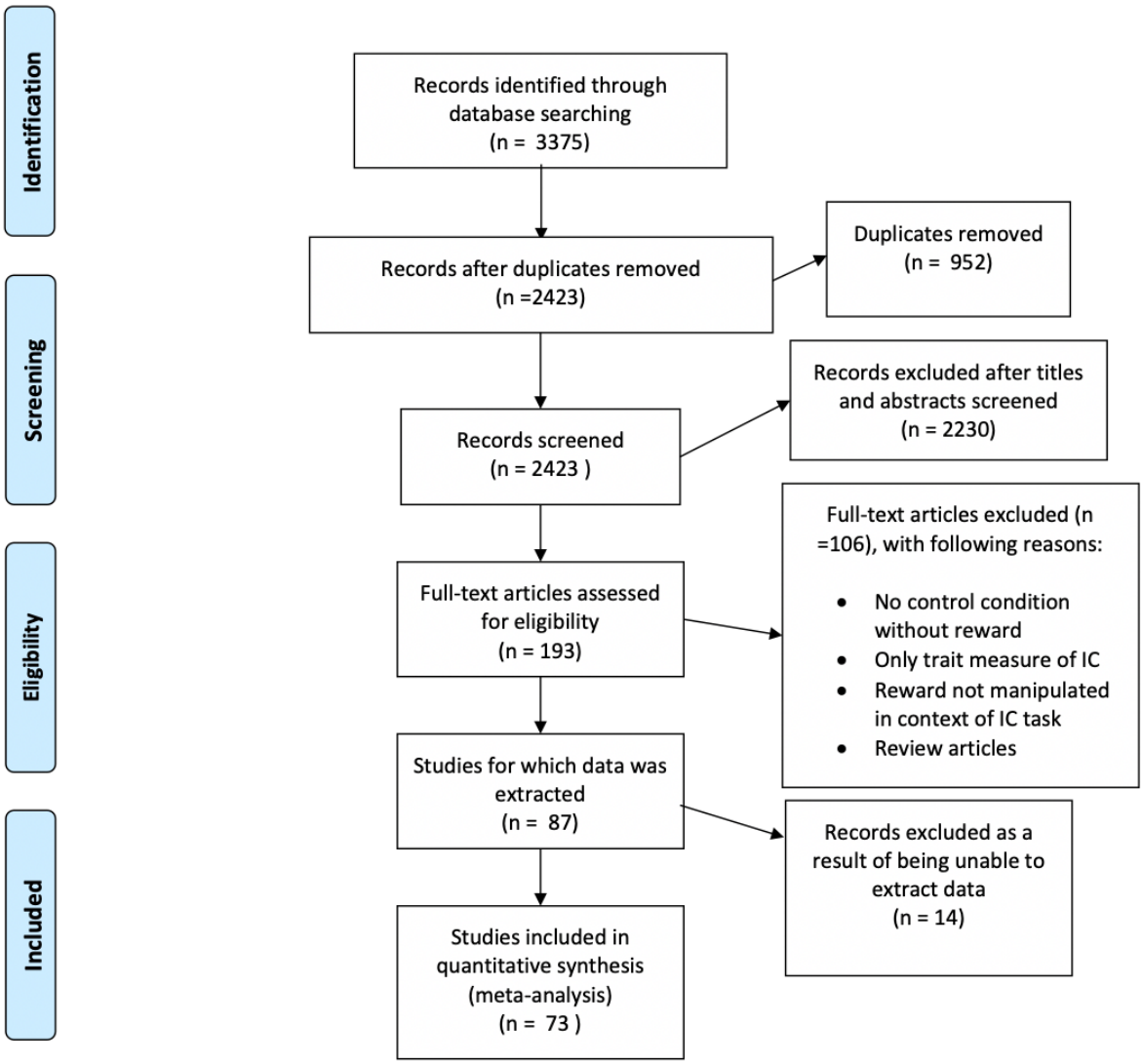
Studies were eligible for the meta-analysis when the following criteria was met. First, the study had to include a validated behavioural measure of IC (outlined in (Diamond, 2013),

either a; Stop Signal Task (SST), Stroop, Go/No-Go (GNG), Flanker, Antisaccade, Simon task. Second, the presence of reward for inhibitory performance (e.g., commission errors, Stop Signal Reaction time, Incongruent trials) was manipulated, e.g., some inhibition/incongruent were rewarded, and others were not. Studies were excluded if there was a reward condition without a control (no reward condition).

Data extraction and coding

The searches yielded a total of 2422 unique papers, an additional one paper was added following reference list searches of included articles. Titles and abstracts of these papers were examined in relation to inclusion criteria, resulting in 193 articles that were eligible for a full-text screening. Following full text screening 87 articles were eligible for data extraction to be used for the meta-analysis, 14 studies (16.09%) were excluded due to no reply to data requests, and 73 articles (80 effect sizes) were included. See supplementary material for the full table of studies included. The PRISMA flow chart can be seen in Figure 1.

Figure 1 PRISMA diagram of systematic search results.



Coding of studies

SB and GK coded and extracted all 73 articles, this included sample characteristics (gender distribution, age, clinical diagnosis), methodological information (measure of inhibitory control, reward manipulation), moderator information, and inhibitory control outcome (mean RT/error rate/accuracy rate for reward and no reward condition). For the Stop Signal Task we extracted SSRTs; Go/No-Go tasks we extracted error / accuracy rates; Antisaccade extracted error / accuracy rates; for the Stroop, Simon and Flanker tasks we used

incongruent RTs (as Prinzmetal, McCool and Park, 2005; demonstrate an increased sensitivity for RTs in cue-driven tasks).

Studies were coded as either adult samples, aged 18 years and above, or child samples if participants were younger than 18 years old. We examined whether studies recruited a significant clinical sample (e.g., ADHD, SUD, Autism Spectrum Disorder: see Appendices 2), vs 'healthy controls'. Given the heterogeneity in clinical samples we also conducted separate analyses on ADHD samples vs healthy controls, and SUD samples vs healthy controls separately.

For full text screening there was near perfect agreement between reviewers (Cohen's k = 0.95, $p < .01$) and substantial agreement for the data extraction stage (Cohen's k = .73, $p < .01$). Any disagreements were resolved by AJ. Information about each study is presented in Appendices 2.

Data analysis

We calculated the Standardised Mean Difference ($SMD = Mean^{REWARD} – Mean^{NON\text{-}REWARD} / SD^{POOLED}$) and the standard error (SE) of this difference, in order to conduct a random effects meta-analysis in 'metafor' for R. We used the SMD to ensure different outcome measures used by different inhibitory control tasks and articles were comparable. For within subjects designs (e.g. Michałowski et al., 2017; Schevernels et al., 2016; Shanahan et al., 2008) the standard error was adjusted using the correlation between the reward and control outcome (in line with Cochrane Recommendations ($SE(SMD) = \sqrt{(1/N)+(SMD^2/2N)} \times \sqrt{2(1- correlation)}$ (Cumpston et al., 2019). As the correlations between inhibition indices (reward and non-reward) were not readily available we chose a correlation of .70, as recommended by previous research (Khoury, Sharma, Rush, & Fournier, 2015; Rosenthal, 1991). However, we also conducted sensitivity analysis using coefficients of 0.50 and 0.90. Outliers were identified by standardising the effect sizes and examining any extreme values

at a < .001 (Z score = +/-3.30), and also examining whether 95% confidence intervals did not overlap those from any other effect size. We examined potential biases in the evidence base (e.g. publication bias) using Egger's test (Egger, Smith, Schneider, & Minder, 1997) for funnel plot asymmetry, and Trim and Fill analyses (Duval & Tweedie, 2000). We also conducted exploratory P-Curve analyses on the p-values of the Z tests (SMD / SE), using the 'dmetar' package (see Supplementary Analyses for P-Curve Figure). P-curve with right skew (e.g., larger distribution of *ps* < .01 - .025) are indicative of a likely 'true' effect when the distribution of p-values is uniformly distributed under the null hypothesis. If there is left skew (e.g., greater distribution of p-values between .025 - .050) this is indicative of selective reporting. Evidential value is demonstrated using the continuous and half-tests of the p-values (Simonsohn, Simmons, & Nelson, 2015)

The meta-analysis was performed using R (R Team). Datasets and the analysis script are available on OSF. Some papers reported multiple studies (e.g., (Hardin, Schroth, Pine, & Ernst, 2007; Padmanabhan, Geier, Ordaz, Teslovich, & Luna, 2011; Scheres, Oosterlaan, & Sergeant, 2001; Sinopoli, Schachar, & Dennis, 2011), as such the primary analysis included 80 effect sizes. The degree of heterogeneity was assessed using I^2. We used the following cut offs for heterogeneity: <25% low, 25-50% modest, and >50% high (Higgins, Thompson, Deeks, & Altman, 2003). In our pre-registration we stated we would also examine proactive control, however very few papers alluded to or measured proactive control, relative to reactive control. Therefore, we were unable to follow this up.

Results

Study characteristics

The majority of studies employed a within-subject (repeated measures) design, in which participants completed the measure of inhibitory control under both reward and non-

reward conditions (e.g., (Charles-Walsh et al., 2016; Marini et al., 2015; Scheres et al., 2001). We also identified 4 studies that used a between subjects design, in which participants were randomly allocated to either the reward or non-reward condition (e.g. (Huguet, Dumas, & Monteil, 2004; Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009; Marx, Höpcke, Berger, Wandschneider, & Herpertz, 2013). A number of studies examined the effect of reward on IC in clinical populations, e.g. ADHD, Substance Use Disorder, and mental health (Byrne & Worthy, 2019; Hardin et al., 2007; Miyasaka & Nomura, 2019b).

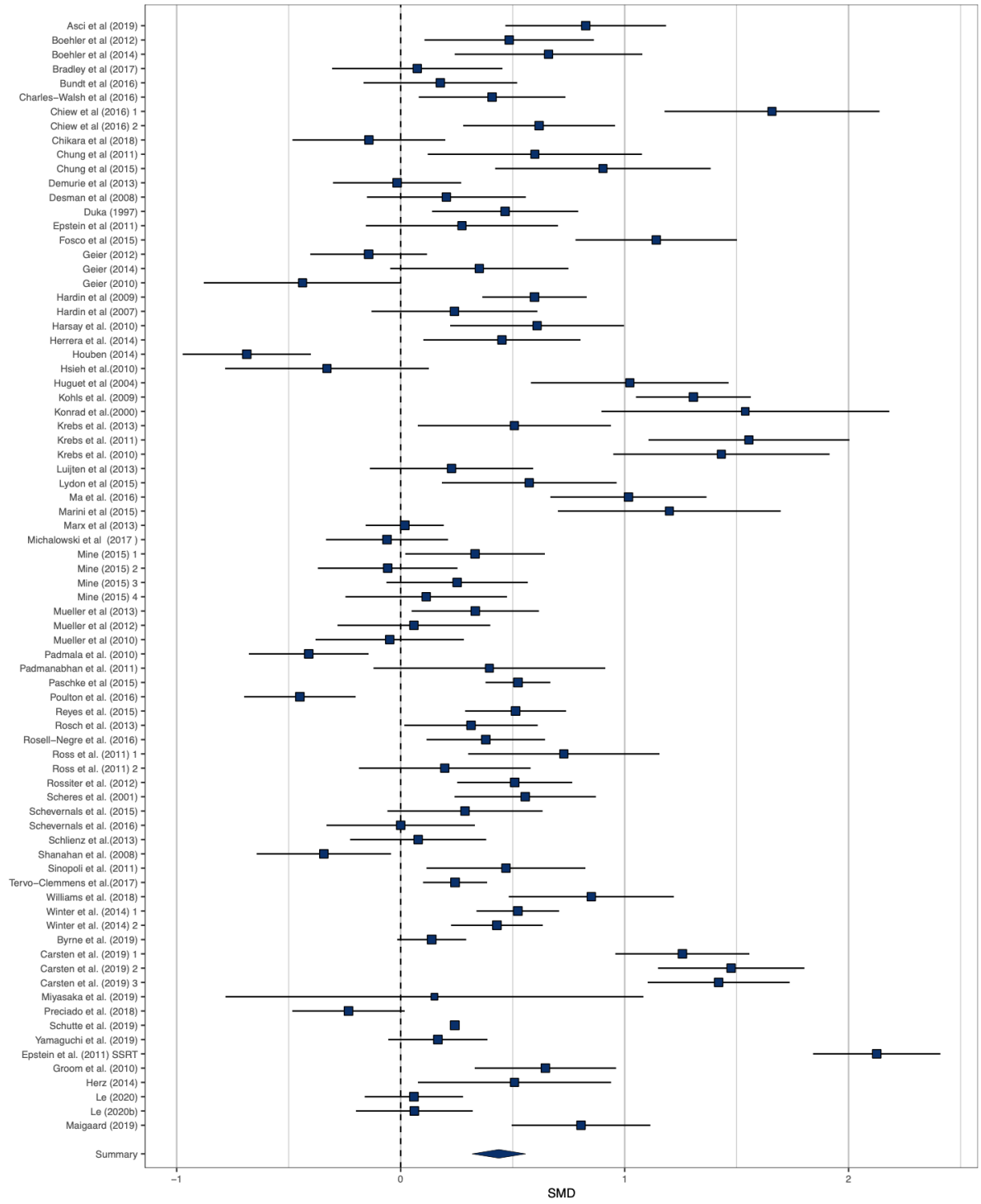
Of the studies included, the majority (78.75%) used monetary rewards (both hypothetical and real; e.g. (Poulton et al., 2016; Williams et al., 2018), a small number (17.50%) used 'points' as rewards (e.g. (Miyasaka & Nomura, 2019b)), and 3.75% used social rewards (e.g. (Kohls, Peltzer, et al., 2009). Inhibitory control was measured using a variety of tasks. Of the 80 effect sizes, *N* = 19 (23.75%) were measured using GNG; *N*=16 (20.00%) using SST; N= 13 (16.25%) using Flanker; *N*= 18 (22.50%) using Anti-saccade; *N* = 11 (13.75%) using Stroop and *N*= 3 (3.75%) Simon task.

Primary hypothesis: The effect of reward on inhibitory control (Fig 2)

Our main analysis consisted of 80 effect sizes. There was a small but statistically significant effect of the presence of reward improving inhibitory control (SMD=0.429 [95% CI= 0.288, 0.570]; Z=5.97; p<.001, I^2 = 96.7%). Two studies had a Z score +/- 3.30 and were removed, which did not substantially influence the effect size (SMD = .438 [95% CI = 0.319, 0.557), Z = 7.20, p < .001, I^2 = 95.2%. A leave-one-out analyses demonstrated limited variability in the effect size (min SMD = 0.413, max SMD = 0.453: all model ps < .001). Trim and Fill analyses did not impute any studies, but Egger's test was significant and suggested funnel plot asymmetry (Z = 2.339, p = .019: see Figure 3 for funnel plot). Exploratory p-curve analyses demonstrated evidential value (full curve Z = -23.98, p < .001

and half curve Z = -20.10, $p < .001$). Sensitivity analyses demonstrated that the effect size was SMD= 0.297 (95% CI= 0.194, 0.400) if the within-subjects correlation was imputed as r = .50, and SMD= 0.715 (95% CI= 0.522, 0.907) if the correlation was imputed as r = .90. Overall, there was a small, significant effect of reward on inhibitory control, which was robust to outliers and influential cases.

Figure 2: Forest Plot of effect sizes for rewarded vs non-reward inhibitory control



Potential moderators of the effect of reward on inhibitory control

Task type

Using data with outliers removed we conducted a-priori moderation on task type. There was a significant moderation effect ($X^2(5) = 16.79$, p = .005). There was a significant effect of reward all tasks: Go / No-go task (k = 18: SMD = 0.300 (95% CI = 0.127, 0.472), Z = 3.407, $p < .001$, $I^2 = 91.25\%$), Stop Signal task (k = 16: SMD = 0.410 (95% CI = 0.050, 0.770), Z = 2.233, $p = .026$, $I^2 = 95.97\%$), Flanker task (k = 13: SMD = 0.407 (95% CI = 0.130, 0.685), Z = 2.877, $p = .004$, $I^2 = 90.56\%$), Simon task (k = 3: SMD = 0.502 (95% CI = 0.126, 0.878), Z = 2.614, $p = .009$, $I^2 = 69.81\%$), Anti-saccade task (k = 18: SMD = 0.286 (95% CI = 0.128, 0.443), Z = 3.554, $p < .001$, $I^2 = 78.09\%$) and Stroop task (k = 10: SMD = 1.029 (95% CI = 0.728, 1.328), Z = 6.711, $p < .001$, $I^2 = 86.36\%$). The moderation effect was likely driven by the large effect sizes in Stroop tasks. Removal of the Stroop tasks from analyses made the moderator effect non-significant ($X^2(4) = 0.986$, $p = .912$). Notably, analysing the tasks separately did not substantially reduce the heterogeneity across effect sizes.

Age

We conducted exploratory moderation analyses on age. There were k= 28 effects from child samples (SMD = 0.515 (95% CI = 0.315, 0.714), Z = 5.053, $p < .001$, $I^2 = 92.18\%$) and k = 50 adult samples (SMD = 0.396 (95% CI = 0.247, 0.544), Z = 5.217, $p < .001$, $I^2 = 95.22\%$). There was no evidence of moderation ($X^2(1) = 0.877$, $p = .349$).

Reward type

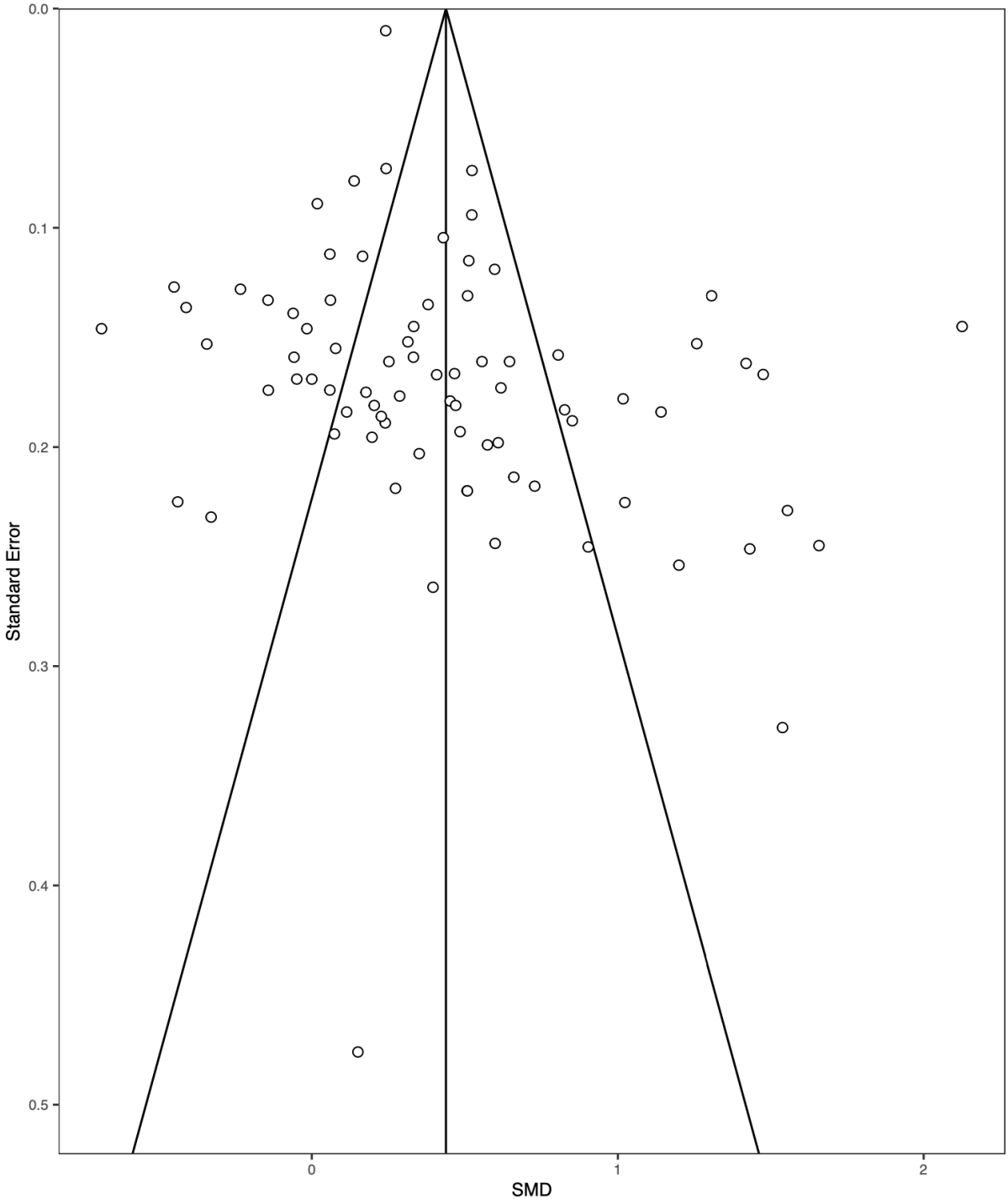
We conducted exploratory moderation analysis on reward type. There were k = 62 effects using monetary reward (SMD = 0.392 (95% CI = 0.266, 0.518), Z = 6.093, $p < .001$,

I^2 = 94.68%), k = 13 effects using hypothetical 'points' (SMD = 0.586 (95% CI = 0.220, 0.952), Z = 3.138, *p* = .002, I^2 = 93.02%), and k = 3 effects using 'other' rewards (SMD = 0.747 (95% CI = 0.208, 1.287), Z = 2.716, *p* = .007, I^2 = 94.79%). There was no evidence of moderation ($X^2(2) = 2.863$, *p* = .239). Again, there was limited evidence these moderator analyses reduced heterogeneity in the effect sizes.

Clinical samples

We conducted exploratory moderation analysis on clinical samples (vs 'healthy controls'). There was no evidence of moderation ($X^2(1) = 2.179$, *p* = .140). When examining ADHD samples vs healthy controls there was no evidence of moderation ($X^2(1) = 0.210$, *p* = .646. Similarly, when examining SUD samples vs healthy controls there was no evidence of moderation ($X^2(1) = 0.609$, *p* = .435).

Figure 3 Funnel plot of the effect sizes plotted against the standard error in the meta-analysis.



Supplementary analyses: Statistical power of included studies

Based on the pooled effect size of SMD =.429, a within-subjects comparison would require 35 participants to detect this effect (one-tailed, 1- β = .80, a = .05). Of the included studies 46 (57.5%) had a large enough sample size to reliably detect this effect.

Discussion

The current meta-analyses demonstrated that the prospect of reward can improve inhibitory control. The overall effect size was small-to-moderate, with considerable heterogeneity across the studies. Analyses indicated the effect of reward on inhibitory control was not moderated by clinical sample or type of reward used. Task type was a significant moderator of the effect of reward on inhibitory control, as the effect size was considerably larger in studies which utilised a Stroop task. The heterogeneity was not explained by any of our moderator variables.

The effect of reward on inhibitory control was consistent with recent hypotheses from theoretical models and research on healthy populations, suggesting rewards can improve momentary inhibitory control. Specifically, we find support for value-based models (Berkman et al., 2017) in which reward appears to increase the value for a given behaviour (inhibitory control), increasing the "gain' compared to the "cost" of inhibition (Duckworth et al., 2016; 2018). Findings also support dual-processes models (Evans, 2008), in which prospect of a reward appears to improve the slower deliberate reflective systems, linked to executive control. These findings are also in line with similar meta-analyses (Jones et al., 2018), providing support for theoretical models which suggest that inhibitory control is a transient variable, which is sensitive to internal and external factors (Jones et al., 2013; Keren & Schul, 2009).

The variability in effect sizes was not explained by clinical diagnoses in our data. This is surprising as the main clinical populations sampled were individuals with ADHD (Demurie

et al., 2016; Desman, Petermann, & Hampel, 2008; I. Ma et al., 2016) and substance use disorder (Charles-Walsh et al., 2016; Chung et al., 2011). Both disorders are characterised by disrupted reward processing (García-García et al., 2014; Tenenbaum et al., 2018), and with this particular sensitivity to rewarding stimuli we may have expected an enhanced effect of reward on inhibitory control for these sub-groups. In the case of SUD populations, the lack of effect of reward may be due to the severity of the condition, e.g., harmful use or dependency (Byrne & Worthy, 2019), yet we did not have enough data to reliably investigate any differences by clinical diagnosis. Similarly, there was no evidence the pooled effects were moderated by age of the participants, which may be surprising given inhibitory control improves with age into adulthood (Davis, Bruce, Snyder, & Nelson, 2003; Kray, Ritter, & Mueller, 2020; Macdonald, Beauchamp, Crigan, & Anderson, 2014).

The effect of reward was significantly moderated by task type, with seemingly larger effects in the Stroop task. Nevertheless, reward does not appear to have a consistent effect across separate inhibitory modalities. Complex measures of inhibitory control such as the Flanker require constant monitoring and updating of rules, further complicated by manipulations of reward, requiring enhanced top-down control leading to increased working memory demand (Garon et al., 2008). IC is dependent upon Working Memory Capacity (WMC: (Burnham, Sabia, & Langan, 2014; Vandierendonck, 2014), allowing maintenance of task goals (Munakata et al., 2011), with poorer WMC and increased WMC load impairing IC (Burnham et al., 2014; Kane & Engle, 2000; Unsworth, Schrock, & Engle, 2004).

A potential mechanism by which reward improves inhibitory control may be through attentional processes. Reward may increase the detection of the inhibitory signal (particularly when the inhibitory and reward signal are the same, see (Schevernels et al., 2015), leading to improved stimulus detection and reactive control (van den Berg, Krebs, Lorist, & Woldorff, 2014; Wang et al., 2018), however future research is needed to clarify these predictions.

Research should also attempt to elucidate any individual differences which might serve to moderate the effects, e.g. reward sensitivity (Capa & Bouquet, 2018). Unfortunately, we could not examine the effect of reward on reactive and proactive control due to lack of data available, therefore conclusions cannot be drawn as to the mechanism that reward effects inhibitory control, e.g., reactive or proactive control. Future studies should attempt to disentangle these effects to improve our overall understanding of inhibitory control (Verbruggen, et al. 2014).

Given reward appears to significantly improve inhibitory control, there are implications for the development of self-control interventions which focus on inhibitory control (e.g., inhibitory control training (ICT)). Recent meta analyses suggest that ICT leads to short term changes in behaviour (Vanessa Allom, Mullan, & Hagger, 2016; Jones et al., 2016). Reward may be used to increase the value of health-related cues (e.g., healthy foods) or devalue unhealthy behaviour-related cues (e.g., unhealthy foods) within these tasks. The opportunity to gain rewards for avoiding health risk and actively engaging in health promotion behaviour (Higgins, Heil, & Lussier, 2004; Vlaev, King, Darzi, & Dolan, 2019) may serve to improve associative learning and strengthen intervention effects (Schultz, 2002; Zhang, Manson, Schiller, & Levy, 2014).

We found evidence of bias in the literature following Egger's test. Whilst this suggests publication bias is having a persuasive influence on the literature, researchers have suggested such analysis are interpreted with caution particularly when there is heterogeneity in the data set (Shi & Lin, 2019). As such, researchers should endeavour to pre-register their work to provide increased transparency. There should be particular focus on replication attempts, as meta-analytic effect sizes are proposed to be nearly three times as large as registered replications (Kvarven, Strømland, & Johannesson, 2019).

We acknowledge the following limitations. First, we did not assess neuropsychological outcomes (such as Event Related Potentials) which were presented in some of the research (Chung et al., 2011; Schevernels et al., 2015). These outcomes may be more sensitive than behavioural measures and provide a deeper understanding of the role of reward on inhibitory control, allowing the formation of a more comprehensive mechanism. Second, reward was only assessed in the form of extrinsic motivation, e.g., in the presence of a reward specific cue. As such, future work should endeavour to examine the work of intrinsically rewarding appetitive stimuli to examine if similar effects on inhibitory control are observed as described here. There is a large amount of variability in the clinical populations in the current meta-analysis, which may vary in their responsiveness to reward, making it difficult to draw conclusions on the moderating effect of clinical diagnosis on reward and inhibitory control. Therefore, interpretation of the (lack of) findings should remain cautious. Future research should seek to look at specific populations in respect to this, to better our understanding on the potential moderating role of given clinical diagnoses.

To conclude the meta-analysis presented here suggests that the presence of reward can improve inhibitory control. Despite previous literature suggesting that individuals diagnosed with ADHD or substance use disorders have increased reward sensitivity, suggested a moderating role of diagnosis, we found no such evidence to support this. With reward significantly improving inhibitory control, this provides a potential avenue of treatment development for ICT, specifically producing a more prolonged behavioural change.

Chapter Summary

This chapter contributed to the overall aim of this thesis by meta-analysing the effect of reward on inhibitory control. Overall, there was a robust effect of reward on inhibitory control, namely improving inhibition. Findings suggest that inhibitory control is a transient state that fluctuates in response to external stimuli (reward). Going forward I sought to examine the effect of reward further due to the apparent robust effect.

Chapter 4

Emptying the file drawer: No consistent evidence for the interaction between appetitive cues and reward on inhibitory control

This chapter presents two experimental studies, both of which are pre-registered on Open Science Framework and being prepared for publication submission. Data is freely available on Open Science Framework (links provided in the main text). To summarize contributions, I designed both studies which were approved by Andrew Jones and Matt Field. I collected all data and wrote the manuscript; feedback was provided by Andrew Jones and Matt Field.

The aim of this chapter was to examine the effect of reward, appetitive stimuli and environment on inhibitory control in heavy drinkers. In addition to main effects of the aforementioned variables, I sought to examine whether there were any interactions to build a more complex understanding of their interaction and mechanism by which they influence inhibitory control.

Abstract

Background: Exposure to appetitive cues impair inhibitory control leading to a disinhibited state. Research suggests that inhibitory control can be facilitated by the prospect of reward. Little research has examined the effect of cues and reward together on inhibitory control in a heavy drinking sample.

Objectives: In two studies we investigated the effect of appetitive cues and reward exposure on inhibitory control.

Methods: Participants recruited were heavy drinkers in both studies. Both studies used a modified stop-signal task. Reward was manipulated using money and magnitudes (high vs low; study two). In respect to appetitive stimuli, the physical environment was manipulated, and task cues used were chocolate, alcohol and neutral stimuli.

***Results*:** Appetitive cues, particularly chocolate, impaired proactive (study one) and reactive (study two) control.In study two the prospect of gaining a reward improved proactive inhibitory control, yet there was no effect on reactive control in either study.

Conclusions: Reward or appetitive cues did not consistently influence both reactive and proactive inhibitory control, as such results should be interpreted tentatively.

Introduction

Inhibitory control is the (in)ability to suppress or change a response that is no longer appropriate (Logan & Cowan, 1984), and is thought to play a key role in self-regulatory behaviours (Baumeister, 2014). The act of inhibiting a response can be separated into multiple component parts; strategic (proactive) adjustments of behaviour, detection of a signal requiring inhibition within the environment, choosing the appropriate response and then enacting the response (reactive control) (Braver, 2012; Braver et al., 2007; Verbruggen, McLaren, & Chambers, 2014). In laboratory settings, inhibitory control can be measured using computerised tasks, such as the Stop Signal task. This task requires the inhibition of pre-potent motor responses following an auditory or visual stop signal (Logan & Cowan, 1984; Verbruggen & Logan, 2008).

Impairments in inhibitory control are thought to play a key role in the development and maintenance of both alcohol use disorders / problem drinking and also overweight and obesity (Goldstein & Volkow, 2011; Volkow, Wang, Fowler, & Telang, 2008; Yucel et al., 2019). Individuals with a diagnosis of alcohol use disorder and heavy drinkers display impaired inhibitory control in comparison to 'light drinkers' (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009; Smith, Mattick, Jamadar, & Iredale, 2014). Longitudinal research has demonstrated that poorer inhibitory control can predict future alcohol use in adolescents (Gordon Fernie et al., 2013), the likelihood of transitioning from heavy drinking to dependence (Rubio, Jiménez, Rodríguez-Jiménez, et al., 2008) and treatment success following relapse (Rupp et al., 2016). In parallel to the alcohol literature, inhibitory control is impaired in individuals with obesity compared to individuals within a healthy body weight range (SMD = .30, CI=0.00, 0.59: (Lavagnino, Arnone, Cao, Soares, & Selvaraj, 2016), and is thought to predict weight gain and weight-loss (Nederkoorn, Braet, Van Eijs, Tanghe, &

Jansen, 2006). However, it is important to note that several equivocal findings have also been published across both domains (Kamarajan et al., 2005; C. Nederkoorn et al., 2009).

Contemporary theories have begun to consider inhibitory control as a state variable (rather than a stable trait) which can fluctuate in response to immediate environmental (e.g. cue-exposure) and internal (e.g. motivation) circumstances (De Wit, 2009; Jones et al., 2013). A recent meta-analysis demonstrated that appetitive (food and alcohol-related) cue exposure had short-term transient impairments on inhibitory control (Jones et al., 2018), as these cues reliability trigger approach behaviours which are incongruent with inhibition (Field, Mogg, & Bradley, 2005; Kemps & Tiggemann, 2015). However, the effects of food-cues on inhibition were weak and unstable, and findings across both cue types were potentially skewed by small-sample sizes and publication biases in the literature (Jones et al., 2018).

The transient effects of motivation on inhibitory control have been investigated in a number of studies which have manipulated the presence of reward for successful inhibition. These studies have demonstrated that the presence of rewards improves inhibitory control in healthy individuals (Schevernels et al., 2015; Schevernels, Krebs, Santens, Woldorff, & Boehler, 2014; Wilbertz et al., 2014). A recent meta-analysis (Chapter 3) (Burton, Knibb, & Jones, 2021) suggests that the effect of reward on inhibitory control is robust (SMD=0.429, 95% CI= 0.288, 0.570), yet little work has examined the more finite effect on reactive and proactive control. Furthermore, research demonstrated that in heavy drinkers prospective rewards can increase both proactive (Rossiter, Thompson, & Hester, 2012) and reactive (Becker, Kirsch, Gerchen, Kiefer, & Kirsch, 2017) control. However, this relationship is not seen in users of opioids (Charles-Walsh et al., 2016) or cannabis (Chung et al., 2011).

Taken together, there is evidence to suggests that appetitive cues and motivation can influence inhibitory control in isolation. However, there has been little direct examination of the interaction between appetitive cues and motivation on inhibitory control. Such investigations are important as they could facilitate more adequately model the relative contributions to inhibitory performance under complex conditions more akin to the real world. For example, individuals may be exposed to alcohol-related cues but are motivated to refrain from drinking as they are attempting to save money. In this case, is the impairing effect of the alcohol-related cues negated by the internal motivation? If increasing the motivation to inhibit attenuates any cue-induced impairments in inhibitory control, this may inform interventions which target inhibitory control as a candidate mechanism of behaviour change (Allom, Mullan, & Hagger, 2015; Jones et al., 2016). Furthermore, individuals who drink problematically and are living with overweight or obesity demonstrate increased sensitivity to reward (Davis, Strachan, & Berkson, 2004; Lyvers, Czerczyk, Follent, & Lodge, 2009), however this relationship may be reversed in substance dependence (a decreased sensitivity to reward; Volkow et al., 2010). On the contrary, it is possible that the effect of cue-exposure (or motivation) is considerably larger in magnitude than motivation (or cue-exposure) suggesting some variables may have a stronger contribution to inhibitory processes that others. It is important to disentangle these predictions as it will further increase our understanding of the role of inhibition and its determinants.

Therefore, the aim of the current experiments was to examine the interactive effects of cue-exposure and reward on inhibitory control in heavy drinkers. In both studies we examined the combined role of cue-exposure and motivation (reward) on inhibitory control, allowing us to test i) the main effect of cue exposure, ii) the main effect of motivation and iii) the interaction between cue-exposure and reward on inhibitory control. Whilst the aims and

hypotheses were broadly consistent, the methods for each experiment differed slightly and are described in detail below.

Experiment one

In experiment one we manipulated the presence of reward for successful inhibition (reward present vs reward absent), and the type of visual cue presented during a stop signal task (alcohol, chocolate, neutral), across different environments (alcohol-related, neutral) in a mixed-design. Alcohol-related cues were matched to individual's preferred beverage in an attempt to increase ecological validity as in previous research (Christiansen & Bloor, 2014; Christiansen, Mansfield, Duckworth, Field, & Jones, 2015b). This study was pre-registered on the Open Science Framework (https://osf.io/pxy2a/).

Method

Participants

We recruited 98 participants (69 female; mean age 21.86 ±7.72 years old), from the university campus and wider community. The sample size was calculated to be a sufficient amount of participants based on a medium effect size d=0.51 with 80% power at an alpha =0.05 (Field and Jones, 2017). Inclusion criteria were age 18+, consume chocolate on at least one occasion per week and drink above UK government guidelines (> 14 units per week). Individuals were excluded from taking part if they self-reported a previous or current diagnosis of a substance use disorder or attention deficit hyperactivity disorder. Descriptive statistics for questionnaire measures are shown in appendix 3. All experiments were approved by the University of Liverpool Research Ethics committee.

Pictorial stimuli

Eight alcohol-related images, eight chocolate-related images (taken from previous studies (Christiansen et al., 2015b; Jones et al., 2012)), and eight neutral images (taken from the IAPT and used in Jones et al. (2015)) were used in the Stop Signal task, across all conditions. Different image-sets of alcohol-related images were included depending on individual's preferred beverage (chosen from; beer, cider, red wine, white wine or rose wine). Images were 135 by 95 mm.

Modified Stop signal task (based on Schevernels et al. (2015))

The beginning of each trial was signalled by a central fixation cross ('+') for 500 ms. The colour of this cross signalled whether inhibition would be rewarded (yellow = reward of 5 pence, blue = no reward). Following this, an alcohol, chocolate or neutral image appeared on the screen rotated either 5° clockwise or counter-clockwise (thus providing a number of reward and image combinations: alcohol+no reward, alcohol+reward, chocolate+no reward, chocolate+reward, neutral+no reward, neutral+reward). Participants had to signal the orientation of the rotation (by pressing the 'K' or 'D' keys for clockwise or counter-clockwise, respectively) as quickly and accurately as possible. On 50% of trials this categorization response was uninterrupted ('Go trials'). On the remaining 50% of trials a stop signal (a red '=') was superimposed over the image. Participants were informed they had to inhibit their response to this cue, if they saw it ('Stop trials'). They were informed not to wait for the stop signal, and to respond as quickly and accurately as possible.

A dynamic tracking algorithm was used to determine stop signal delays, separately for image-type and reward presence (in each experiment). The initial delay was 250 ms following the onset of the image which required a response. If inhibition was required and

successful, the stop signal delay increased by 50 ms on the following stop trial in that category (e.g. alcohol+no reward), making subsequent inhibition harder on that trial category. If participants failed to inhibit the delay decreased by 50 ms, making inhibition easier on that trial category (min delay = 50 ms, max delay = 1000 ms). The task had a total of 648 critical trials, split into nine blocks of 72, and a practice block of 16 trials in which data was not recorded. The primary measures were Stop Signal Reaction Times (SSRTs), proportion of stop errors and Go Reaction times.

Procedure

Participants attended the University and were randomised using block randomisation to either a neutral testing laboratory or a semi-naturalistic bar laboratory. Experiment sessions took place between 12pm and 6pm, in either the bar lab or human psychopharmacology labs, at the University of Liverpool. Participants completed a two week Timeline Follow-Back (TLFB; Sobell & Sobell (1992)) to measure previous alcohol consumption; the Alcohol Use Disorders Identification Test (AUDIT;(Saunders et al., 1993)) to measure hazardous drinking; the Barratt impulsivity Scale (BIS(Patton et al., 1995)) to measure self-reported impulsivity; the Temptation and Restraint Inventory (TRI;(Collins & Lapp, 1992)) to measure drinking restraint; and a Chocolate Use Questionnaire (CUQ), to measure chocolate consumption. Participants were asked to state their preferred alcoholic beverage from beer, cider, red, white or rose wine. They then completed the mSST. Upon finishing participants were fully debriefed and reimbursed with high street vouchers (£20, all participants received the same amount, irrespective of performance). The experiment lasted approximately 60 minutes.

Data reduction and analysis

The following procedures for handling Stop Signal Task data were the same for each experiment below. To compute Go reaction times for each image-type (alcohol, chocolate, neutral) and reward-type combination (present vs absent, or magnitude) we removed any reaction times < 200 ms. We then took the median go reaction time for each image-type and reward-type combination to reduce the influence of any outliers. We also calculated the proportion of successful inhibitions for each image-type and reward-type combination. We computed SSRTs separately for each image-type and reward-type combination using the integration method (Verbruggen, Chambers, & Logan, 2013; Verbruggen & Gordon D Logan, 2009a), with replacement of incorrect/omitted go errors with the maximum reaction time from the distribution of correct reaction times on that trial type (Verbruggen, Aron, Band, & al., 2019). The integration method involves taking the *Nth* reaction time from a ranked (fastest to slowest) distribution and subtracting the mean stop signal delay. *N* is determined based on the multiplying the number of reaction times in the distribution by the probability of inhibition errors. For example, with 72 reaction times in the distribution and probability of inhibition failure at 25% the Nth reaction time would be 72 * .25 = 18(th reaction time in the distribution). We removed any SSRTs which were negative from analyses, as these likely reflect a violation of the task rules (Congdon et al., 2012), and also any SSRTs in which the participant made <10% or >90 inhibition errors.

Results

For each dependent variable we conducted a three-way mixed ANOVA, with a between subjects factor of environment (2: semi-naturalistic, standard), within subjects factor of cue (3: alcohol, chocolate, neutral) and reward (2: reward, no reward).

Reaction times: Three participants were removed for an outlying number of Go errors (< 174). There was a significant cue x reward x environment interaction ($F(2, 184) = 3.26$, $p = .041$, $\eta_p^2 = .034$), subsumed under this there was a main effect of cue ($F(2, 184) = 17.20$, $p < .001$, $\eta_p^2 = .157$) and a significant main effect of condition ($F(1, 92) = 12.92$, $p < .001$, $\eta_p^2 = .123$). There were no other main effects or interactions (Fs < 3.30 *ps* > .073). The main effect of cue demonstrated that reaction times were significantly faster to alcohol-related cues (879.04, SE = 15.23) compared to chocolate (904.67, SE = 14.27; $p < .001$) and neutral cues (892.90, SE = 13.87; $p = .003$). Reaction times to chocolate cues were significantly slower than neutral cues ($p < .001$). The main effect of condition demonstrated that reaction times were significantly faster in the bar lab (841.01, SE = 19.93) compared to the neutral lab (943.39, SE = 20.38; $p = .001$). To examine the cue x reward x environment interaction, we ran 3 (cue) x 2 (reward) ANOVAs separately by environment. In the bar lab there were significant main effects of cue ($F(2, 94) = 9.51$, $p < .001$, *np2* = .168) and reward ($F(1, 47) = 5.70$, $p = .021$, *np2* = .108) but no cue x reward interaction ($F(2, 94) = 2.24$, $p = .112$, *np2* = .045). The main effect of cue demonstrated reaction times were significantly faster to alcohol-related cues (826.19, SE = 23.66) compared to chocolate (852.98, SE = 23.13; $p < .001$) and neutral cues (843.85, SE = 21.62; $p = .011$). There was no difference between chocolate and neutral cues ($p = .103$). The main effect of reward demonstrated that reaction times were faster on rewarded (836.00, SE =22.88) compared to non-rewarded trials (846.01, SE = 22.38; $p = .021$). In the neutral lab there was a significant main effect of cue ($F(2, 90) = 8.08$, $p = .001$, *np2* = .152) but no main effect of reward ($F(1, 45) = 0.23$, $p = .635$, *np2* = .005) or cue x reward interaction ($F(2, 90) = 1.59$, $p = .210$, *np2* = .034). The main effect of cue demonstrated reaction times were significantly faster to alcohol-related cues (931.89, SE = 18.93) compared to chocolate cues (956.35, SE = 16.35; $p < .001$) but not neutral cues

(941.95, SE = 17.15; p = .113). Reaction times were significantly faster to chocolate cues compared to neutral cues (p = .012).

SSRTs: No SSRTs were removed based on the exclusion criteria. There were no significant main effects or interactions (Fs < 2.31, *ps* > .123). There was weak statistical evidence for a difference in condition (F(1, 92) = 3.54, p = .063, η_p^2 = .037) with SSRTs longer in the bar (247.93, SE = 7.22) compared to neutral lab (228.51. SE = 7.37).

Exploratory analysis

Trigger failures: The effect of reward on trigger failures was examined based on a recent paper by Doekemeijer, Verbruggen, and Boehler (2021), suggesting they are more sensitive to reward. There was no significant main effect or interaction of reward or any other condition (Fs<3.88, *ps*>.05).

Table 1 Mean and SD for Go RTs, Go Errors, Stop Errors, and SSRTs in Study 1

Environment	Semi-naturalistic						Standard					
Cue type	Alcohol		Chocolate		Neutral		Alcohol		Chocolate		Neutral	
Reward type	Reward	No reward	Reward	No reward	Reward	No reward	Reward	No reward	Reward	No reward	Reward	No reward
Go RTs	842.14 (141.43	847.48 (131.79)	863.05 (127.45)	871.93 (125.20)	855.97 (125.52)	863.81 (126.250)	880.68 (130.03)	881.65 (126.67)	911.61 (130.90)	901.99 (128.31)	893.20 (126.59)	903.66 (115.80)
Go Errors	3.89 (3.37)	2.55 (4.57)	4.57 (4.02)	5.22 (5.76)	5.55 (4.96)	5.86 (5.54)	5.65 (4.94)	4.23 (5.94)	7.52 (5.10)	8.56 (6.85)	8.94 (5.84)	8.27 (6.08)
Stop Errors	23.39 (3.11)	22.67 (3.13)	22.71 (3.98)	22.53 (3.71)	22.74 (3.25)	22.55 (3.44)	20.81 (2.25)	20.65 (2.34)	20.23 (2.45)	20.08 (2.58)	20.46 (2.41)	20.40 (2.25)
SSRTs	234.13 (59.24)	202.45 (84.71)	222.54 (87.30)	220.46 (78.18)	214.36 (81.64)	225.11 (83.28)	190.61 (74.82)	183.54 (72.72)	188.85 (73.58)	174.38 (87.58)	183.90 (68.61)	183.90 (68.61)

Experiment two

In experiment two, we manipulated the probability of reward magnitude (red '+' = 0% , blue '+' = 50%, green '+' = 100%) and the type of visual cue presented during the task (alcohol, chocolate, neutral). The probability of reward was not explicit and required participants to learn on a block-by-block basis. Previous research suggests that as likelihood of receiving a reward increases, so too does the facilitation of inhibitory control (Herrera et al., 2017; Herrera, Speranza, Hampshire, & Bekinschtein, 2014). We hypothesised (i) SSRTs will be shorter in the 100% reward condition compared to 50% and 0% conditions. SSRTs will be longer for (ii) alcohol cues compared to other cues. (iii) SSRTs will be longer in the semi-naturalistic environment compared to an un-naturalistic. (iv) There will be an interaction between cue type and environment, specifically in the semi-naturalistic environment for alcohol cues SSRTs will be significantly longer. (v) Environment and reward will interact, specifically in a semi-naturalistic environment the prospect of a high reward magnitude can facilitate IC performance. (vi) Inhibitory control performance, both proactive and reactive, will predict alcohol consumption on a bogus taste test. Experiment 2 was pre-registered on the Open Science Framework (https://osf.io/wg6d3).

Method

Participants

Sixty-four participants (46 female; mean age 22.38 (±8.01) years old) were recruited (demographic information is in table 1). This sample size was calculated to be a sufficient number of participants based on a medium effect size from experiment 1 F=0.15 with 90% power at an alpha =0.05.

Stop Signal Task

The task was similar to experiment 1. Reward probability (0%, 50%, 100%) was manipulated on a block-by-block basis using a coloured fixation cross (described above) for 250 ms. Following this an alcohol, chocolate or neutral image was presented either 5 degrees clockwise or counter-clockwise and participants had to respond to the orientation as quickly as possible, using the same keys as previous experiments. There were 75 'go trials' for each image and reward-type (alcohol+0%, alcohol+50%, alcohol+100%, chocolate+0%, chocolate+50%, chocolate+100%, neutral+0%, neutral+50%, neutral+100%). On 25% of trials a stop signal occurred for each image and reward combination.

Procedure

Participants attended the University and provided informed consent. Participants completed the experiment in both the bar and semi-naturalistic laboratory, with a week between testing sessions. Each session lasted for 90 minutes.

Participants were asked to pick their drink of choice from beer, cider, white or rose wine. They then completed the TLFB, AUDIT, BIS and CUQ. The researcher explained the mSST to the participants and were shown the vouchers they could win based on their performance in the task, as part of the cover story. During the mSST participants would sniff their drink of choice and bring the liquid up to their lips, after which they would complete the DAQ. A baseline measure of craving was taken before the presentation of alcohol at the start of the session. Upon completion of the first session participants completed a filtered debrief, and were fully debriefed at the end.

Results

Changes in SSRT were analysed using a 3 (cue: alcohol, chocolate, neutral) x 2 (environment: semi-naturalistic, standard) x 3(reward: 100%, 50%, none) repeated measures ANOVA, with Bonferroni corrected pairwise comparisons.

Reaction Times: Nine participants were identified as outliers for Go Errors (>85 errors) and removed from further analysis. There was a significant main effect of reward ($F(2, 126) = 6.932$, $p=.001$, $\eta_p^2=.114$), but no main effect of environment ($F(1, 63) = .227$, $p=.636$, $\eta_p^2=.004$) or cue type ($F(2, 126) = 1.237$, $p=.294$, $\eta_p^2=.022$). The main effect of reward demonstrated reaction times were faster in no reward trials (716.34, SE=18.32) compared to 50% (744.95, SE=19.20; $p<.001$) or 100% (801.89, SE=31.75, $p=.004$) reward trials. Reaction times were significantly quicker in the 50% reward trials compared to the 100% trails ($p=.05$).

Stop Signal Reaction Time: Two participants were identified as extreme inhibitory failure (>190 stop errors in total) and were removed from subsequent analysis. There was a significant main effect of cue type ($F(2, 126) = 3.10$, $p<.05$, $\eta_p^2=.06$) but no main effect of environment ($F(1, 51) = 2.35$, $p=.131$, $\eta_p^2=.04$) or reward ($F(2, 126) = 2.11$, $p=.127$, $\eta_p^2=.0.04$). SSRTs were significantly longer for chocolate cues (268.76, SE=6.85) compared to neutral cues (260.48, SE=6.26, $p=.01$).

Exploratory analysis

Trigger failures: There was a significant main effect of reward on trigger rate failures ($F2,126)=7.18$, $p<.001$, $\eta_p^2=.09$). Trigger rates were significantly higher in the no reward

condition compared to the 100% reward condition (1.35, SE=.38, $p<.01$) and 50% reward condition (1.13, SE=.38, $p<.05$).

Stop Signal Reaction Time's predictive ability of alcohol consumption

In a regression model bogus taste test alcohol consumption was added as the dependent variable and SSRTs across all variables (e.g. cue type and reward type) were predictor variables. SSRTs did not predict a significant amount of variance in bogus taste test data at timepoint one ($F(9,63)=.198$, $p=.993$) or timepoint two ($F(9,62)=1.459$, $p=.188$). For both timepoint one and timepoint two there was a high degree of multicollinearity (VIF>1.52).

Environment	Semi-naturalistic									Standard						
Cue type	Alcohol			Chocolate			Neutral			Alcohol			Chocolate			
Reward type	100% reward	50% reward	No reward	100% reward	50% reward	No reward	100% reward	50% reward	No reward	100% reward	50% reward	No reward	100% reward	50% reward	No reward	100% reward
Go RTs	779.09 (147.28)	735.35 (153.47)	716.76 (150.70)	803.08 (155.12)	753.25 (162.21)	736.89 (160.75)	791.23 (157.05)	754.44 (163.58)	733.94 (160.35)	761.84 (145.31)	750.70 (153.96)	703.83 (152.12)	786.14 (159.11)	758.80 (154.55)	717.50 (157.47)	919.11 (1163.20)
Go Errors	4.03 (4.94)	3.33 (4.04)	2.94 (3.56)	5.72 (8.17)	5.17 (7.36)	4.64 (5.67)	5.30 (6.70)	4.20 (6.37)	4.48 (5.27)	3.84 (6.15)	2.41 (4.05)	2.38 (3.31)	4.47 (6.61)	3.03 (4.66)	3.58 (4.31)	4.67 (7.26)
Stop Errors	10.37 (1.75)	11.98 (4.41)	11.86 (4.07)	10.19 (1.84)	11/67 (4.28)	11.79 (4.08)	10.24 (2.21)	11.84 (4.32)	11.77 (3.80)	10.24 (2.17)	10.33 (2.06)	11.29 (2.87)	9.81 (2.29)	10.38 (2.22)	11.05 (2.84)	10.24 (2.21)
SSRTs	258.71 (67.26)	294.36 (140.32)	282.05 (106.54)	259.30 (55.25)	305.81 (143.16)	280.89 (107.89)	250.28 (70.03)	297.43 (135.94)	280.34 (101.56)	275.53 (128.40)	266.09 (98.39)	267.303 (105.62)	261.90 (138.92)	281.47 (120.72)	292.12 (141.05)	254.33 (113.14)

Table 2 Mean and SD for Go RTs, Go Errors, Stop Errors, and SSRTs in Study 2

Discussion

In two experiments we examined the role of appetitive cues and motivation on inhibitory control. We observed no consistent effect of motivation or exposure to appetitive cues on reactive control, nor an interaction between the two. I found limited evidence of inhibitory improvements in the presence of reward specifically to reactive control.

Our first hypothesis across both experiments that appetitive cues would impair inhibitory control was not consistently supported. The main effect of cue-type was evident in study two, which demonstrated chocolate cues impaired proactive inhibitory control compared to alcohol and neutral cues. While experiment one demonstrated that alcohol cues significantly facilitated reactive control compared to chocolate and neutral cues. Both experiments used cues which were personalised to the individuals drinking preference, which should have increased the magnitude of cue-specific inhibitory control deficits (Fatseas et al., 2015). Taken together, the findings from the two experiments are in direct contrast to theoretical models which suggest inhibitory control is transient, and is impaired by appetitive cues which provoke approach behaviours (Jones et al., 2018). They also contrast empirical evidence presented in a recent meta-analysis demonstrating this effect (Burton et al., 2021). However, as discussed it is possible that this pooled-effect is overestimated (at least for alcohol-related cues) due to biases (small study / publication) in the literature.

Similarly, we were unable to demonstrate consistent effects of motivation on response inhibition. Across both experiments it was hypothesised that the prospect of a reward would improve reactive inhibitory control, yet irrespective of reward or magnitude of reward in comparison to no reward heavy drinkers did not exhibit an improvement in inhibitory control. Findings contradict previous research in which reactive inhibitory control has been improved

in the presence of reward related stimuli (Boehler et al., 2014; Chung et al., 2011; Herrera et al., 2017; Burton et al., 2021), however there are a number of contradictory findings (Herrera et al., 2014; Verbruggen & McLaren, 2018). Finally, we did not demonstrate any convincing evidence for the interactive effects of motivation and cue-exposure which is perhaps unsurprising given the lack of main effects. This, along with the lack of main effects suggests attempts to improve inhibitory control to appetitive cues using motivational mechanisms (e.g. rewards) are unlikely to be effective.

As the trial-by-trial reward-related information was present at the beginning of each trial (rather than when inhibition is prompted, see Langford, Schevernels, and Boehler (2016) we also examined whether the information leads to a slowing of reaction times which might be indicative of strategic, proactive slowing (see Baines, Field, Christiansen, and Jones (2019b)). Motivation did not consistently affect proactive slowing, only in presence of an increased likelihood of reward (0% chance, compared to 50% and 100%: experiment 2). These findings suggest the reward-related information must be salient or be presented alongside more nuanced comparators (e.g. not simply present vs absent) to influence behaviour (Stoppel et al., 2011). Such results are interesting given that in the debrief the majority of participants did not believe they would receive a reward or payment, and highlights a disconnect between motivation – behaviour, which is in contrast with theoretical models of behaviour such as the Theory of Planned Behaviour and self-determination theory (Hagger & Chatzisarantis, 2009). Nevertheless, the slowing of reaction times with increased motivation is indicative of proactively slowing. It is possible that participants were engaging in proactive inhibitory control mechanisms, and were not relying on the 'late correction' mechanisms of reactive control (Braver, 2012; Braver et al., 2009) which may explain why we did not observe any differences in reactive control. Previous findings suggest that

performance-contingent rewards increase the efficiency of proactive control (Chung & Barch, 2015; Strang & Pollak, 2014). This supports previous findings by Schevernels et al., (2014; 2015). However, as these analyses were exploratory (not-preregistered) our interpretation remains cautious, until replicated.

It is possible that we demonstrated limited evidence for motivational impairments in inhibitory control as heavy drinking adults have a blunted response toward non-substance related cues (Beck et al., 2009; Schacht, Anton, & Myrick, 2013). Hyposensitivity towards reward-related stimuli has been linked to reduced dopamine levels and lack of motivation inhibit when the prospect of reward is available (Byrne & Worthy, 2019). Future research could examine differences in drinking status to clarify this.

These findings should be interpreted in light of the limitations of the studies. Stop signal paradigms require multiple tasks (e.g. reward cue tracking, stop signal tracking, stimulus orientation) with task switching literature suggesting mixing tasks can reduce performance (Karbach & Kray, 2009; Zinke, Einert, Pfennig, & Kliegel, 2012) potentially increasing demands on working memory as complexity increases (Simmonds et al., 2008). Study one used a 50% inhibition rate, potentially inflating SSRTs as a result and potentially priming an inhibition response. Across the studies no consistent measure of the salience or belief of receiving the rewards was administered, as such researchers had no way to know if the participants believed they would receive a reward or if participants perceived the reward presented in the task as meaningful to the individual.

In conclusion, findings tentatively support the notion that inhibitory control is a state rather than a trait that fluctuates in response to internal and external factors. Despite the use

of personalised alcohol stimuli to increase reliability (Christiansen et al., 2015a), no consistent effect of appetitive cues was observed. Future research should seek to investigate the role of motivation and cue reactivity on inhibitory control, using adequately powered studies and publication of both significant and null findings to reduce the publication bias in the current literature.

Chapter Summary

This chapter contributed to the overall aims of the thesis by providing weak evidence that inhibitory control is a state variable, fluctuating in response to reward and appetitive cues. However, effects were not consistent across the two studies for reward or appetitive stimuli. Findings that showed an effect of reward on inhibitory control were not pre-registered so require further examination and replication by future research. I found little evidence for appetitive cues inducing a state of disinhibition, even in an environment rich with alcohol stimuli; this was further explored using more naturalistic techniques in the next chapter.

Chapter 5

An ecological momentary assessment study of fluctuations in inhibitory control and its predictive validity of alcohol use

This chapter presents an experimental study which has been prepared for publication. This study was pre-registered on Open Science Framework (link is provided in text). To summarise contributions to this chapter, I designed the study which was approved by Andrew Jones. I collected and analysed the data. I wrote the manuscript and Andrew Jones provided feedback on this.

The aim of this study was to examine the fluctuations in inhibitory control in a naturalistic environment, and if said fluctuations were associated with alcohol use. I also aimed to examine if baseline measures of inhibitory control and alcohol consumed on a bogus taste test (in a controlled environment) were associated with real world results. I also sought to examine if inhibitory control fluctuated across environments, to assess whether it is a transient state like variable.

Abstract

Background: To examine fluctuations in inhibitory control and its predictive validity of alcohol consumption in a naturalistic setting.

Objectives: In the current study we investigated inhibitory control using an ecological momentary assessment paradigm to investigate the relationship with alcohol consumption and other factors (e.g. location, craving, emotions) in the real-world. We hypothesised that fluctuations in inhibitory control throughout the day would predict alcohol consumption.

Methods: Heavy drinkers (N=54) were asked to complete a battery of questions and a stop signal task four times per day, at random intervals between 10am and 10pm for one week. Participants were asked to record their location, craving, emotions and alcohol consumption at each assessment. Inhibitory control was assessed using stop signal task with personalised alcohol- and generic neutral-related cues.

Results: Multilevel modelling demonstrated that neutral SSRTs (*B*= .004; 95% CI .002, .006) and frequency of craving (*B*=.049; 95% CI .039, .059) predicted subsequent alcohol use occasions. Intensity (*B*=-.036; 95% CI -.059, -.013) and frequency (*B*=.026; 95% CI .002, .050) of craving significantly predicted variance in alcohol consumption.

Conclusions: Findings do not provide consistent evidence that fluctuations alone in inhibitory control predict alcohol consumption. Future research should examine the interaction between inhibitory control and craving in the real-world, to better our understanding of the complex relationship.

Introduction

Inhibitory control, otherwise known as response inhibition, is the (in)ability to stop, change or delay inappropriate behaviour under certain circumstances (Logan, Cowan, & Davis, 1984b). For example, applying the breaks when you see a red traffic signal. Inhibitory control is an underlying component of both impulsivity and executive functioning (Bickel et al., 2012), while also being encompassed under the broader construct of self-control (Fujita, 2011). The ability to inhibit motoric behavioural responses has been operationalised in controlled environments using experimental tasks such as the stop signal task (Verbruggen & Logan, 2008). In these tasks participants are required to execute a motor response on the majority of trials without interruption (e.g. 75%), reinforcing a dominant response. On a minority of trials they are required to withhold the reinforced response following a 'stop signal'.

Deficits in inhibitory control are observed in individuals suffering from alcohol dependence and non-dependent individuals who drink 'heavily' (Christiansen et al., 2012; Houston et al., 2014; Smith et al., 2014). Deficits are associated with *ad-libitum* alcohol consumption in the laboratory (Jones, Field, Christiansen, & Stancak, 2013). However, it is not clear whether such deficits are a cause or consequence of substance misuse (De Wit, 2009; López-Caneda et al., 2013; Verdejo-García et al., 2008). Longitudinally, inhibitory control can predict relapse (Rupp et al., 2016) and the transition from heavy drinking to dependence (Rubio, Jiménez, Rodríguez-Jiménez, et al., 2008), along with the initiation and escalation of alcohol use in adolescents (Fernie et al., 2013; Nigg et al., 2006).

Much of the cross-sectional research into inhibitory control implies that it isa stable trait within individuals, however more recent theoretical models hypothesise that the ability to inhibit behaviour can fluctuate within individuals over time (Jones et al., 2013), which makes it more difficult for individuals to engage their inhibitory control in response to temptation. Jones et al. (2013) reviewed the evidence and observed that transient changes in inhibitory control were evident in response to; environmental influences (De Wit, 2009; Jones et al., 2013), stress (Roos et al., 2017), reward/ extrinsic motivation (Burton et al., 2021) and exposure to alcohol-related cues and contexts which are thought to increase craving (Czapla et al., 2015; Jones & Field, 2015). Furthermore, laboratory-based studies have demonstrated that fluctuations in inhibitory control, a result of experimental manipulations, may influence subsequent alcohol consumption suggesting a causal relationship (Field & Jones, 2017; Jones, Cole, Goudie, & Field, 2011; Jones, Guerrieri, et al., 2011).

The evidence base to-date is mostly from laboratory-based studies. However, these studies are limited by retrospective recall, demand characteristics and a suppression of craving / consumption behaviours (Jenkins, McAlaney, & McCambridge, 2009; Monk et al., 2015). Substance use is contextually driven and time sensitive and in order to examine the link between inhibitory control and alcohol use, assessments must be made repeatedly in congruent contexts and at strategically selected moments (Lau-Barraco & Linden, 2014). As such, Ecological Momentary Assessment (EMA) methods are well enabled to examine the precursors of substance use behaviours in real-world environments (Shiffman, 2009; Shiffman, Stone, & Hufford, 2008). EMA is the repeated sampling of participants' subjective states and behaviour in naturalistic settings. EMA studies allow for the examination of temporal relationship between substance related cues, fluctuations in craving, self-control and

substance use (Fatseas et al., 2015; Remmerswaal, Jongerling, Jansen, Eielts, & Franken, 2019; Serre et al., 2015). EMA allows for daily assessments of alcohol consumption providing more reliable estimates than retrospective diary measures (Monk et al., 2015). Such methods have been used to investigate cognitive precursors, such as attentional bias and inhibitory control, in substance use (Jones, Tiplady, Houben, Nederkoorn, & Field, 2018; Reshmi Marhe, Waters, van de Wetering, & Franken, 2013; Waters, Marhe, & Franken, 2012).

To date only one study has investigated the relationship between day-to-day fluctuations in inhibitory control and whether it can predict alcohol consumption in heavy drinkers (Jones et al., 2018). Jones et al. measured inhibitory control using a stop signal task twice per day, between 10am and 6pm. Their findings demonstrated that average daily inhibitory control did not predict daily alcohol use, however fluctuations over the course of the day did, suggesting fluctuations may be a risk factor for heavy drinking. However, these findings were exploratory, and focused on only two sessions administered per day, potentially not capturing the dynamic nature of inhibitory control.

The present study aims to extend the findings of Jones et al. (2018) by administering four daily assessments compared to two to allow us to investigate dynamic changes in inhibitory control, mood and craving in relation to alcohol consumption, while examining within day fluctuations. Further to this, baseline sessions of the Stop Signal Task (SST) were used to cross-validate findings from the mobile SST given to participants. Baseline taste-test results were compared to actual drinking behaviour in EMA sessions to examine the predictive reliability of the taste test as an analogous measure of drinking behaviour. Use of a

baseline session will allow EMA sessions to be cross-validated against a baseline session in a controlled environment, allowing us to examine the reliability of EMA session results.

We hypothesised that i) fluctuations in inhibitory control would predict subsequent alcohol consumption, specifically decreased in inhibitory control (reduced SSRTs) will lead to increased alcohol consumption, ii) fuctuations in inhibitory control over the course of the day will influence the predictive ability for later alcohol consumption. iii) Alcohol consumed on an ad-libitum taste test will predict alcohol consumption in real world environment. iv) Inhibitory control performance will fluctuate as a result of location of the testing location, e.g. if participants are in an environment with alcohol-cues present inhibitory control will decrease. This study was pre-registered on the Open Science Framework (https://osf.io/gc94x/).

Method

Participants

We recruited 57 heavy drinking individuals into the study. Three participants withdrew from the study due as they were unable to commit to the testing schedule, leaving 54 (47 females, mean 24.30 ±7.67 years old) in the final sample. Participants were recruited from the local community through the use of adverts both physically and via social media. Heavy drinking was defined as regularly drinking in excess of UK government guidelines, which is <14 UK units for both men and women (NHS, 2016). To be eligible, participants had to be 18+ and own an iPhone (due to the experiment software only compatible with iOS operating systems). Participants were excluded if they self-reported a current or previous diagnosis of a substance use, psychiatric or neurological disorder. Our a-priori sample size estimation was 55 participants. This was based on simulation research by Maas and Hox

(2005) suggesting that sample size >50 participants (as level 2 units) lead to unbiased standard errors in multilevel models. We aimed for 55 to account for 10% attrition. Upon completion of the full study participants were reimbursed with £20.The study protocol was approved by the local research ethics committee (approval number: 3854).

Baseline Measures

Timeline follow-back

Participants completed a two week retrospective diary of all alcoholic beverages they consumed via the alcohol Timeline Follow Back (TLFB). The TLFB is regularly used to assess frequency and quantity of alcohol consumption. Participants were asked to record the number of units they consumed on a day-to-day basis for the previous 14 days. A unit's guide was provided for standard measurements of a variety of drinks, e.g. a small glass of wine or bottle of beer. Total units consumed during the previous 14 days and binge drinking frequency were the outcome measures.

Alcohol Use Disorders Identification Task

The Alcohol Use Disorders Identification Task (AUDIT) was used to assess hazardous drinking. The AUDIT is a ten-item scale, with each item given a score from 0-4, with a maximum score of 40. The internal consistency of the AUDIT in this sample was α=.75.

Barratt impulsivity scale (BIS)

The BIS measures self-reported trait impulsivity. The scale is comprised of 30 questions, scored from 1-4 ‘rarely’, ‘occasionally’, ‘often’ and ‘always’. The total score is made by summing the three subscales; Attention, Non-Planning and Motor Impulsiveness. The internal consistency of the BIS total score was α=.57, Attention subscale α=.44, Non-Planning subscale α=.43, Motor Impulsiveness subscale α=.54.

Temptation and Restraint Inventory (TRI)

The TRI measures drinking restraint, using a 15-item scale, loading onto five subscales; Govern (difficulty controlling alcohol consumption), Restrict (attempts to limit drinking), Emotion (negative affect as a reason to drink), Cognitive Emotion Preoccupation (CEP; thoughts about drinking) and Cognitive Behavioural Control (CBC; plans to reduce drinking/ worry about controlling drinking). Items are rated on a 9-point Likert scale from 1 ‘never’ to 9 ‘always’. The internal consistency for the overall scale was α=.78, CEP subscale α=.74, CBC subscale α=.73.

Brief Self-Control Scale (SCS)

The self-control scale assesses an individual’s general trait level self-control. The scale is scored using a 5-point Likert scale, from 1 ‘not at all’ to 5 ‘very much’, on 13 items,

loading onto four factors; self-discipline, healthy habits, impulsivity and self-regulation. The internal consistency for the overall scale was α=.61.

Brief Desire for Alcohol Questionnaire (DAQ)

The DAQ allows the assessment of moment-to-moment craving for alcohol. The abbreviated DAQ is scored on a 7-point Likert scale, from 1'strongly disagree' to 7'strongly agree'. It is based upon three subscales that assess, intention to drink, negative reinforcement and positive reinforcement, and ability to control drinking (Kramer et al., 2010).

Ad-libitum alcohol taste test

In the ad-libitum taste test participants are given access to a set amount of alcohol and asked to rate it on multiple perceptual factors (Field & Eastwood, 2005; Jones et al., 2011), providing an unobtrusive measure of alcohol consumption (the rating scales are of secondary importance). In this study participants were presented with two alcoholic beverages and one soft drink. Participants were presented with 3 units of alcohol, exact measure in ml varied across drink choice due to strength differences. Participants were also given a soft drink (cola) that was the same amount in ml as the alcoholic drinks. The dependent variable was the percentage of alcohol consumed. Additionally, a 100mm Visual Analogue Scale (VAS) was administered to examine participant's thirst prior to the taste test ('*How thirsty are you right now on a scale of 0 (not thirsty at all) – 100 (extremely thirsty)*)'.

EMA Measures

Stop signal task

The stop signal task (Verbruggen & Logan, 2008) was programmed in Inquisit 5, based on Jones et al. (2018). The screen background was white with a black fixation cross. On each trial, following the presentation of the fixation cross for 250ms an alcohol- or neutral- stimulus was presented in the centre of the screen, rotated 45 degrees to the right or left. On go trials, participants had to respond to the orientation of the image by pressing a left button on the touch screen if the image was rotated to the left and a right button if the image was rotated to the right. The categorisation of this response was uninterrupted on 75% of trials, and these are referred to as 'go trials'. On the remaining 25% of trials a stop signal (a red '=' sign) was superimposed over the go stimuli, after a variable delay (stop signal delay) after the onset of go stimuli. Participants were instructed to withhold their categorisation response on trials a stop signal was presented.

A dynamic tracking algorithm was used to set stop signal delays (Verbruggen & Logan, 2008). In a given session, the first delay was set at 250ms following onset of the go stimulus. If participants were able to successfully inhibit, the delay increased by 50ms on the subsequent stop trial and decreased on unsuccessful inhibition (min delay= 50ms, max delay=1000ms). Participants were presented with a total of 192 trials, with 48 stop trials and 144 go trials.

Stimuli

Eight personalised alcohol-related pictures for four different drink categories (beer, white wine, rose wine and cider), and eight neutral images (e.g., plug socket, shells, books) were used in the Stop Signal task, across all conditions. All images were presented in the same size and brightness, in an attempt to match the perceptual characteristics. Images were rotated 45 degrees to the right or left, as part of the classification component of the SST (see Jones & Field, 2015). Alcohol stimuli was personalised for the participant's preferred drink (e.g. if the individual preferred cider to beer, they were only shown cider-related images) (Christiansen & Bloor, 2014) to increase the strength of the manipulation and internal reliability (Christiansen et al., 2015).

EMA self-report measures

At the beginning of each assessment participants were asked "*How [energetic/sad/drowsy/happy] do you feel right now?"* with similar questions for craving "*How strong is your craving for alcohol right now?"* and were asked to respond on a 0 – 100 visual analogue scale (0 = not at all, 100 = extremely). Smoking behaviour was assessed, "*Have you had a cigarette since your last assessment?"* since their last assessment, via a 'yes' or 'no' answer. Following completion of the stop signal task, participants were asked to report their location for the assessment into one of six categories ('work', 'home', 'traveling', 'bar', 'restaurant' or 'other'). To control for distractors, participants were asked to record

whether they completed the session 'alone' or 'in the presence of others', and if they were interrupted and if so, how many times. Finally, participants were asked if they had consumed alcohol *"How much alcohol have you consumed since your last assessment?",* in which the number of units consumed was reported, and to provide a breathalyser reading using a portable breathalyser supplied to them by the researcher.

Procedure

Participants were pre-screened via an online questionnaire. Eligible participants were invited to take part in the baseline session in the Human Psychopharmacology Laboratories at the University of Liverpool. Upon arrival they provided informed consent. They then completed the battery of questionnaires for the baseline session (AUDIT, BIS, TRI, SCS, DAQ). Following completion, the EMA app was loaded onto the participant's phone. Alcohol images were personalised to the individual. Participants then completed their first full session using the app and were asked if they had any questions. Finally, they completed the ad-libitum alcohol consumption measure. Before leaving the laboratory, participants were given a printed guide of units, instructions and contact details for the researcher should they incur any problems. They were also provided with a portable breathalyser in an attempt to biochemically verify self-reported consumption.

During the EMA phase participants were randomly prompted (Random Assessment: RA) four times per day, between the hours of 10am and 10pm, in 3-hour time windows with a final breathalyser session at 10pm. Notifications were sent via email to participants to complete a session at the next available opportunity, completing all self-report measures from the EMA session and the stop-signal task. Participants took part in the study for 7 full days,

beginning the day after the baseline session. Upon completion they were asked to return the breathalyser and attend a debrief session. Any data that had been stored on the participants phone from EMA sessions via the app was uploaded to the database.

Data reduction and analysis

To compute Stop Signal Reaction Times (SSRT), SPSS 25 was used, and for subsequent analysis R was used with the 'Dplyr' and 'Lme4' packages. We computed SSRTs separately for each image-type using the integration method (Verbruggen et al., 2013; Frederick Verbruggen & Gordon D Logan, 2009a), with replacement of incorrect/omitted go errors with the maximum reaction time from the distribution of correct reaction times on that trial type (Verbruggen, Aron, Band, et al., 2019).

Multilevel modelling is the most appropriate method for analysis of repeated measures, due to the nature of nested data it takes into account the dependence between observations as a result of data clustering (e.g. stop signal performance may fluctuate over time, but should be highly correlated with other time points). Use of multilevel modelling allows for unequal number of data points across participants (resulting from missing data) (Hayes, 2006; Quené & Van den Bergh, 2004). A mean centred approach (Paccagnella, 2006) was adopted to assess an individual's fluctuations in SSRTs in respect to their own mean SSRT, rather than that of the groups, within the models. Improved model fit was assessed via reductions in AIC/BIC values (Burnham & Anderson, 2004) for binary outcomes (with reduction in AIC/BIC > 10 indicative of a better fitting model), and reductions in Log-likihood statistics (Leckie, 2019) for continuous outcomes.

In a deviation from the pre-registered outcome, we did not use alcohol consumption in units as our primary outcome, but rather recoded the variable to a drinking occasion (vs no drinking occasion). This was due to the large proportion of sessions in which alcohol consumption was reported as zero (80.15%), skewing the distribution of quantity of alcohol units consumed. Location was initially coded as; home, work, travel, bar or restaurant, or other. Eight-hundred and sixteen of the data points were classified as being at home, with 484 being split across the remaining categories. For the purpose of the analysis, location was coded as either at home or not, due to some locations not having adequate data points for analysis. Similarly with breathalyser readings, due to the amount of 0 readings, data was classified as either a positive reading or not.

Results

Participant characteristics

Participants (Table 3) had a mean age of 24.30 (SD = 7.67). On average participants consumed 42.83 units of alcohol in a two-week period prior to the baseline session, exceeding the 'heavy drinking' threshold (~28 units over the two-week period), with an average of 3+ binge sessions over the same period.

Compliance

Participants completed 1298 Random Assessments (RA; of a possible 1512: 85.85%) and 326 Breathalyser Assessments (BA; of a possible 378). Participants reported being interrupted during 401 RAs (30.89%) and completed 610 in the presence of others (47.00%). On 149 RAs participants completed the session while reporting a positive breathalyser reading (11.03%). RAs were coded as confounded if there was a report of interruption or a

positive breathalyser reading, sensitivity analysis was carried out for the main analysis reported below, in which confounded RAs were removed. 11.04% (149) sessions were removed due to a positive alcohol breathalyser reading and 19.85% (268) for reporting alcohol consumption prior to the session. Further to sessions confounded by alcohol, those with interruptions and in which the participant had smoked were removed, as part of a sensitivity analysis. Results did not significantly differ when confounded sessions were removed and the main analyses re-run.

Fluctuations in inhibitory control, both proactive and reactive, to alcohol stimuli & environment.

Exploratory analyses examined if inhibitory control, reactive control (SSRTs) and proactive control (proactive slowing on Go RTs) fluctuated in response to alcohol stimuli. A 2 (cue type: alcohol, neutral) x 2 (environment: home, not home) mixed ANOVA was used to examine fluctuations in SSRTs. There was no main effect of cue type ($F(1,1348)=3.65$, $p=.056$, $\eta_p^2=.003$). Yet there was an interaction between cue type and environment. Alcohol SSRTs did not fluctuate between home (M=291, SD=56.5) and other environments (M= 293, SD= 87.5) ($t(825.74)= .392$, $p=.795$). Neutral SSRTs did significantly fluctuate between the home (M=294 SD= 61.3) and other environments (M=287, SD=75.2) ($t(979.37)=2.006$, $p<.05$).

The same analysis was conducted for Go RTs. There was a main effect of cue type on Go RTs ($F(1,1348)=12.25$, $p<.01$, $\eta_p^2=.009$), whereby alcohol Go RTs were significantly faster than neutral Go RTs ($t(1349)=3.28$, $p<.01$).

Multilevel model predicting alcohol consumption

The dependent variable was the total number of units of alcohol consumed on a daily basis, as inferred from the recall questions in each session. To examine the stratification of alcohol use data, a single-level model consisting of days was fitted in comparison to a two-level model of days nested within participants. For the single level binomial model fit measures were AIC=1353.61 and BIC=1364.03. For the two-level model (assessment > participant) model fit indices were AIC=1333.20 and BIC=1343.60, indicative of a better fit to the data than the single level model. A three-level model (assessment > day > participant) demonstrated minimal change in model fit AIC=1328.20 and BIC 1343.80, as such a two level model was used.

Model A (Table 4) included baseline alcohol consumption on a bogus taste test, alcohol and neutral SSRTs respectively for baseline and RA sessions, to examine if they can account for whether the participant consumed alcohol or not. Neutral SSRTs (z=3.59, p<.001) explained a significant amount of variance in the model, alcohol SSRTs (z=0.84, p=.401) and baseline alcohol consumption (z=1.44, p=.151), did not explain a significant proportion of variance. In Model B (Table 4), we added in the participant level variables of frequency of craving, location, sad, happy, energetic, and drowsy. Neutral SSRTs, frequency of craving and drowsiness significantly predicted variance in whether individuals consumed alcohol or not.

In sensitivity analysis we removed all sessions in which no alcohol consumption was reported and the outcome was amount (in units) consumed. The single level model had model fit measures of AIC=1456.41 and BIC= 1463.60. The two-level model demonstrated model fit of AIC=1453.15 and BIC 1463.92, explaining 8.08% of variance at the participant level, Model C (Table 4) used the same predictor variables as Model B, with reported alcohol

consumption as the dependent variable. Significant predictors were baseline alcohol consumption (t=2.57, $p<.05$), intensity of craving (t=-3.08, $p<.001$), frequency of craving (t=2.10, $p<.05$) and sad (t=2.32, $p<.05$). Note, that this model has considerably lower statistical power.

Exploratory analyses

Our final analysis was exploratory and examined if the aforementioned predictors from previous models could predict total daily alcohol consumption. To examine the stratification of total daily alcohol consumption, a single-level model consisting of days was fitted in comparison to a two-level model of days nested within participants. The two-level model was found to be a significantly better fit than the single-level model (χ^2 (1) =140.65, $p<.001$).

The initial model, Model D (Table 5), had the predictor variables of baseline alcohol consumption on a bogus taste test and alcohol and neutral SSRTs respectively for baseline and RA sessions to see if they can account for daily alcohol consumption. Baseline alcohol consumption accounted for a significant amount of variance in the model (t=3.84, $p<.001$), neutral (t=1.13, $p=.258$) and alcohol SSTR's (t=.83, $p=.408$) did not account for a significant amount of variance. Model E (Table 5) included the variables from Model D and intensity of craving, frequency of craving, sad, happy, energetic and drowsy. Baseline alcohol consumption accounted for a significant amount of variance in the model (t=3.85, $p<.001$)

and frequency of craving (t=5.79, p<.001) accounted for a significant amount of variance, 1.70% more than Model E.

Table 3 Participant characteristics and measurements from baseline, random and breathalyser assessments. Values are means (standard deviation)

Participant-level baseline variables	
Age (years)	24.30 (7.58)
TLFB consumption	42.83 (18.84)
TLFB binge frequency	3.39 (2.08)
AUDIT	11.94 (5.59)
BIS total	74.52 (7.05)
TRI	49.90 (18.38)
SCS	32.87 (4.10)
DAQ	29.22 (14.21)

Daily level	**Baseline**	**1st RA**	**2nd RA**	**3rd RA**	**4th RA**	**BA**
Alcohol SSRT	313.86 (62.36)	288.17 (69.35)	284.44 (65.44)	294.02 (73.68)	297.31 (73.12)	-
Neutral SSRT	307.07 (62.37)	287.80 (68.65)	285.81 (64.31)	292.25 (60.56)	297.06 (74.92)	
Craving intensity	27.41 (21.51)	17.76 (18.66)	21.99 (21.58)	29.40 (26.32)	32.97 (28.48)	-
Craving frequency	24.83 (23.40)	21.46 (20.92)	20.75 (21.43)	60.04 (25.32)	31.89 (28.21)	
Sad	24.38 (17.99)	28.70 (1.93)	26.39 (19.39)	27.33 (19.59)	24.58 (18.51)	-
Energetic	50.69 (21.89)	41.67 (23.70)	42.71 (23.57)	43.45 (22.70)	41.15 (24.71)	-
Happy	64.50 (15.23)	57.98 (19.78)	59.02 (18.63)	61.05 (18.91)	63.28 (18.77)	-
Drowsy	38.81 (26.05)	46.12 (26.24)	42.99 (26.48)	43.33 (26.27)	45.60 (26.48)	-
Units consumed	-	1.42 (3.18)	0.29 (1.48)	0.44 (1.55)	0.83 (2.34)	-
Breathalyser reading	-	0.002 (0.02)	0.002 (0.02)	0.01 (0.03)	0.03 (0.10)	0.05 (0.15)

Abbreviations- TLFB, Timeline Follow back. AUDIT, Alcohol Use Disorders Identification Test. BIS, Behavioural Impulsivity Scale. TRI CEP, Temptation and Restraint Inventory Cognitive Emotional Preoccupation, TRI CBC, Temptation and Restraint Inventory Cognitive Behavioural Control. SCS, Self-Control Scale. DAQ, Desire for Alcohol Questionnaire. SSRT, Stop Signal Reaction Time. RA, Random Assessment. BA, Breathalyser assessment.

Table 4 Multilevel model examining participant-level and daily-level predictors of alcohol consumption

Parameter	Model A Estimate (95% CI)	Model B Estimate (95% CI)	Model C Estimate (95% CI)
Outcome	Binary	Binary	Continuous
Participant Level			
Alcohol consumed on a bogus taste test	.248 (.-.102, .595)	.247(-.159, .653)	1.115 (.260, 1.948)*
Daily Level			
Neutral SSRT	.004 (.002, .007)***	.004 (.002, .006)**	.007 (-.0006, .0146)
Alcohol SSRT	.001 (-.001, .003)	.001 (-.001, .002)	.001 (-.006, .008)
Craving intensity		.002 (-.008, .012)	-.036 (-.059, -.013)**
Craving frequency		.049 (.039, .059)***	.026 (.002, .050)*
Location		.262 (-.073, .597)	-.596 (.-1.503, .322)
Sad		-.011 (-.023, 0001)	.041 (.007, .075)*
Energetic		-.0004 (-.010, .009)	-.004 (-.030, .022)
Happy		.007 (-.007, .021)	.026 (-.005, .056)
Drowsy		.013 (.005, .021)**	.003 (.-.020, .025)

SSRT, Stop Signal Reaction Time.
*$p<.05$, **$p<.01$, ***$p<.0001$*

Table 5 Multilevel model examining participant-level and daily-level predictors of daily total alcohol consumption

Parameter	Model D Estimate (95% CI)	Model E Estimate (95% CI)
Outcome	Continuous	Continuous
Participant Level		
Alcohol consumed on a bogus taste test	1.79 (.879, 2.703)***	1.795(.881, 2.709)***
Daily Level		
Neutral SSRT	.002 (-.002, .006)	.002 (-.002, .005)
Alcohol SSRT	.002 (-.002, .005)	.001 (-.003, .005)
Craving intensity	-	-.002 (-.018, .014)
Craving frequency	-	.049 (.032, .065)***
Location	-	.242 (.881, .2709)
Sad	-	.001 (-.018, .019)
Energetic	-	.001(-.013, .016)
Happy	-	.010(-.009, .029)
Drowsy	-	.006 (-.012, .014)

SSRT, Stop Signal Reaction Time.
p<.05, **p<.001,*p<.0001*

Discussion

The aim of this study was to examine if momentary fluctuations in inhibitory control could predict alcohol consumption. Hypothesis one was partially supported, as increased neutral SSRTs predicted a subsequent alcohol consumption occasion. Hypothesis two was not supported as there was no effect of within day fluctuations on alcohol use or location, contrary to our hypotheses. Hypothesis three was supported, as baseline measurements of ad-libitum alcohol consumption accounted for a significant amount of variance in subsequent decisions to consume alcohol (and amount consumed). Hypothesis four was partially supported, reactive inhibitory control performance did not fluctuate as a result of location yet there was evidence of proactive slowing when participants were at home.

Whilst the findings from this study demonstrated limited evidence for inhibitory control predicting alcohol use, this was only for SSRTS to generic neutral stimuli. These findings are broadly in line with Jones et al. (2018) who demonstrated daily fluctuations in inhibitory control were predictive of alcohol use, using arbitrary cues ('x' and 'o', on the stop signal task). However, it was surprising that there was no evidence for alcohol SSRTs given theoretical predictions and associations between alcohol-related cues, inhibitory control and alcohol consumption in the laboratory (Czapla et al., 2015; Jones et al., 2013; Weafer & Fillmore, 2015). Recent pre-registered work suggests that exposure to alcohol cues alone is not enough to create inhibitory deficits, but priming (consumption) may influence reactive components of inhibition (Baines, Field, Christiansen, & Jones, 2019a). It is possible that participants demonstrated habituation to alcohol-related cues, and as such they exerted limited effects as the testing sessions persisted (Courtney, Ghahremani, & Ray, 2015). These findings then lend some support to wider theoretical models which posit the importance of inhibitory control on subsequent alcohol consumption (Goldstein & Volkow, 2011; Kalivas & Volkow, 2005; Paz et al., 2016), but less so the importance of cue-specific inhibition (Jones et al., 2013).

Interestingly, in support of hypothesis three, ad-libitum alcohol consumption in the laboratory predicted alcohol use in the real world. Findings extend the work of Jones et al. (2018) who had no baseline measure of alcohol consumption, showing that the bogus-taste test is analogous of real-world alcohol consumption. Lab-based consumption predicted real-world consumption, such findings are promising given previous speculation over the validity of the bogus taste-test (Robinson, Hardman, Halford, & Jones, 2015; Robinson, Kersbergen, Brunstrom, & Field, 2014; Leeman et al., 2009; Leeman et al., 2013). Particularly for the

field of experimental medicine the bogus taste-test appears to be an effective proxy for alcohol consumption, enabling it's use to develop interventions to reduce alcohol consumption within a lab based environment (Field et al., 2020).

Location of the assessments did not explain a significant amount of variance in the alcohol use data. Due to the lack of variation in assessment locations outside of participant's home, data was recoded as either being at home or out of home, reducing the specificity of our measurement and potentially negating the effect of environment on alcohol use. Social and contextual changes can influence alcohol use (Correia, Murphy, & Barnett, 2012), with bars and private residences in particular leading to high levels of alcohol consumption (Wray, Merrill, & Monti, 2014). Importantly, environments alone do not influence alcohol use. There is likely to be an interaction with social contexts, and the cognitive response evoked by situational cues (Vengeliene, Foo, & Kim, 2020) and social context (Erskine-Shaw, Monk, Qureshi, & Heim, 2017).

Frequency of craving consistently accounted for a significant proportion of alcohol use in exploratory analysis, with intensity of craving only accounting for a significant proportion of alcohol use when only sessions with reported alcohol consumption were analysed. Intensity suggested an inverse relationship with substance use, yet this may be a result of a lack of power. Another possible explanation may be the fact this was only in assessments where participants drank, meaning intensity of craving is only predictive when there is low motivation to abstain and the salience of cravings is increased, with previous work suggesting craving and motivation to change interact (Browne, Wray, Stappenbeck, Krenek, & Simpson, 2016). Craving has been shown to be consistently associated with substance use in EMA studies (Serre et al., 2015), given that craving predicted total day

consumption suggest a temporal proximity of the predictive validity of craving. In a sample of patients diagnosed with addiction, both substance related and person specific cues have been shown to increase craving and predict subsequent substance use (Melina Fatseas et al., 2015) consistent with our findings. Interestingly this relationship is proposed to be mediated by subjective self-control (Remmerswaal et al., 2019), an non-related measure of inhibitory control to that of the experimental paradigms, showing an interplay between control processes and motivations to consume alcohol. Craving and disinhibition have previously been shown to mediate the effect of cue exposure on substance use (Field & Jones, 2017), yet the interaction of all four variables has not been explored to our knowledge.

Our study has multiple strengths, we accounted for baseline measures of inhibitory control and alcohol use (via the ad-libitum taste test) allowing cross-validation between control conditions and real-world environments. In respect to alcohol use as a dependent variable, we conducted a variety of analyses such as daily consumption, only reported alcohol use session, and as a binary variable, allowing for robust analyses. Confounds (e.g. disturbances or alcohol intoxication) were accounted for as part of sensitivity analyses. The experimental paradigm used a greater number of testing sessions, allowing for dynamic changes in inhibitory control to be examined in comparison to previous studies (Jones et al., 2018), and importantly compliance was at acceptable levels for Ecological Momentary Assessment studies (Stone & Shiffman, 2002).

There were also a number of limitations. Firstly, we did not examine when alcohol was last consumed in relation to a testing session, and therefore cannot determine the effect of alcohol on inhibitory control or vice versa demonstrating the need for a more finite understanding of conditions during the initiation of drinking. Future research should ask

participants to complete sessions following alcohol consumption (Collins, Kashdan, & Gollnisch, 2003). Secondly, participants were not given a cut-off as to when they had to complete the testing session by, meaning that immediate fluctuations may not have been caught. Future EMA studies should control for the intended time of the RA and when the participant completed the session using cut-off points to examine the sensitivity of the paradigm. Our sample was comprised of heavy drinkers limiting the generalisability of findings, future research should seek to examine different groups of drinkers such as, light drinkers and those with substance use disorders to examine fluctuations in inhibitory control in the real world.

To summarise, we found no consistent association between inhibitory control or fluctuations of inhibitory control and alcohol use. Despite previous research we found a limited effect of location or motivation to alcohol related stimuli on inhibitory control. Frequency of craving most consistently accounted for a unique amount of variance in alcohol use, suggesting it may be a risk factor for alcohol use.

Chapter Summary

This chapter contributed to the overall aims of this thesis by demonstrating limited evidence that inhibitory control fluctuates in response to one's environment. While neutral SSRTs did fluctuate, alcohol SSRTs did not. Nevertheless, this may be due to location having to be condensed to at home or not reducing the measure's sensitivity. Findings do not support the notion that inhibitory control is predictive of alcohol consumption in any of the analysis models in this chapter. Findings did support the predictive validity of the bogus taste test as a proxy measure of alcohol consumption within laboratory settings. These findings are discussed in more length, alongside the other findings reported in previous chapters, in chapter seven.

Chapter 6

Limited evidence of associations between executive functioning and alcohol involvement in UK adolescents

This chapter presents a pre-registered cross-sectional study which has been published in Alcohol & Alcoholism (2021). The study was pre-registered on Open Science Framework (link is provided in text) and data is freely available along with analysis scripts. The format of the original article has been modified to match the other chapters in this thesis, however the content remains the same to that of which was published. To summarize contributions to this chapter, I designed the study which was approved by Andrew Jones. I collected data with Jo-Anne Puddephat, Jasmine Warren, Laura Baines and Florence Sheen. I analysed the data and wrote the manuscript. All authors provided feedback on the article before submission to Alcohol & Alcoholism and after the peer review process.

The aim of this study was to examine if executive functioning, inhibitory control and working memory were related to alcohol use and involvement in adolescents. Due to increased reward sensitivity in adolescents (Knyazev, 2004; Lopez-Vergara et al., 2012; Pardo, Aguilar, Molinuevo, & Torrubia, 2007) I sought to examine if reward could influence inhibitory control, and if this in turn was related to alcohol use or involvement. This built on the work from chapter three and four examining the role of reward in inhibitory control in relation to alcohol use in an under-researched population.

Abstract

Background: Deficits in motor inhibitory control and working memory have been hypothesised to be both a cause and consequence of heavy alcohol use. Adolescence is a critical developmental stage for inhibitory control and working memory, and also when individuals are most likely to initiate alcohol use. This study aimed to examine whether inhibitory control and working memory would predict alcohol use and involvement in a group of UK adolescents.

Methods: We recruited 220 (N = 178, female) adolescents, aged between 16-18, from eight higher education settings in the Merseyside region of the United Kingdom. Alcohol use was examined using the Timeline Follow-Back and involvement (and related problems) using the Adolescent Alcohol Involvement Scale. A reward-based inhibitory control task (Go/No-Go) was used to examine inhibition and reward sensitivity and a Self-Ordered Pointing task was used to measure working memory.

Results: Multiple regression demonstrated that neither inhibitory control (b = .02 (95% CI: -.21, .24) or working memory (b = -.12 (95% CI: -.30, .07) were significant predictors of alcohol use (units consumed). Inhibitory control (b = .61 (95% CI: .12, 1.09), specifically in the no reward condition, and school deprivation (b = .67 (95% CI: .06, 1.28) significantly predicted alcohol-related problems.

Conclusions: Our findings demonstrated limited evidence that deficits in specific mechanisms of executive functioning (i.e. motor inhibition and working memory) were associated with alcohol-related problems in UK adolescents. This study adds to an increasing body of literature suggesting weak or non-existent links between inhibitory control, working memory and alcohol use.

Introduction

In the United Kingdom (UK), initial experimentation with alcohol typically begins during early adolescence (Fernie et al., 2013). Eight percent of 11 year olds report consuming alcohol, which rises to 69% by age 15 (ONS, 2015). Alcohol consumption during adolescence is associated with a range of negative health outcomes, including neurocognitive deficits (Zeigler et al., 2005), short term physical harm, and risky behaviours (Boden & Fergusson, 2011). Furthermore, earlier onset of alcohol use is associated with increased risk of developing a substance use disorder in later life (Hingson & Zha, 2009). Encouragingly, recent work suggests that alcohol consumption is on the decline in youth drinkers in the UK (Oldham et al., 2019), with similar findings from Europe and North America (Looze et al., 2015; Norstrom & Svensson, 2014; Raninen, Livingston, & Leifman, 2014), however the prevalence of adolescent drinking is still a concern given the associations with a range of negative health outcomes (Zeigler et al., 2005).

Adolescence is a key developmental stage for (executive) cognitive functions and impulsive behaviour. Broadly speaking, impulsivity can be viewed as the opposite of a general cognitive ability, with the two constructs overlapping both theoretically and in measurement instruments (Bickel et al., 2012). Both constructs have been previously implicated as both the cause and consequence of excessive alcohol consumption (Coskunpinar, Dir, & Cyders, 2013). Key components of both constructs are inhibitory control and working memory (Bickel et al., 2012; Miyake et al., 2000). Inhibitory control is the ability to control or adjust one's behaviour in response to internal or external factors (Diamond, 2013; Logan et al., 1984b), and is multifaceted, encompassing a variety of conscious and subconscious behaviours, such as memory, attention and motor movements

(Diamond, 2013). Computerised tasks have been developed to objectively assess motor inhibitory control such as Stop Signal Tasks (SST) and Go/No-Go, in which a dominant motor response is established and participants are required to inhibit this response on a minority of trials (Diamond, 2013; Logan & Cowan, 1984). Luna, Garver, Urban, Lazar, and Sweeney (2004) suggest that basic level response inhibition, voluntary initiation, and suppression of behaviours, is present in early childhood and develops further during adolescence.

Working memory is a cognitive system which enables the provisional storage of information, no longer perceptually present, working with said information for complex cognitive abilities even in the presence of distractors (Baddeley, 1992; Engle, 2002). Working memory can be assessed using tasks such as the Self-Ordered Pointing Task (SOPT) (Petrides & Milner, 1982). Both inhibitory control and working memory are thought to develop during adolescence (Blakemore & Choudhury, 2006; Betty Jo Casey, Getz, & Galvan, 2008; Luna et al., 2004). Miyake and Friedman's (2012) review of executive functions states both inhibitory control and working memory demonstrate unity and diversity (they are correlated, yet separable; see also (Diamond, 2013)). Executive function deficits have been previously associated with risk related behaviour, with arguments made for slower maturation of the prefrontal cortex in adolescence along with developing cognitive control leading to risky behaviours (Casey et al., 2008; Steinberg, 2007).

Adolescence is also a period of increased reward sensitivity, which is associated with alcohol use across adolescent populations (Knyazev, 2004; Lopez-Vergara et al., 2012; Pardo, Aguilar, Molinuevo, & Torrubia, 2007). Heightened reward sensitivity has been operationalised as increased impulsive decision making and decreased inhibitory control to

rewards (Peeters et al., 2017). Findings suggest that reward sensitivity can promote adolescent alcohol use, with reactivity to rewarding cues able to predict current (van Hemel-Ruiter, de Jong, Oldehinkel, & Ostafin, 2013) and future alcohol use (van Hemel-Ruiter, de Jong, Ostafin, & Oldehinkel, 2015). Extrinsic motivation (through explicit rewards) can facilitate inhibitory control in both healthy and heavy drinking samples (Chung et al., 2011; Schevernels et al., 2015; Schevernels et al., 2014; Wilbertz et al., 2014) and adolescent samples (Demurie et al., 2016; Kohls, Peltzer, et al., 2009; Winter & Sheridan, 2014). From a neuroeconomics perspective, the use of extrinsic reward stimuli may increase the attributed value of inhibiting behaviour (Guttman, Moeller, & London, 2018) and provide a more comprehensive representation of the psychological mechanism of inhibitory control (Poulton et al., 2016).

Within individuals who drink alcohol, individual differences in impulsivity/executive functioning have been shown to be associated with quantity and frequency of consumption and related problems, escalation of use (Bø et al., 2017; Fernie et al., 2013), and transition to heavy drinking within adolescence (Wetherill, et al 2013). Elevated levels of impulsivity and motor disinhibition can pre-date alcohol involvement (and related problems) acting as a potential risk factor for heavy drinking and dependence following experimentation in adult samples (Dawe et al., 2004; Ersche et al., 2012; Whelan et al., 2014). Some research suggests the relationship between alcohol use and impaired executive functions to be weak or non-existent (Balodis, Potenza, & Olmstead, 2009; Caswell et al., 2016; MacKillop, Mattson, MacKillop, Castelda, & Donovick, 2007; Peeters et al., 2014). It is possible that individual, and methodological, differences across studies account for discrepant findings. For example, inconsistencies may be the result of the measure used (Fernandez-Artamendi, Martinez-Loredo, Grande-Gosende, Simpson, & Fernandez-Hermida, 2018; Smith et al., 2014).

Deficits in inhibitory control are suggested to be dose-dependent, with deficits appearing to be smaller in heavy non-dependent drinkers compared to dependent drinkers (Smith et al., 2014), particularly in females (Nederkoorn, Baltus, Guerrieri, & Wiers, 2009; Smith & Mattick, 2013; Smith, Mattick, & Sufani, 2015).

Inhibitory control is thought to fluctuate in response to environmental, psychological and physiological cues, acting as transient state triggers (De Wit, 2009; Inzlicht & Berkman, 2015; Jones et al., 2013). Heavy drinking episodes are often triggered by alcohol related cues (e.g. sight of alcohol) causing transient impairments of motor inhibitory control (Gauggel et al., 2010; Ryan et al., 2010) increasing craving and alcohol seeking behaviours (Christiansen et al., 2017). Evidence points to motor inhibitory control mediating the relationship between alcohol-cue exposure and subsequent alcohol consumption (Field & Jones, 2017). To our knowledge no study has examined the effect of alcohol-specific impairments in inhibitory control or working memory in adolescence. It is possible that that any association between executive functions and alcohol use (or involvement) is better explained by exposure to alcohol-related cues (or alcohol-context) than by non-alcohol-related cues, due to their potentially compromising effects. Indeed, 'hot' executive functions (those linked to emotional responding and reward sensitivity) are better linked to risky behaviours in adolescence (Prencipe et al., 2011). However, it is also possible that due to a shorter drinking history that adolescents may be less sensitive to exposure to alcohol-related cues.

Therefore, the aim of the present study was to examine the role of motor inhibitory control, reward sensitivity, and working memory on alcohol-related problems in adolescents in the Merseyside area of the UK through executive function measures containing alcohol-related cues. We hypothesised i) that individual differences in motor inhibitory control will

predict alcohol-related problems, with reward-specific inhibitory control predicting unique variance ii) individual differences in working memory will predict alcohol-related problems iii) individuals will have lower commission errors on the Go/No-Go task in the reward condition compared to the no reward condition. Hypotheses, methods and analysis plans were pre-registered on Open Science Framework (https://osf.io/yd9ua).

Methods

Participants

Two hundred and twenty participants (N=220, 18.75% male) were recruited from psychology courses across eight further education centres in the Merseyside area of the UK. Participants were eligible to take part if they were aged between 16 to 18 years of age (mean age=16.73 years, SD=0.68), had no previous or current diagnosis of substance use, ADHD, and/or a psychiatric or neurological disorder (sixteen participants were removed based on this criteria). Participants were asked if they had a current or previous diagnoses of the aforementioned by indicating yes or no on a check box. Inclusion/exclusion criteria were assessed via self-report. All participants provided informed consent, and both parents and further education centres received an information pack with details of the study prior to commencement. At the time of data collection in the Merseyside region, 20% of the population were considered to be among the most deprived in the UK (Taib et al., 2018), with 31.8% of children living in poverty in the region (Poverty, 2019), 27.6% of adults in the region drinking over the recommended government guidelines and 20.2% binge drinking, both above the average for England (England). The University of Liverpool Research Ethics Committee approved the study.

Our sample size was constrained to the availability and willingness of higher education institutions to be involved in the research. However, our informal power calculation suggested 187 participants were needed to detect a R^2 increase of .05 (explained by Inhibitory Control and Working Memory as tested predictors), and four covariates (age, sex, scores on the Family Affluence Scale (FAS) and school deprivation), at 80% power. We decided on an R^2 increase of .05 as Henges and Marczinski (2012) reported a correlation of r =.22 (R^2 = .048) between inhibition failures and total number of drinks consumed in 108 young social drinkers.

Self-report measures

Demographics and socio-economic status (SES)

Participants reported their gender and age before completing the six-item Family Affluence Scale (FAS; Currie, Elton, Todd, & Platt, 1997). Questions required participants to report on ownership of family car(s), whether they have their own bedroom, number of computers in the home, number of bathrooms etc. FAS is a well validated measure of SES in ages as young as 11 years old, and it has been shown to correlate well with other measures of SES such as disposable income (Hobza, Hamrik, Bucksch, & De Clercq, 2017; Torsheim et al., 2004). Scores ranged from 0 to 6 (higher score indicative of higher SES), with a mean score of 3.52 (SD=1.40).

Alcohol use

Participants completed a two-week retrospective diary of all alcoholic beverages they consumed, the Timeline Follow Back (TLFB), to assess frequency and quantity of alcohol

consumption. Participants were asked to record the number of units they consumed on a daily basis for the previous 14 days. A guide of units was provided for standard measurements of a variety of drinks, e.g. a small glass of wine or bottle of beer. Total units consumed during the previous 14 days and binge drinking frequency were the outcome measures.

Adolescent Alcohol Involvement Scale (AAIS) (Mayer & Filstead, 1979)

Participants completed the Adolescent Alcohol Involvement Scale, a 14-item self-report questionnaire measuring alcohol abuse and alcohol-related problems. Questions are rated on a 7-point Likert type scale, with a total possible score of 79. Options at the lower end are anchored at 0, e.g. question 2, "*When did you last drink alcohol?"*, 0=never used alcohol and 7=today. The 14-items are deemed to share sufficient common variance to create a composite alcohol use score (McKay & Dempster, 2016).

Index of Multiple Deprivation

For each school the level of deprivation was coded according to the Index of Multiple Deprivation (IMD) (Noble, Wright, Smith, & Dibben, 2006). IMD classifies deprivation based on the proportion of deprived individuals in an area (Cemlyn, Fahmy, Gordon, & Bennett, 2002; Noble et al., 2006). School deprivation scores ranged from 1 to 8, with 1 being high deprivation and 8 being low deprivation.

Behavioural measures

Go/No-Go Task

A hypothetical reward Go/No-Go task was administered (Demurie et al., 2016) consisting of 224 trials, of which 75% (N=168) were Go trials and 25% (N=56) No-Go trials, with half of all trials being rewarded. The fixation cross was presented at the start of a trial for 500ms, and the colour of the cross denoted if experiment 'points' (the reward) could be won for a correct response or not (Yellow=reward, Blue=no reward). Point-based rewards have been used in previous inhibitory control studies and participants respond with motivation to obtain these points, as they would a reward with actual monetary value (e.g. Geier et al., 2012; Marx et al., 2013; Miyasaka et al., 2019). Go and No-Go stimuli were presented on screen for up to 2000ms. On Go trials participants were shown images of soft drinks whereby they had to press the space bar as quickly as possible, while No-Go cues were images of alcohol drinks where they had to refrain from pressing the space bar. Between each trial was an inter-trial interval of 1000ms. Average Go reaction time (RT) and commission errors were calculated for both reward and no reward conditions. Before completing the task, participants were given a brief on the instructions, with 20 practice trials which could be repeated if necessary.

Self-Ordered Pointing Task (SOPT)

A modified SOPT was used to assess working memory (Petrides & Milner, 1982), that has been used in adolescences in relation to substance-use previously (Bourque et al., 2016; Carbia et al., 2017; Thush et al., 2008). We used alcohol rather than neutral-related images to invoke cue-exposure and ensure consistency with the Go/No-Go task. Participants were shown a set of alcohol related images (e.g. glass of beer), displayed in an array (grid

format), and asked to select one using their mouse. Following the selection of an image, a new page was displayed with the previous images, all images were automatically re-arranged into different positions. Participants were asked to select an image, while avoiding clicking the same image in a block, and avoid clicking the same position in the array. There were three blocks of 6 (2x3 array), 8 (2x4 array), 10 (2x5 array) and 12 (3x4 array) image arrays. The number of trials for each block was in accordance with the number of images in the array. Between each trial was an inter-trial break of 1000ms. At the end of all blocks, participants were told their total number of errors, as a measure of working memory. The SOPT has been shown to demonstrate good psychometric properties and relationships with other measures of working memory (Cragg & Nation, 2007; Ross, Hanouskova, Giarla, Calhoun, & Tucker, 2007).

Procedure

Schools were visited during the months of March to December 2019. Multiple visits to each school to assess longitudinal associations were planned, however we were unable to do this due to the Covid-19 pandemic. Our procedure for testing was identical across all schools. Before the visit, schools were sent information sheets and discussed the study with the lead author. Parents and guardians were informed about the study at least one week before the scheduled site visits. Consent was obtained on site from the students in line with British Psychological Society guidelines, as all participants were aged 16+. Participants were either tested at their school or at the University of Liverpool, with group sizes ranging from 10 to 20 participants and multiple researchers present. All participants sat at individual computers or laptops to complete the experiment. Participants completed the battery of

questionnaires, followed by the Go/No-Go task and SOPT. Upon completion of the study participants and teachers were debriefed as to the purpose of the study.

Data reduction and analysis

Data was cleaned and analysed in R, using the 'dplyr' and 'lme4'packages (r-script can be found on OSF). Average Go RT was calculated for both reward and no reward conditions. Outliers were identified using box plots and removed from individual analyses. Reaction time data from one individual was removed due to non-responding on Go-Trials. Ten participants (4.54%) were removed for outlying commission errors. Commission errors were calculated for reward and non-reward trials, along with an overall number of commission errors. Total errors were recorded on SOPT as the measure of working memory.

We examined whether multilevel modelling was appropriate for data analysis due to the use of nested data (individuals > schools). These models were not a better fit of the data – however, this is consistent with Fernie et al. (2013)'s data which indicated any clustering effect of school effects was nominal, as such we used standard linear regression analyses

Results

Participant demographics (Table 6)

Demographic information for the complete sample stratified by school are reported in Table 6. Of the 220 participants, 57.73% had consumed alcohol in the previous two weeks to the testing sessions. Fifteen (6.82%) of the participants were classified as heavy drinkers in accordance with UK guidelines, having drunk twenty-eight or more units over a two-week

period. One individual reported drinking an implausible amount (265 units) as such we rescaled this to the next largest value + 1. Average consumption was 14.01 units (SD=13.32: range 1-73).

Table 6 School characteristics, means and standard deviations

School Code	**1** **N=20**	**2** **N=95**	**3** **N=20**	**4** **N=23**	**5** **N=11**	**6** **N=10**	**7** **N=26**	**8** **N=15**	**Total** **N=220**
Male %	15.00	0	100.00	47.82	27.27	100	0	0	18.75
Age	16.80 (.52)	16.55 (.58)	16.4 (.50)	16.57 (.59)	17.71 (.30)	16.90 (.32)	17 (.49)	16.8 (.41)	16.73 (.68)
TLFB Total	4.75 (12.00)	6.25 (8.61)	7.46 (12.60)	11.23 (15.74)	21.36 (22.93)	16.5 (17.37)	.5 (2.16)	5.87 (9.05)	8.95 (21.39)
AAIS	29.65 (14.62)	28.57 (11.15)	31.45 (9.48)	34.74 (7.31)	33.64 (13.44)	31 (8.98)	25.04 (11.65)	29.87 (6.74)	29.76 (11.12)
FAS	3.25 (1.59)	3.82 (1.38)	3.2 (1.28)	3.13 (1.33)	3.27 (1.85)	3.8 (1.14)	2.88 (1.42)	3.67 (.82)	3.49 (1.40)

The effect of reward on inhibitory control

To analyse the effect of reward on inhibitory control we conducted a paired samples t-test on commission errors in reward and no reward conditions. There was a significant difference in commission errors between the reward (M=6.48, SD=4.43) and no reward (M=5.18, SD=3.82) conditions (t(209) = 4.84, p<.001, d=.31), this result remained significant with commission error outliers in the sample (t(219)=4.97, p<.001). In an exploratory analysis, we analysed the effect of reward on Go RTs using a paired t-test. There was a

significant difference between go RTs in reward (M=435.29, SD=49.27) and no reward (M=443.53, SD=63.85) conditions (t(209)=3.16, p<.01, d=.15), this result was non-significant with commission error outliers in the sample (t(219)=1.03, p=.306).

Predicting individual alcohol use (TLFB – Table 7)

A multi-level model was not a significantly better fit for the data than the single-level model (χ^2 (1)=3.34, p>.05), as such ordinary least squares multiple regression was used to analyse the data, see Table 7. Model A included commission errors and SOPT errors as predictors. Model A did not explain a significant amount of variance in the data, F(2,207)=.365, p=.695, BF_{01}=22.75, adjusted R^2<.01, with bayes factors ranging from 0.15 to 0.19. In Model B commission errors was split into reward and no reward with SOPT errors as predictor variables. Model B did not significantly account for the variance in TLFB data, F(3,206)=.700, p=.553, BF_{01}=49.59, adjusted R^2<.01, bayes factors ranging from 0.15 to 0.27. In Model C, we included the variables from Model A and included age, gender, FAS and school deprivation as covariates. In Model C, commissions errors were not split into reward or no reward commissions due to no significant association in Model B. Model C explained 12.46% of variance in the data, F(6,203)=5.960, p<.001, BF_{01}=.12, adjusted R^2=.125. Gender (β=-9.31 (95% CI: -13.50 to -5.13), p<.001) and age (β=3.19 (95% CI: .83 to 5.54), p<.01) were significant predictors, suggesting as age increased so does alcohol consumption. Males consumed significantly more units of alcohol (M=15.30, SD=18.4) than females (M=5.34, SD=9.38: t(49)=3.49, d=.88). There was limited evidence of multicollinearity across the three models (VIFS<1.76). For sensitivity analyses, these same models were ran without outliers removed, the results did not differ for any of the models reported above. A logistic regression in which individuals who reported drinking vs not

drinking as the outcome did not substantially change the pattern of results, nor a model in which only alcohol consumers were included in the analysis.

Table 7 Unstandardized beta values and 95% confidence intervals indices for multiple regression models TLFB

	Predictor Variable							
	Reward errors	No reward errors	Total errors	SOPT errors	Gender	Age	FAS	School deprivation
Model A	-	-	.05 (-.19, .29)	-.08 (-.28, -.11)	-	-	-	-
Model B	-.19 (-.66, .28)	.34 (-.21, 0.88)	-	-.09 (-.29, .10)	-	-	-	-
Model C	-	-	.02 (-.21, .24)	-.12 (-.30, .07)	-9.31 (-13.50, -5.13)	3.19 (.83, 5.54)	.20 (-0.99, 1.38)	.33 (-.32, 0.98)

Legend: errors = commission errors; FAS = Family Affluence Scale; SOPT = self-ordered pointing task

AAIS (Table 8)

A multi-level model was not a significantly better fit for the data than the single-level model (*p>.05),* as such a multiple regression was used to analyse the data, see Table 8. In Model A we included SOPT errors and commission errors as predictors. The multiple regression model was not significant, $F(2,207)=1.617$, $p=.201$, $BF_{01}=6.66$, adjusted $R^2=.005$, accounting for 0.5% of variance in the data. Model B was run with commission errors split into reward and no reward conditions and SOPT errors. The model was not significant, $F(3,206)=1.95$, $p=.123$, $BF_{01}=7.63$, adjusted $R^2=.013$, accounting for 1.3% of variance in the

data. However, no reward errors were significantly associated with the AAIS score (β=.53 (95% CI: .04 to 1.02), *p*=.033) and commission errors were thus split into reward and no reward commissions in Model C. Results for Model C (including covariates) suggest that no reward commission errors and school deprivation predict 5.05% of the variance ($F(7,202)=2.59$, $p=.014$, $BF_{01}=3.42$, adjusted $R^2=.050$). As no reward commission errors increased, AAIS score increased (β=.61 (95% CI: .12 to 1.09), *p*=.014). School deprivation had a significant relationship with AAIS score (β=.67 (95% CI: .06 to 1.28), *p*=.031), as deprivation decreased AAIS score increased. There was limited evidence of multi-collinearity across the models (VIFs < 1.78). For sensitivity analyses, these same models were ran without outliers removed. The results did not differ for any of the models reported above.

Table 8 Unstandardized beta values and 95% confidence intervals for multiple regression models AAIS

	Predictor Variable							
	Reward errors (95% CI)	No reward errors (95% CI)	Total errors (95% CI)	SOPT errors (95% CI)	Gender (95% CI)	Age (95% CI)	FAS (95% CI)	School deprivation (95% CI)
Model A	-	-	.17 (-.04, .39)	-.11 (-.28, .07)	-	-	-	-
Model B	-.11 (-.53, .30)	.53 (.04, 1.02)	-	-.11 (-.29, .06)	-	-	-	-
Model C	-.15 (-.57, .26)	.60 (.12, -.1.08)	-	-.12 (-.29, .06)	-2.15 (-6.07, 1.78)	1.94 (-.27, 4.15)	-.01 (-1.22, 1.09)	.67 (.06, 1.28)

Legend: errors = commission errors; FAS = Family Affluence Scale; SOPT = self-ordered pointing task

Discussion

In the present study we examined if measures of executive function – motor inhibitory control and working memory - were associated with alcohol-related problems or consumption in a sample of adolescents. We also examined whether reward sensitivity interacted with motor inhibitory control to predict unique variance in alcohol-related problems. We found a significant association between no reward commission errors and alcohol-related problems, yet no other measures of executive functioning were significant for alcohol-related problems or consumption. In the presence of a reward, participants' motor inhibitory control was significantly poorer.

Contrary to our hypotheses motor inhibitory control (as measured by commission errors on a Go/No-Go task) was not associated with alcohol consumption, with Bayesian analysis suggesting findings were evidence supportingthe null hypothesis. Motor inhibitory control performance in no reward conditions was associated with alcohol-related problems. Specifically, as motor inhibitory control became poorer alcohol-related problems increased. These findings provide limited support for theoretical models or empirical data which suggest that motor inhibitory control and working memory are associated with alcohol involvement (Carbia et al., 2018; Field & Jones, 2017; Mahedy et al., 2018). The lack of a consistent association across different studies may be due to the precision of the measure of inhibitory control administered, and the samples used (e.g. lighter vs heavier drinkers).

The presence of reward increased commission errors, an effect which in isolation is unexpected (Chung et al., 2011; Schevernels et al., 2014; Wilbertz et al., 2014). However, there is evidence that reward can impair inhibition (Demurie et al., 2016; Kohls, Herpertz-

Dahlmann, et al., 2009; Miyasaka & Nomura, 2019a; Padmanabhan et al., 2011), and it is possible that prompting reward on Go trials led to faster reaction times (which we observed, in comparison to non-rewarded trials), which in turn increased inhibition errors due to a speed – accuracy trade-off (Leotti & Wager, 2010). Alternatively, Pessoa (2009) suggests that a deleterious effect of reward on inhibitory control is the result of a (finite) resource allocation to maximise the chance of reward, causing other cognitive systems to suffer. The effect of reward should be examined with different reward types (e.g. hypothetical or actual, financial or non-financial) as evidence suggests reward salience changes as age develops (Miyasaka & Nomura, 2019a). As such, the current reward may not have been sufficiently salient to the participants.

Interestingly SES, reported through the FAS, did not explain a significant proportion of the variance in drinking behaviour among adolescence. This is in line with previous work, which shows no clear pattern between drinking behaviour and SES in adolescence (Hanson & Chen, 2007). School-level deprivation scores did explain a significant proportion of drinking behaviour, but there was not a significant difference between levels of school deprivation and drinking behaviour. This may be explained by the difference in the number of participants recruited from each school deprivation group, which was assigned based on school postcode.

Findings from the current study should be assessed in light of limitations. Our sampling was limited to one geographical area in the UK, characterised by greater than average deprivation. Future studies should attempt to recruit from multiple geographical locations to increase the representativeness of these findings. Second, a two-week timeline follow-back may not have been sufficiently long enough to capture alcohol consumption in adolescents, as access to alcohol in these samples may be varied (Jones-Webb et al., 1997).

Future research should attempt to replicate these findings using measures of alcohol use over longer time periods (Buu et al., 2014). In relation to this, self-reported consumption may be prone to memory biases and under-reporting (Livingston & Callinan, 2015). Third, due to testing constraints we were unable to assess other factors which might be related to both executive functioning and alcohol use, such as impulsive personality traits and mental health. Fourth, we used an unbalanced Go/No-Go design to assess inhibitory control. Future research should use a counter-balanced Go/No-Go design (in which the contingency for responding / inhibiting to neutral cues is reversed) to disentangle any attentional bias towards alcohol-related cues. Similarly, we combined reward sensitivity and inhibitory control within the Go/No-Go task, which is both a strength and limitation as it provides a more realistic outcome given the interdependency of these processes, but limits direct conclusions for either in isolation. Finally, we originally aimed to conduct follow up assessments for each participant, however these were unable to take place due to the COVID-19 pandemic, and so we are limited to cross-sectional associations. Examination of prospective associations throughout adolescence may demonstrate different results (e.g. Fernie et al, 2013).

Findings from the current study have implications for both alcohol research in adolescents and examination of executive functioning in this population. The majority of models and empirical studies hypothesise an overly simplistic association between the two variables, with varying degrees of support (see Fernie et al., 2013, Wiers et al. 2010). However, inhibitory control is sensitive to a number of inputs, including reward and motivation, and in order to make clearer predictions about behaviour the interactions between inhibitory control and external/internal inputs should be modelled. This is the first study, to our knowledge, to examine more complex relationships between inhibitory control and alcohol consumption in such a manner with this population.

To conclude, this study found limited evidence of associations between measures of executive functioning (motor inhibition and working memory) and alcohol use/involvement in adolescents. This adds to a growing number of studies which suggest that the link between inhibitory control (and working memory) and alcohol use is weaker than first thought. To examine the role of executive functioning on alcohol use more accurately, future studies should use multiple measures of constructs of executive functioning, allowing for multiple of individual associations and a combined composite measure.

Chapter Summary

This chapter contributed to the overall aim of the thesis by showing limited evidence of executive functions and alcohol use. The prospect of reward appeared to significant impair inhibitory control, but this may have been a result of speed-accuracy trade off, as go RTs were significantly faster in the reward condition. I found no consistent evidence for an association between inhibitory control and alcohol use/involvement, with Bayesian analysis suggesting evidence for the null hypothesis. Findings taken together suggest the association between executive functions and alcohol use may be weaker than once thought, which is discussed at more length in the following chapter.

Chapter 7

General Discussion

This thesis had an overall aim of examining the effect of environment and rewards on inhibitory control in order to develop more complex theoretical models of inhibitory control in heavy drinkers. It was inspired by two main bodies of research. First, evidence from Verbruggen et al. (2014) suggests that models of inhibitory control are too simplistic, conceptualised as purely reactive stopping in the literature and not accounting for proactive control. In health research, behaviours are described as competing with one another (i.e. consume a substance or not) via cognitive valuation process that leads to an individual executing a given behaviour (Berkman, 2018). While the two lines of research are distinctly separate fields, in terms of heavy drinkers there are aspects that can be drawn upon to improve our theories of inhibitory control. Second, evidence that inhibitory control is a fluid state and that potential environmental or psychological risk factors that may cause state fluctuations in heavy drinkers (De Wit, 2009; Jones et al., 2013). This thesis attempted to extend this to an individual's environment and motivations and their proactive effect on behaviour, e.g. if an individual knew they were attending a bar they may be primed to consume more alcohol. Knowledge acquired for this thesis can be used to develop and update theories of addiction (e.g. De Wit, 2009; Goldstein & Volkow, 2002), and develop health interventions for heavy drinkers. As a result, non-dependent heavy drinkers were recruited for three of the four studies, with the final study recruiting 16- to 18-year-olds, examining an at-risk population for heavy drinking with the potential to transition on to substance use disorders. The current chapter summarises the main findings from each study and provides a discussion of findings across studies in relation to past literature and contemporary theories of addiction.

Summary of main findings of each study

Chapter three was a meta-analytic investigation of the effect of reward on inhibitory control. Given the inconsistencies in findings from individual studies, I conducted a meta-analysis to see if there was a significant pooled effect of reward on inhibitory control, to provide a basis for the presented experimental chapters. I aimed to examine whether the effect of reward was moderated by a range of factors including inhibitory control task, type of reward, clinical vs non-clinical samples, and age to try and account for the heterogeneity in the current evidence base. I hypothesised that reward would improve inhibitory control. These findings were supported by the pooled effect size estimates, which were robust to influential cases and outliers. No specific directional hypotheses were made in regard to the effect of moderators, given the equivocal findings of the individual studies. Nevertheless, the effect of reward on inhibitory control does not appear to be moderated by reward type, clinical sample vs non-clinical sample, or age. There was a significant moderation by task type with a significant effect with all tasks. This was likely driven by large effect sizes in Stroop tasks, upon their removal the moderation was not significant. Analysing tasks separately did not produce a substantial reduction in heterogeneity across effect sizes. Findings suggest that inhibitory control is a transient state that fluctuates in response to rewards irrespective of moderators. There was a lack of research to investigate the effect of reward on reactive and proactive control and provide a more complex model of inhibitory control. Evidence of bias in the literature was found suggesting that results should be interpreted with caution due to potentially inflated effect sizes and small sample sizes.

Following on from this, chapter four aimed to examine whether exposure to appetitive cues can lead to a disinhibited state, and if the prospect of reward can facilitate inhibition across two experiments. A Stop-Signal task was manipulated to show different reward cues (embedded in stop signals and fixation crosses), with a variety of response stimuli (alcohol, chocolate or neutral cues), and environments (bar lab or a standard lab). Results did not provide consistent evidence for an influence of reward or appetitive cues on inhibitory control. Results suggest that a high reward (compared to a low or no reward) and increased likelihood of reward can induce proactive slowing (reduced RTs for reward trials) and reduce trigger failures, suggesting a downstream effect of reward on inhibitory control. Participants displayed an increase in proactive inhibitory control as a result of a reward being present. There was no consistent effective of appetitive cues on influence inhibitory control. Despite the use of personalised alcohol cues, which should increase deficits of inhibitory control (Fatseas et al., 2015), our findings contrast that of meta-analyses of empirical evidence (Jones et al., 2018; Burton et al., 2021). Such meta-analyses rely on pooled-effects, which in the case of alcohol-related cues may be overestimated as a result of small study and publication biases.

Results presented from lab-based studies showed no effect of environment on inhibitory control or alcohol use, potentially due to completion in highly controlled lab environments. In an extension of chapter four, chapter five sought to examine fluctuations in inhibitory control in the real world using Ecological Momentary Assessment (EMA) techniques. Building on Jones et al. (2018) this provided a naturalistic examination of inhibitory control and alcohol use. The study aimed to examine if fluctuations in inhibitory control could predict alcohol consumption. Based upon previous literature it was hypothesised that inhibitory control would fluctuate in response to the participant's current

environment, with an aim to identify high-risk situations that may result in impaired inhibitory control and initiation of drinking. Inhibitory control was assessed using a Stop-Signal task using both alcohol and neutral stimuli, along with assessments of emotions, location, intensity, and frequency of cravings. Inhibitory control performance for neutral stimuli significantly accounted for whether a participant drank or not, but not the frequency of alcohol consumed, throughout the day. Exposure to alcohol-cues did not predict alcohol consumption. Interestingly, frequency of craving consistently accounted for alcohol use and quantity consumed, with a positive relationship between the variables. This supports robust links between craving and alcohol use established in laboratory and naturalistic studies. Participant location at the time of assessment did not explain a significant amount of variance in the alcohol use data, however I saw little variance in location data and had to adjust the variable within the models.

Lastly, chapter six aimed to investigate the association between executive functioning (inhibitory control and working memory) and alcohol involvement in adolescents (16 to 18 years old), extending the literature and previous research to an under-researched sample with greater reward sensitivity (Altikulaç, Bos, Foulkes, Crone, & van Hoorn, 2019). A modified Go/No-Go task was used based upon Demurie et al. (2016) examining the effect of reward and cue type (alcohol and neutral) on inhibitory control. Due to testing restraints of the population, surrounding ethics and recruiting the population, the effect of environment could not be examined. A measure of working memory was included due to the potential interplay with inhibitory control (Diamond, 2013), and to build a more complex understanding of the relationship between inhibitory control, reward and alcohol involvement. The presence of a reward in the fixation crosses of the Go/No-Go task significantly impaired inhibitory control as commission errors increased, primarily through enhanced responding to Go stimuli on

reward trials producing a speed-accuracy trade-off (Leotti & Wager, 2010). Neither inhibitory control nor working memory was associated with alcohol use, with Bayesian analysis suggesting evidence for the null hypothesis. Interestingly, inhibitory control in the no reward conditions was associated with alcohol-related problems, as the former became poorer the latter increased. The lack of consistent associations between inhibitory control and alcohol use and involvement may be due to the measure of inhibitory control and the sample of interest (e.g., light vs heavy drinkers, young vs adult populations).

Contributions to theoretical models

Below I outline the direct contributions my research has had on the key theoretical predictions surrounding inhibitory control and reward in alcohol use.

Inhibitory control as a state variable

The findings from my empirical work suggest there is limited evidence for theoretical models that suggest inhibitory control is a state variable that fluctuates in response to psychological factors and environmental triggers (De Wit, 2009; Jones et al., 2013). There was some evidence that reward caused momentary fluctuations in inhibitory control particularly from the findings of the meta-analysis, yet this did not specify differences in reactive or proactive control, findings across chapters four and six do not show a consistent effect of reward in inducing momentary fluctuations of inhibitory control. Contrary to a plethora of research I found no consistent fluctuations in inhibitory control in response to

appetitive cues, both within tasks and across environments, despite the use of personalised alcohol stimuli.

It is possible that the lack of evidence is because current models of inhibitory control are too simplistic in the mechanistic process in which factors influence inhibitory control and subsequently affect alcohol consumption, with no theory that accounts for the effect of reward and environment on said phenomena. Recent work suggests that self-regulatory behaviours, such as response inhibition, are shaped by reward systems (Lopez et al., 2017) with particular emphasis given to the probability of reward driving such inhibitory mechanisms (Zajkowski, Krzemiński, Barone, Evans, & Zhang, 2021). To provide more accurate models and underlying mechanisms of inhibitory control, in clinical and none-clinical populations, a modular approach accounting for multiple factors is proposed to provide the most accurate and reliable approach (Sebastian, Forstmann, & Matzke, 2018).

One such internal influence hypothesised to influence inhibitory control is reward. Taken together, findings across the four chapters suggest that there is a complex relationship between reward and inhibitory control in heavy drinkers. My meta-analysis provides support for the notion of reward improving inhibitory control, but there was insufficient data to differentiate and examine reactive and proactive control. Across two studies in chapter four I found no consistent evidence for an effect of reward on inhibitory control, across a variety of rewards and types of presentation. Particularly in study two, of chapter four, evidence was found to suggest reward improves proactive control but not reactive control consistently. Proactive control was only improved in high reward contexts, with similar findings in chapter six, suggesting the presence of a reward alone is not enough but it must be salient to the individual to influence their behaviour (Liljeholm & O'Doherty, 2012; Stoppel et al., 2011).

In chapter four, study two demonstrated that as reward increased so too does the proactive slowing/inhibitory control. Findings fit with previous experimental work in the field that suggest the expectation of a reward alone can be intrinsically motivating to a person (Herrera et al., 2019). Herrera et al. describe this as a "kick start" effect inducing a rapid immediate release of dopamine, yet the cue would have to be a rapid salient cue (Liljeholm & O'Doherty, 2012). The absence of said "kick start" effect in our sample of heavy drinkers may be a result of blunted responding to non-substance related stimuli (Schacht et al., 2013), leading to hyposensitivity reward-related stimuli reducing their influence on inhibitory control (Byrne & Worthy, 2019). Goal-directed behaviour requires the recruitment of a variety of cognitive processes (Verschure, Pennartz, & Pezzulo, 2014) that influence inhibitory control.

Findings from the meta-analysis suggest that reward does not have a consistent effect on inhibitory control, depending on the measure used, which may help explain discrepancies observed in this line of research. Complex measures of inhibitory control such as the Flanker and Stop-signal task require increased top-down processing to account for updating of rules and monitoring (Garon et al., 2008). One such underlying process is working memory (Burnham et al., 2014; Vandierendonck, 2014), which when under increased load can results in an impairment of inhibitory control (Burnham et al., 2014) as task goals are not maintained (Munakata et al., 2011). For working memory to be able to reach this point to maintain task rules, the information must first attract the attention of the individual.

Another plausible explanation is that the reward is not attenuated, if attentional processes do not pick up on task relevant signals the correct behavioural response is not

executed (Xu et al., 2017). Neuroimaging research during a rewarded Stop-Signal task, showed that the facilitation of inhibition was an indirect effect that stemmed from increased attentional processing (Wang et al., 2018). Little research accounts for rewarding distractors when attempting to explain the relationship between inhibitory control and reward. A recent meta-analysis has shown, even when told to ignore distractors, rewarding cues (e.g., food or alcohol) can disrupt cognitive processes (Rusz, Le Pelley, Kompier, Mait, & Bijleveld, 2020). Alcohol-related stimuli in an environment may disrupt processing of rewarding stimuli, such as alcohol adverts (Courtney, Rapuano, Sargent, Heatherton, & Kelley, 2018), especially when consumption of alcohol is an individual's goal (Brown, Duka, & Forster, 2018). Rather to increase the prospect of reward improving inhibitory control, the reward must align with the individual's goals creating a bias towards goal related stimuli rather than substance-related, as shown in smokers and heavy drinkers (Brown, Duka, & Forster, 2018; Brown, Forster, & Duka, 2018; Godara, Van Bockstaele, & Wiers, 2020).

An alternative explanation for the lack of consistent significant effect of reward on inhibitory control is how SSRTs were calculated. SSRT estimations result in a wide body of literature not accounting for the influence of trigger failures (responding during stop trials), and the influence on the mean SSD in turn leading to an overestimation of SSRTs (Band et al., 2003; Verbruggen, Aron, Band, et al., 2019). Trigger rates themselves provide a measure of response inhibition, being able to account for group differences of individuals with ADHD (Weigard, Heathcote, Matzke, & Huang-Pollock, 2019) and schizophrenia (Matzke, Curley, Gong, & Heathcote, 2019), yet no such examination has been conducted in substance use populations. Re-examination of the effect of reward on SSRTs when trigger failures are accounted for shows the effect of reward is abolished, but trigger failures are reduced intermittently when there is a prospect of reward (Doekemeijer et al., 2021). To summarise

the effect of reward on inhibitory control is complex, appearing to not be a direct effect on inhibitory control itself but rather subcomponents and supporting cognitive processes.

Theoretically it is hard to draw concrete conclusions given the inconsistencies of findings presented in this thesis of the role of reward and inhibitory control in heavy drinkers. Within the studies presented alcohol related cues are used, which themselves are rewarding (Boileau et al., 2003; Leeman et al., 2014). Given the shared nature of inhibitory control and reward pathways (Bari & Robbins, 2013; Weafer et al., 2017) alternative cognitive processes and factors must be examined. Alternatively a recent paper by Weafer et al. (2021) suggests that, following alcohol consumption, social drinkers are increasingly responsive to the rewarding effects of alcohol which is related to their inhibitory control. Potentially this disinhibiting effect of alcohol may extend to cues, both in experimental tasks and one's environment, highlighting the need to account for attention and the role it plays in inhibitory control fluctuations.

The effect of environmental cues on inhibitory control & it's effect on alcohol related behaviours

Across the chapters presented in this thesis there was no consistent evidence of an effect of environment or appetitive cues on inhibitory control. In chapter four, study two, there was weak evidence for an effect of environment on inhibitory control whereby inhibitory control was poorer in a bar environment. Chapter five used an EMA paradigm and found that inhibitory control to neutral stimuli fluctuated across environments, findings may have been a result of having to collapse locations due to insufficient numbers. In respect to

alcohol specific cues no consistent effect on inhibitory control was observed, or predictive validity of alcohol-related behaviours.

Findings displayed no consistent effect of task embedded appetitive cues on inhibitory control, particularly in inducing disinhibited states contrary to prior research (Field & Jones, 2017; Monk et al., 2016). Theoretically alcohol-related should have been salient to the individual through the use of personalised stimuli to increase internal reliability (Christiansen et al., 2015a). Jones et al. (2018) meta-analysis suggests that appetitive cues do indeed impair inhibitory control, yet suggest that for alcohol-related effects is a result of publication bias in the literature. More recent work has also failed to demonstrate an effect of cue reactivity on inhibitory control (Baines et al., 2019a; Field & Jones, 2017; Weafer & Fillmore, 2012). SST's, with various manipulations, may diminish the effect of appetitive cues on inhibitory control, through increased cognitive demands such as response selection as well as response inhibition may influence RTs and inhibition errors (Simmonds et al., 2008).

While experimentally embeddedcues may have less of an effect on an individual's inhibitory control, one expected based on prior literature the environment (i.e. in a bar or alcohol rich environment) as a whole would have. Laboratory based studies presented in this thesis showed no support for the theoretical notion that inhibitory control is a "state" like variable that can fluctuate in response to alcohol stimuli in an individual's environment. A lack of significant effect may be due to participants being observed; experimenter presence has previously negated experimental manipulations of inhibitory control (Yu, Tseng, Muggleton, & Juan, 2015). Chapter five provides tentative support for the aforementioned theory, while environment only influenced neutral SSRTs, inhibitory control in general did

fluctuate over the days showing state changes occurring, similar to that of Jones et al. (2018) and Remmerswaal et al. (2019).

EMA research found no association of inhibitory control and alcohol use on a given day. Remmerswaal et al. (2019) showed impairments in self-control were predictive of the likelihood of drinking at subsequent occasions. However, with objective measures of inhibitory control as in Jones et al. (2018), it appears that changes in inhibitory control throughout the day rather than inhibitory performance per se predict alcohol consumption. Participants were not assessed during 'temptation episodes' (Waters et al., 2012), when cognitive biases are enhanced for substances and are predictive of (re)lapse (Marhe et al., 2013). One would expect inhibitory control to be poorer during temptation episodes (Muraven, Collins, & Neinhaus, 2002), this would be the case particularly when the resource model is applied to inhibitory control (Muraven et al., 2002).

An individual's drinking environment is characterised by both physical and psychological attributes that potentially interact at both a given moment and shape future drinking occasions (Freisthler, Lipperman-Kreda, Bersamin, & Gruenewald, 2014; Stanesby, Labhart, Dietze, Wright, & Kuntsche, 2019). The use of EMA methodologies allowed the examination of these complex relationships, more so than labs, to better inform theoretical models. Previous work has shown that being at home and in public places (i.e. bars) are particularly high risk situations for heavy drinking due to contextual factors and previous drinking experiences (Freisthler et al., 2014; Trim, Clapp, Reed, Shillington, & Thombs, 2011). Findings presented in this thesis show little evidence for an effect of environment (as in location or cues within an environment) on inhibitory control or alcohol consumption. Stanesby et al. (2019) note how most EMA studies fail to investigate context and are

normally in younger populations, meaning the current literature fails to investigate the complexity of an individual's real-world environment.

This thesis examined the effect of reward and environment on inhibitory control in an attempt to build a more complex understanding of what constitutes effective inhibitory control. I found very little support that reward or environmental cues cause fluctuations in inhibitory control. Given that inhibitory control and alcohol consumption was examined across multiple environments, laboratory and real-world, with a variety of factors (mood and previous consumption) attempts were made to build more complex models of inhibitory control in heavy drinkers and accounts for their drinking behaviour. Findings provide little support for the theoretical notion that inhibitory control is a transient state that fluctuates in response to one's internal and external environment (De Wit, 2009; Jones et al., 2013). Future research should seek to clarify if inhibitory changes during temptation episodes influence or predict alcohol consumption, while accounting for other factors such as craving and mood.

Methodological inconsistencies & pre-registered examinations

Findings presented in this thesis do not consistently support prior research on cue reactivity or the effect of reward on inhibitory control. A potential explanation for this may be due to the methodologies employed and the inconsistency in scientific practices (procedures and analysis) between the current and previous research. A recent systematic review highlighted how the robustness of findings from cognitive research is questionable given the variability in design and analysis among other issues (Pennington, Jones, Bartlett, Copeland, & Shaw, 2021). Pennington and colleagues particularly note discrepancies in

stimulus presentation and matching. A strength of the experiments presented in this thesis are that experimental stimuli were based on size and luminosity. In addition, alcohol stimuli were personalised to the individual's preference to increase reliability (Christiansen et al., 2015a) and the majority of studies matched these to appetitive stimuli to account for the incentive value of appetitive stimuli in general and to differentiate as to whether there is an alcohol-specific effect (Monk, Qureshi, Pennington, & Hamlin, 2017; Pennington, Qureshi, Monk, Greenwood, & Heim, 2019). Nevertheless, despite previous research not utilising the same methodological standardisation, which may explain inconsistent findings, the work presented in the current thesis did where possible to ensure methodological rigour.

Inconsistent methodological paradigms across the field may lead to inconsistent results and lack of generalisability. Little previous research has examined the effect of reward and environmental cues in the manner employed in this thesis. SST and GNG assess response inhibition, the use of reward provides an extra layer of complexity with motivational inhibition potentially competing with the motivational salience of environmental cues. Given the difference in methodologies, effects observed may be context specific rather than fluctuations in inhibitory control using a common process (Gärtner & Strobel, 2021; Miyake et al., 2000). Inhibitory control is multi-faced (Diamond, 2013) with a variety of tasks to measure respective sub-components (e.g. response inhibition and SST/GNG)(Bari & Robbins, 2013) meaning comparison across tasks is difficult due to different sub-components examined.

Beyond methodological inconsistencies with previous literature, analysis techniques of indices, and their measurement, of inhibitory control may cause inconsistencies (e.g. the use of SSRTs and how they are calculated). Recent work by Doekemeijer et al. (2021)

highlights how when re-analysing SSRTs for SSTs previous “robust” effects are no longer present demonstrating the influential effect of how inhibitory control indices are calculated. As methods progress and new calculations of indices of tasks are developed to provide more reliable estimates of cognitive processes, prior research will need to be reanalysed to avoid a potential inflation in effect sizes within the literature due to unreliable measures (Verbruggen et al., 2019).

In continuation of the influential effect of analysis techniques on findings, inferential analysis can influence conclusions. Different analytical techniques can yield different findings, in combination with publication bias and the potentially for research QRPs, might lead to inflation of effect sizes (Schäfer & Schwarz, 2019). Inflation of effect sizes is particularly salient in work that is not pre-registered, making it difficult to draw accurate conclusions of an effect in turn reducing the power of future research (Fraley & Vazire, 2014; Schäfer & Schwarz, 2019). Psychological literature is proposed to have 96% positive results compared to 44% in registered reports indicative of publication bias (Scheel, Schijen, & Lakens, 2021). There is a common misconception that a p-value represents whether the null hypothesis is true or not, and this shows an overreliance and misunderstanding of null-hypothesis testing(Lakens, 2021). If individuals reject null hypotheses based upon their *p*-value, ceasing the line of research, publication biases may be increased. An alternative analysis technique is Bayesian analysis, allowing comparison between hypotheses (null and alternative) and providing quantifiable degrees of evidential strength for either (Dienes, 2016) . Use of Bayesian analysis may allow production of more accurate theories of psychological concepts, by requiring theories that make accurate predictions to allow for clear hypotheses to be tested (Vanpaemel, 2010).

Issues highlighted in respect to methodological and analytical techniques threaten the replicability and reproducibility of science, especially with the lack of specific experimental design nuances not being disclosed. In an examination of attentional bias, Pennington et al. (2021) call for the pre-registration of cognitive alcohol research to provide more robust measures and results, improving the rigor and facilitate replication attempts. All chapter of this thesis were pre-registered to allow future replication attempts, along with the disclosure of stimuli, data and analysis scripts were possible on Open Science Framework, (remaining information will be uploaded upon publication of the relevant chapters). Transparency of *a-priori* and exploratory hypotheses should allow for ease of replication (Munafò et al., 2017) and reduce bias in the literature (Nosek, Ebersole, DeHaven, & Mellor, 2018). Studies which are pre-registered and have used *a-priori* hypotheses have shown higher replicability (Swaen, Teggeler, & van Amelsvoort, 2001), increases in null findings (Kaplan & Irvin, 2015), and reduction in effect sizes (Schäfer & Schwarz, 2019). Through the use of pre-registration we can account for problematic research practices that may distort the true nature of effects (Schäfer & Schwarz, 2019).

Clinical Implications

Findings from this thesis have multiple clinical implications, mainly in respect of the use of inhibitory control as a marker of alcohol misuse and inhibitory control training. Previous research has highlighted the role of inhibitory control as a marker of initiation and relapse to alcohol addiction (Moeller et al., 2016), with its importance to alcohol addiction being increasingly salient compared to other substances (Luijten et al., 2014). A range of theoretical perspectives argue that the lack of control and increasing drive aspects are key to the initiation and continuation of substance abuse (Iacono, Malone, & McGue, 2008; Verdejo-García et al., 2008; Volkow et al., 2010). Arguments have been made for the use of

neuropsychological measures (i.e. inhibitory control) to standardise the diagnosis of substance use disorders and remove the ambiguity of an individual's diagnosis, with tracking of said markers being used as surrogate measures of treatment success and predictors of relapse (Jentsch & Pennington, 2014; Marhe, Luijten, & Franken, 2014; Perlis, 2011).

Although the potential clinical uses of inhibitory control as a marker for substance use has been raised, concerns around the rigour of such measures has been raised by Franken and van de Wetering (2015). Franken and van de Wetering discuss how the reliability of the measures to assess given behavioural constructs are consistently questionable, and by extension so is their predictive validity. A more fundamental issue is that of causality, with many measures of inhibitory control being assessed in experimental conditions void of confounding factors. Research presented in this thesis attempted to examine inhibitory control in complex environments and found little robust association with alcohol use questioning its validity as a predictive marker. Recent work has proposed inhibitory control should be examined alongside other cognitive constructs to provide a more comprehensive representation of cognitive performance (Yücel et al., 2019). In chapter six attempts were made to examine inhibitory control alongside working memory to account for alcohol use with little evidence provided. A potential explanation for inconsistencies across the field is the difference in measures of inhibitory control used, such as stop-signal tasks being a more complex measure compared to a go/no-go task, highlighting a need for standardised measures.

Inhibitory control training involves reinforcing the capacity for an individual to override an impulsive action and influence how specific stimuli are assessed (Aron et al., 2014; De Pretto, Hartmann, Garcia-Burgos, Sallard, & Spierer, 2019). One such stimuli that

has been the subject for specific inhibitory control training interventions is alcohol, due to impairments of reactive control increasing the likelihood of increased alcohol consumption (Jones et al., 2020). In laboratory based experiments numerous studies have shown a small-to medium effect (Allom et al., 2016; Jones et al., 2016) of inhibitory control training at reducing alcohol consumption in comparison to control groups (Di Lemma & Field, 2017; Houben, Havermans, Nederkoorn, & Jansen, 2012). However, the effect of inhibitory control training is questionable given the effect of publication bias driven by small sample sizes inflating effect sizes (Adams, Lawrence, Verbruggen, & Chambers, 2017; Schonberg et al., 2014), and not surviving a context shift to an alcohol-related environment with Bayesian analysis suggesting it has no effect (Jones et al., 2020).

A potential explanation for this lack of effect is due to the use of rewarding stimuli reducing the intrinsic value of inhibition, with recent work suggesting manipulation of said valuation may facilitate inhibitory training (De Pretto et al., 2019). As discussed in chapter one the use of extrinsic rewards provides a means to facilitate inhibitory control, particularly in inhibitory control training, by increasing the intrinsic value of inhibiting. Yet there are issues, the reward must be salient to the individual, or more salient than the stimuli required to inhibit to, to provide a meaningful change. In the research presented the presence of money as a reward exhibited no consistent effect on inhibitory control, but their salience compared to stimuli presented was not assessed. Such research may provide a means to improve the efficacy of inhibitory control training following further refinement of the paradigm and the role of reward in inhibitory mechanisms.

Limitations & future research

The work presented in this thesis does have methodological limitations that potentially may have impacted on findings. Firstly, samples used consisted of participants recruited via opportunity sampling. In chapter four the majority of participants were heavy drinking undergraduate students, whereas chapter five contained a mix of undergraduates and the general population, and chapter six consisted of those in sixth form colleges, making comparison between all chapters somewhat difficult. However, I aimed to recruit individuals, in chapters four and five who were motivated to reduce their drinking. Indeed, *'a desire to reduce your alcohol consumption'* was a part of the inclusion criteria for these studies. These individuals were recruited because if they there was no motivation to inhibit their behaviour towards alcohol or related cues the generalisability of the findings would be compromised. Given that the dual process model suggests that students and adolescents have little motivation to reduce their consumption, due to normative behaviours (Faulkner, Hendry, Roderique, & Thomson, 2006) and social environment (Littlefield, Sher, & Wood, 2009), they present as high risk for escalating issues surrounding alcohol. The majority of students remain heavy drinkers with little motivation to reduce their drinking (Field et al., 2020), tending to remain the case until they leave university and 'mature out' (Littlefield, Sher, & Wood, 2010), yet I did attempt to measure "concerns about drinking" using the TRI (see Appendices 1.D). It remains plausible that samples used in this thesis had little motivation to reduce their drinking and heterogeneity between samples, in turn contributing to the lack of associations between inhibitory control measures and alcohol use. Future research may rectify such issues by targeting specific populations, e.g. those that have left university or have not yet attended as in chapter four, to examine how inhibitory control relates to alcohol use if at all.

In an extension of the caveats mentioned surrounding recruitment, future research should endeavour to examine both light drinkers and individuals diagnosed with alcohol use disorders. It would have been useful to examine the difference in alcohol cue exposure and the reward between groups. In respect to alcohol cues, contradictory results between heavy and light drinkers have been observed for inhibitory control in general (Czapla et al., 2016; Nederkoorn et al., 2009), along with more finite differences in reactive and proactive control (Sharma, 2017). Use of individuals diagnosed with alcohol use disorder may allow us to better understand the relationship between inhibitory control, environment (both alcohol and reward stimuli) and alcohol use, given that these individuals show a greater degree of inhibitory impairment (Smith et al., 2014). To the best of my knowledge, there is no research that examines the differential effects of reward between and drinker status, which is particularly important given the difference in incentive salience attributed to alcohol stimuli meaning extrinsic rewards may be more or less effective in different drinking groups (Bujarski et al., 2018; Ihssen, Cox, Wiggett, Fadardi, & Linden, 2010). Understanding what rewards may particularly interrupt the effect of alcohol on inhibitory control may increase the efficacy of behavioural interventions in clinical environments.

Finally, the majority of the research presented in this thesis used the Stop-Signal task (Logan et al., 1984b), limiting the generalisability to other inhibition measures, however recent evidence questions the validity of stop-signal task as a measure of executive functioning. Particularly in respect to the indices of inhibitory control used, recent research has shown that a previously apparent consistent effect of reward on inhibitory control was abolished following the removal of errors in the SSRT calculation (Doekemeijer et al., 2021). I removed said errors during my calculations for SSRTs, yet previous literature may not have

meaning that there may be a significant amount of publication bias in the literature potentially compromising the power and sample size of future and the current research. Further to this if such issues exist for the effect of reward it may extend to other factors, such as cue reactivity, highlighting the need for pre-registered and open science to clarify the effects. In addition the ability to compare findings across inhibitory control tasks is questionable, given their poor associations (Von Gunten, Bartholow, & Martins, 2019) and correlations (Gärtner & Strobel, 2021). This challenges the commonly held belief that inhibition tasks are valid and reliable measures of inhibition, potentially impacting findings.

Conclusion

In conclusion, the results of this thesis demonstrated that heavy drinker's inhibitory control does not fluctuate consistently in response to reward or alcohol cues. In adult heavy drinkers, reward appeared to improve proactive control in one study, while in adolescents it appears to impair reactive control, suggesting reward effects different ages differently. I failed to replicate the apparent robust effect of reward found in chapter four, suggesting the effect isn't consistent in heavy drinkers or other mechanisms (e.g. attention) should be examined. Similarly, there was a failure to replicate an apparently robust effect of alcohol-cue exposure impairment, or to appetitive stimuli in general, on inhibitory control or an association with alcohol use. The lack of evidence supporting the notion that inhibitory control is related to alcohol use indicates the relationship is overemphasized. The findings drawn together make it difficult to theorise the role and mechanism that reward and environmental cues effect inhibitory control and in turn alcohol use. Findings refute theories that suggest inhibitory control is a crucial mechanism in the initiation and maintenance of substance addiction, impacting the clinical use of the current findings and future findings that suggest inhibitory control is a state variable.

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Appendices

Appendices 1 Questionnaires.

Appendices 1.A Timeline Follow Back (TLFB)

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

Appendices 1.B: Alcohol Use Disorders Identification Test (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

Appendices 1.C Barratt Impulsivity Scale

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

Appendices 1.D Temptation and Restraint Inventory

Instructions: Please read each of the following questions carefully. Circle the number that represents your answer to each question. BE SURE TO CIRCLE ONLY ONE NUMBER FOR EACH QUESTION. Remember that your honest response -- the one that makes the most sense to you personally is the response we want. Don't worry about how other people would answer, we want your views. Please work as quickly as you can, while giving the most honest and accurate answer you can to each question. In general, your first impressions are the best.

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

1	2	3	4	5	6	7	8	9
Never								Always

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

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

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

stimulate concerns about the need to limit your drinking?

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

1	2	3	4	5	6	7	8	9
Never								Always

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

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

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

1	2	3	4	5	6	7	8	9
Never								Always

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

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

Appendices 1.E Chocolate use questionnaire

In an average week, can you please indicate how much chocolate (roughly, in standard sized bars, e.g. Mars, Snickers, Twix, etc) you would consume?

_____

How many bars of chocolate do you usually keep at home?

_____

Please give an indication of how you feel about the following statements;

How much do you like to eat chocolate?

0	1	2	3	4	5	6	7
Not a lot							Lots

How often do you feel the urge to eat chocolate?

0	1	2	3	4	5	6	7
Never							Always

How strongly do you feel this urge?

0	1	2	3	4	5	6	7
Not very strongly							Very strongly

To what extent do you feel you need to eat chocolate?

0	1	2	3	4	5	6	7
Not at all							A large extent

How difficult do you find it to stop eating chocolate once you have started?

0	1	2	3	4	5	6	7
Not at all							Extremely

Appendices 1.F Self-control scale

		Not at all				Very much
1.	I am good at resisting temptation.	1	2	3	4	5
2.	I have a hard time breaking bad habits.	1	2	3	4	5
3.	I am lazy.	1	2	3	4	5
4.	I say inappropriate things.	1	2	3	4	5
5.	I never allow myself to lose control.	1	2	3	4	5
6.	I do certain things that are bad for me, if they are fun.	1	2	3	4	5
7.	People can count on me to keep on schedule	1	2	3	4	5
8.	Getting up in the morning is hard for me	1	2	3	4	5
9.	I have trouble saying no.	1	2	3	4	5
10.	I change my mind fairly often.	1	2	3	4	5
11.	I blurt out whatever is on my mind.	1	2	3	4	5
12.	People would describe me as impulsive.	1	2	3	4	5
13.	I refuse things that are bad for me.	1	2	3	4	5
14.	I spend too much money.	1	2	3	4	5
15.	I keep everything neat.	1	2	3	4	5
16.	I am self-indulgent at times.	1	2	3	4	5
17.	I wish I had more self-discipline.	1	2	3	4	5
18.	I am reliable.	1	2	3	4	5
19.	I get carried away by my feelings.	1	2	3	4	5
20.	I do many things on the spur of the moment.	1	2	3	4	5
21.	I don’t keep secrets very well.	1	2	3	4	5
22.	People would say that I have iron self-discipline.	1	2	3	4	5
23.	I have worked all studied all night at the last minute.	1	2	3	4	5
24.	I’m not easily discouraged.	1	2	3	4	5
25.	I’d be better off if I stopped to think before acting.	1	2	3	4	5
26.	I engaged in healthy practices.	1	2	3	4	5
27.	I eat healthy foods.	1	2	3	4	5
28.	Pleasure and fun sometimes keep me from getting work done.	1	2	3	4	5
29.	I have trouble concentrating.	1	2	3	4	5
30.	I am able to work effectively toward long-term goals.	1	2	3	4	5

31.	Sometimes I can't stop myself from doing something, even if I know it is wrong.	1	2	3	4	5
32.	I often act without thinking through all the alternatives.	1	2	3	4	5
33.	I lose my tempter too easily.	1	2	3	4	5
34..	I often interrupt people/	1	2	3	4	5
35.	I sometimes drink or use drugs to excess.	1	2	3	4	5
36.	I am always on time.	1	2	3	4	5

Appendices 1.G Brief Desire for Alcohol Questionnaire

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

Appendices 1.H Family affluence scale

Please circle, the appropriate response for each question.

1. **Does your family own a car or another motorized vehicle?**

   No Yes, one Yes, two

2. **Do you have your own bedroom?**

   No Yes

3. **How many computers (including laptops and tablets, not including game consoles and smartphones) does your family own?**

   None One More than two

4. **How many bathrooms (room with a bath/shower or both) are there in your home?**

   None One Two More than two

5. **Does your family have a dishwasher?**

   No Yes

6. **How many times did you and your family travel out of the UK for holiday/vacation last year?**

   Never Once Twice More than twice

Appendices 1.I Adolescent Alcohol Involvement Scale

The questions below also refer to your use of alcohol. Circle all the answers which describe your use of alcohol. Even if none of the answers seem exactly right, please pick the ones that come closest to being true. If a question doesn't apply to you, you may leave it blank.

1. How often do you drink alcohol?
a. never.
b. once or twice a year.
c. once or twice a month.
d. every weekend.
e. several times a week.
f. every day.
g. several times a day.

2. When did you last drink alcohol?
a. never used alcohol.
b. not for over a year.
c. between 6 months and 1 year ago.
d. several weeks ago.
e. last week.
f. Yesterday.
g. Today.

3. I usually start to drink because: (CIRCLE ALL THAT APPLY)
a. I like the feeling.
b. to be like my friends.
c. I am bored; or just to have fun.
d. I feel stressed, nervous, tense, full of worries or problems.
e. I feel sad, lonely, sorry for myself.

4. What do you drink, when you drink alcohol?
a. wine.
b. beer.

c. mixed drinks.

d. spirits (vodka, whisky, etc.).

e. a substitute for alcohol.

5. How do you get your alcohol or drugs? (CIRCLE ALL THAT YOU DO)

a. Supervised by parents or relatives.

b. from brothers or sisters.

c. from home without parents' knowledge.

d. get from friends.

e. buy my own (on the street or with false ID)

6. When did you first have an alcoholic drink? (CIRCLE ONE)

a. never.

b. after age 15.

c. at ages 14 or 15.

d. at ages 12 or 13.

e. at ages 10 or 11.

f. before age 10.

7. What time of day do you use alcohol? (CIRCLE ALL THAT APPLY TO YOU)

a. at night.

b. afternoons/after school.

c. before or during school.

d. in the morning or when I first awaken.

e. I often get up during my sleep to use alcohol.

8. Why did you first drink alcohol? (CIRCLE ALL THAT APPLY)

a. curiosity

b. parents or relatives offered

c. friends encouraged me; to have fun

d. to get away from my problems

e. to get drunk

9. When you drink alcohol, how much do you usually drink?

a. 1 drink

b. 2 drinks

c. 3-4 drinks

d. 5 -9 drinks

e. 10 or more drinks

10. Whom do you drink with? (CIRCLE ALL THAT ARE TRUE OF YOU)

a. parents or adult relatives

b. with brothers or sisters

c. with friends or relatives own age

d. with older friends

e. alone

11. What effects have you had from drinking? (CIRCLE ALL THAT APPLY TO YOU)

a. loose, easy feeling

b. got a little drunk

c. got drunk

d. became ill

e. passed out

f. used a lot and next day didn't remember what happened.

12. What effects has using alcohol had on your life? (CIRCLE ALL THAT APPLY)

a. none.

b. has interfered with talking to someone.

c. has prevented me from having a good time.

d. has interfered with my school work

e. have lost friends because of drinking.

f. has gotten me into trouble at home.

g. was in a fight or destroyed property.

h. has resulted in an accident, an injury, arrest, or being punished at school for using alcohol.

13. How do you feel about your use of alcohol? (CIRCLE ALL THAT APPLY)

a. no problem at all.

b. I can control it and set limits on myself.

c. I can control myself, but my friends easily influence me.

d. I often feel bad about my drinking.

e. I need help to control myself.

f. I have had professional help to control my drinking.

14. How do others see you in relation to your alcohol? (CIRCLE ALL THAT APPLY)

a. can’t say or normal for my age.

b. when I use I tend to neglect help my family or friends.

c. my family or friends advise me to control or cut down on my use

d. my family or friends tell me to get help for my alcohol use

e. my family or friends have already gone for help about my drinking.

Appendices 1.J Funnelled debrief

1. What was the purpose of this experiment?

2 . The computer task was designed to…….

Measure reaction times in response to the target stimuli [ ]

Assess my cognitive processing [ ]

Train me to think more quickly [ ]

Measure reaction times to correlate with alcohol use [ ]

Assess my behavioural impulsivity (response inhibition) [ ]

I do not know the purpose [ ]

3. The purpose of this taste test was to…….

Measure my liking for each drink [ ]

Measure my preference for each drink [ ]

Measure my preferences to each drink in response to the computer task [ ]

Measure how much I drank in response to the computer task [ ]

Find out which drink I preferred [ ]

Measure whether I would drink less/more beer in response to my answers on the questionnaire [ ]

I do not know the purpose [ ]

Appendices 2 Meta-analysis tables of included studies

Study	N	Mean Age	Females %	Heavy drinkers	Clinical sample	Diagnosis	Substance or food	Design	DV	Reward type	Hypothetical	Results
Go/No-Go task												
Asci et al. (2019)	24	23.42	66.67	NA	NA	NA	NA	Within	No-go errors	Money	No	R>C (reduced no-go errors in reward condition compared to control)
Charles-Walsh (2016)	68	37.96	29.41	NA	Yes	Opioid dependent	NA	Within	Accuracy rate	Money	No	R=C (no significant difference in accuracy rates)
Demurie et al. (2016)	127	11.31	22.11	NA	Yes	ADHD, ASD	NA	Within	Commission errors	Money	NA	R=C (no significant differences between conditions)
Desman et al. (2008)	38	10.27	0	NA	Yes	ADHD	NA	Within	Commission errors	Money	NA	R>C (reduced no-go errors in reward condition compared to control)
Epstein et al. (2011a)	13	8.11	26.89	NA	Yes	ADHD	NA	Within	Accuracy rate	Money	No	Not reported
Epstein et al. (2011b)	93	8.11	25.51	NA	Yes	ADHD	NA	Within	Accuracy rate	Points	Yes	R>C (significantly improved accuracy rate in reward compared to control condition)
Groom et al. (2010)	56	12.53	3.57	NA	Yes	ADHD	NA	Within	Commission errors rate	Points	NA	NA
Kohls et al. (2009)	48	10.44	22.34	NA	Yes	ADHD, TBI	NA	Between	False alarm rate	Money, social, mixed	No	R>C (significantly reduced false alarm rates in the reward compared to control condition)

Le et al. (2020a)	49	23	50	NA	NA	NA	NA	Within	Accuracy rate	Money	Yes	R<C (accuracy rate was significantly lower in the reward condition compared to control)
Le et al. (2020b)	35	NA	53.06	NA	NA	NA	NA	Within	Accuracy rate	Money	Yes	R<C (accuracy rate was significantly lower in the reward condition compared to control)
Luijten et al. (2013)	36	25.68	63.69	NA	NA	NA	NA	Within	Accuracy rate	Money	NA	R=C (no significant difference for accuracy rates between conditions)
Lyndon et al. (2015)	18	31.06	27.78	NA	NA	NA	Smokers	Within	Accuracy rate	Money	No	R>C (Significantly greater accuracy rate in smoking as usual group)
Michalowski et al. (2017)	64	22.18	78.12	NA	NA	NA	NA	Within	Commission error rate	Money	NA	R=C (no significant difference between conditions)
Miyasaka et al. (2019)	40	11.55	0	NA	Yes	ADHD	NA	Within	Commission error rate	Points	No	R>C (significantly reduced commission error rate in reward compared to no reward condition)
Poulton et al. (2016)	84	22.90	84.76	Yes	NA	NA	NA	Within	Accuracy rate	Points	NA	R>C (significantly improved accuracy in the reward compared to control condition)
Rossiter et al. (2012)	85	26.20	55.04	Yes	NA	NA	NA	Within	Accuracy rate	Money	No	R>C (significantly increased accuracy rates in the reward

												compared to control condition)
Schevernals et al. (2016)	21	25.20	80.95	NA	NA	NA	NA	Within	Accuracy rate	Money	No	R=C (no significant difference in accuracy rate across conditions)
Schutte et al. (2019)	49	23.90	65.56	NA	NA	NA	NA	Within	Commission error rate	Money	No	R>C (Commission error rates were significantly lower in the reward compared to the no reward condition)
Winter et al. (2014) *Study 1*	77	8.20	65.00	NA	NA	NA	NA	Within	Commission error rate	Toy	Yes	R>C (significantly reduced error rates in the reward compared to control condition)
Winter et al. (2014) *Study 2*	60	7.36	58.33	NA	NA	NA	NA	Within	Commission error rate	Toy	Yes	R>C (significantly reduced error rates in the reward compared to control condition)
Stop-Signal task												
Boehler et al. (2012)	18	22	83.33	NA	NA	NA	NA	Within	SSRT	Money	No	R>C (shorter SSRTs in reward condition compared to control)
Boehler et al. (2014)	16	22.80	93.75	NA	NA	NA	NA	Within	SSRT	Money	No	R>C (shorter SSRTs in reward condition compared to control)
Byrne & Worthy (2019)	98	19.31	63.27	Mild substance use	NA	NA	NA	Within	SSRT	Money	Yes	R=C (No significant difference in SSRTs)
Chikara et al. (2018)	20	23.30	10	NA	NA	NA	NA	Within	SSRT	Money	NA	R=C (No significant difference in SSRTs)

Epstein et al. (2011a)	13	8.11	26.89	NA	Yes	ADHD	NA	Within	SSRT	Money	NA	R=C (SSRTs were not significantly shorter for reward compared to control)
Epstein et al. (2011b)	93	8.11	25.51	NA	Yes	ADHD	NA	Within	SSRT	Points	Yes	R=C (SSRTs were not significantly shorter for reward compared to control)
Fosco et al. (2015)	59	10.85	15.52	NA	Yes	ADHD	NA	Within	SSRT	Money	No	NA
Herrera et al. (2014)	21	31.00	50	NA	NA	NA	NA	Within	SSRT	Money	NA	R=C (SSRTs were not significantly shorter for reward compared to control)
Houben et al. (2014)	35	20.97	100	NA	NA	Restrained eaters	NA	Within	SSRT	Money	No	R=C (SSRTs were not significantly shorter for reward compared to control)
Konrad et al. (2000)	47	10.44	22.34	NA	Yes	ADHD, TBI	NA	Within	SSRT	Points	NA	R=C (SSRTs were not significantly shorter for reward compared to control)
Marx et al. (2013)	78	26.73	47.43	NA	Yes	ADHD	NA	Between	SSRT	Points	No	NA
Padmala et al. (2010)	35	22.00	54.29	NA	NA	NA	NA	Within	SSRT	Money	No	R<C (SSRTs were significantly longer in the reward condition compared to control)
Scheres et al. (2001)	113	10.48	25.22	NA	Yes	ADHD, ODD	NA	Within	SSRT	Money	No	R>C (SSRTs were significantly shorter in the reward condition compared to control)

Schevernals et al. (2015)	20	25.20	80.95	NA	NA	NA	NA	Within	SSRT	Points	No	R=C (SSRTs did not significantly differ between conditions)
Shanahan et al. (2008)	55	11.10	38.18	NA	Yes	ADHD	NA	Within	SSRT	Money	Yes	R=C (SSRTs did not significantly differ between conditions)
Sinopoli et al. (2011)	112	12.44	47.73	NA	Yes	ADHD, TBI	NA	Within	SSRT	Points	NA	R>C (SSRTs were significantly shorter in the reward compared to control condition)
Flanker task												
Bradley et al. (2017)	16	16.30	45.45	NA	Yes	Depression/anxiety	NA	Within	RT	Money	No	R=C (no significant difference in RT between conditions)
Chiew & Braver (2016) *Study 1*	24	19.50	54.17	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition

												were significantly lower than the control condition)
Chiew & Braver (2016) *Study 2*	24	20.30	37.50	NA	NA	NA	NA	Within	RT	Money	No	R=C (no significant difference in RT between conditions)
Hsieh et al. (2010)	24	20.60	50.00	NA	NA	NA	NA	Between	RT	Money	NA	R=C (no significant difference in RT between conditions)
Marini et al. (2015)	16	22.70	24.24	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Mine et al. (2015) *Study 1*	26	37.77	20.20	NA	NA	NA	NA	Within	RT	Money	NA	R=C (no significant difference in RT between conditions)

Mine et al. (2015) *Study 2*	24	19.40	58.33	NA	NA	NA	NA	Within	RT	Money	NA	R=C (no significant difference in RT between conditions)
Mine et al. (2015) *Study 3*	24	21.10	25.00	NA	NA	NA	NA	Within	RT	Money	NA	R>C (RTs in the reward condition were significantly lower than the control condition)
Mine et al. (2015) *Study 4*	18	20.70	38.89	NA	NA	NA	NA	Within	RT	Money	NA	R=C (no significant difference in RT between conditions)
Paschke et al. (2015)	125	25.49	51.20	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Rosch et al. (2013)	55	11.35	21.82	NA	Yes	ADHD	NA	Within	RT	Points	No	NA

Schlienz et al. (2013)	25	40.00	48.00	NA	NA	NA	Smokers	Within	RT	Money	NA	NA
Williams et al. (2018)	55	47.00	64.58	NA	NA	NA	NA	Within	RT	Money	Yes	R>C (RTs in the reward condition were significantly lower than the control condition)
Yamaguchi et al. (2019) *Study 1*	48	20.44	66.67	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Yamaguchi et al. (2019) *Study 2*	48	20.44	72.92	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Yamaguchi et al. (2019) *Study 3*	48	20.98	60.42	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward

												condition were significantly lower than the control condition)
Simon task												
Bundt et al. (2016)	20	22.60	80.00	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Herz et al. (2014)	14	23.00	50.00	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Maigaard et al. (2019)	104	9.67	23.07	NA	NA	NA	NA	Within	RT	Money	No	R=C (no significant difference in RT between conditions)
Stroop task												

Carsten et al. (2019) *Study 1*	46	18.54	82.61	NA	NA	NA	NA	Within	RT	Money	Yes	R>C (RTs in the reward condition were significantly lower than the control condition)
Carsten et al. (2019) *Study 2*	45	19.07	75.56	NA	NA	NA	NA	Within	RT	Money	Yes	R>C (RTs in the reward condition were significantly lower than the control condition)
Carsten et al. (2019) *Study 3*	46	18.61	89.13	NA	NA	NA	NA	Within	RT	Money	Yes	R>C (RTs in the reward condition were significantly lower than the control condition)
Huguet et al. (2004)	80	NA	100.00	NA	NA	NA	NA	Between	RT	Money	NA	R=C (no significant difference in RT

												between conditions)
Krebs et al. (2013)	14	22.60	71.43	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Krebs et al. (2011)	19	22.60	52.63	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Krebs et al. (2010) *Study 1*	20	22.50	70.00	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Krebs et al. (2010) *Study 2*	16	22.65	56.25	NA	NA	NA	NA	Within	RT	Money	No	R>C (RTs in the reward

												condition were significantly lower than the control condition)
Ma et al. (2016)	58	15.33	29.31	NA	Yes	ADHD	NA	Within	RT	Money	No	R=C (no significant difference in RT between conditions)
Rossell-Negre et al. (2016)	71	36.77	14.01	NA	Yes	Cocaine dependent	Cocaine	Within	RT	Money	No	R>C (RTs in the reward condition were significantly lower than the control condition)
Antisaccade task												
Chung et al. (2011)	24	16.95	50.00	NA	Yes	SUD	NA	Within	Error rate	Money	NA	R>C (Reduced error rate in reward condition compared to control)
Duka et al. (1997)	24	29.30	NA	NA	NA	NA	NA	Within	Accuracy rate	Money	NA	>C (Increased accuracy rate in

												reward condition compared to control)
Geier et al. (2012)	106	17.98	53.77	NA	NA	NA	NA	Within	Error rate	Points	No	>C (significantly reduced error rates in the reward compared to control condition)
Geier et al. (2014)	34	35.15	50.00	NA	NA	NA	Smokers	Within	Error rate	Money	No	R>C (significantly reduced error rates in the reward compared to control condition)
Geier et al. (2010)	34	18.50	52.94	NA	NA	NA	NA	Within	Accuracy rate	Money	NA	R>C (significantly increased accuracy rates in the reward compared to control condition)
Hardin et al. (2009)	50	12.93	50.00	NA	Yes	Anxiety disorder	NA	Within	Accuracy rate	Money	NA	R>C (significant

												ly increased accuracy rates in the reward compared to control condition)
Hardin et al. (2007)	77	17.67	48.05	NA	Yes	Anxiety disorder, depression	NA	Within	Accuracy rate	Money	NA	R>C (significantly increased accuracy rates in the reward compared to control condition)
Harsay et al. (2010)	56	51.73	48.05	NA	Yes	Parkinsons	NA	Within	Accuracy rate	Money	No	R>C (significantly increased accuracy rates in the reward compared to control condition)
Mueller et al. (2013)	63	15.95	47.62	NA	Yes	CAH	NA	Within	Error rate	Money	No	R>C (significantly reduced error rates in the

												reward compared to control condition)
Mueller et al. (2012)	46	11.20	56.52	NA	Yes	Adopted children, with a history of neglect	NA	Within	Error rate	Money	No	NA
Mueller et al. (2010)	43	13.85	46.51	NA	Yes	Paediatrics with bipolar	NA	Within	Accuracy rate	Money	No	NA
Padmanabhan et al. (2011)	30	20.60	60.00	NA	NA	NA	NA	Within	Error rate	Money	No	R>C (significantly reduced error rates in the reward compared to control condition)
Preciado et al. (2018)	31	23.96	77.42	NA	NA	NA	NA	Within	Error rate	Money	No	R=C (no significant effect of reward on error rates)
Reyes et al. (2015)	103	15.60	39.81	NA	NA	Obese & Healthy	NA	Within	Error rate	Money	NA	R>C (significantly reduced error rates in the reward compared to control condition)
Ross et al. (2011)	16	21.80	31.70	NA	NA	NA	NA	Within	Error rate	Money	No	R>C (significant

												ly reduced error rates in the reward compared to control condition)
Tervo-clemmens et al. (2017)	116	12.44	47.43	NA	Yes	Increased risk of SUD/ early onset SUD	NA	Within	Accuracy rate	Money	Yes	R>C (significantly increased accuracy rates in the reward compared to control condition)

R, Reward; C, Control; SSRT, Stop Signal Reaction Time; SUD, Substance Use Disorder; ADHD, Attention Deficit Hyperactivity Disorder; ASD, Autistic Spectrum Disorder; TBI, Traumatic Brain Injury; CAH, Congenital Adrenal Hyperplasia; ODD, Oppositional Defiant Disorder; NA, Not Reported

Appendices 3 Supplementary data for chapter four

Experiment 1: Alcohol choice

There was a significant association between gender and alcohol choice, $\chi^2(4)=24.17$, $p<.001$. Males were 1.71 times more likely to choose beer over females. Females were more likely to choose cider (1.31), red (9), white (11) and rose (8.33) wine than males.

Table 6 Alcohol choice by gender

	Male	Female	Total
Cider	13	17	30
Beer	12	7	19
White Wine	1	9	10
Red Wine	0	11	11
Rose Wine	3	25	28

*Table 7 Means ±SD of questionnaires, for condition and overall. Italicised measures are subscales, * are second order factors.*

Measure	Semi-Naturalistic Environment (Mean ±SD) N=49	Standard (Mean ±SD) N=49	Total (Mean ±SD) N=98
AUDIT	14.43 ± 4.63	13.33 ± 4.91	13.88 ± 4.78
AUDIT Consumption	7.43 ±1.57	7.51 ±1.54	7.50±1.55
AUDIT Dependence	2.12 ±1.75	2.18 ±2.10	2.15 ±1.92
AUDIT Alcohol Related Problem	4.88 ±2.84	3.36 ±2.38	4.25 ±2.68
Chocolate Stored	3.37 ±7.08	2.96 ±2.96	3.16 ±5.40
Chocolate Consumed	2.71 ±1.54	3.31 ±2.05	3.01 ±1.83
Index of Chocolate Use	16.59 ±6.35	18.12 ±6.12	17.36 ±6.25
BIS	72.69 ±7.79	72.55 ±7.40	72.62 ±7.56
BIS Attention	11.53 ±1.84	11.67 ±2.08	11.60 ±1.95
BIS Cognitive Instability	7.61 ±1.48	7.65 ±1.47	7.63 ±1.47
BIS Motor Impulsivity	17.79 ±2.66	17.65 ±2.62	17.72 ±2.62
BIS Perseverance	37.45 ±1.42	37.96 ±1.51	37.70 ±1.48
BIS Self Control	13.43 ±2.19	12.76 ±2.56	13.09 ±2.39

BIS Cognitive Complexity	12.02 ±2.50	12.08 ±2.53	12.05 ±2.51
*BIS Attentional**	19.14 ±2.89	19.33 ±2.89	19.23 ±2.78
*BIS Motor**	55.27 ±3.47	55.61 ±3.38	55.44 ±3.41
*BIS Non-Planning**	25.45 ±3.13	24.84 ±3.74	25.14 ±3.44
SCS	37.43 ±7.95	35.20 ±6.72	36.32 ±7.41
SCS Self-Discipline	25.49 ±5.75	24.35 ±4.68	24.91 ±5.25
SCS Impulse Control	11.94 ±2.82	10.86 ±2.74	11.40 ±2.82
TRI	48.37 ±18.93	45.69 ±22.21	47.03 ±20.57
TRI Govern	10.41 ±5.32	9.06 ±5.26	9.73 ±5.31
TRI Restrict	11.02 ±5.49	10.01 ±5.07	10.56 ±5.28
TRI Emotion	12.34 ±6.07	12.02 ±6.90	12.18 ±6.46
*TRI Concern About Drinking**	8.04 ±4.12	8.08 ±5.16	8.06 ±4.64
*TRI Cognitive Preoccupation**	6.55 ±4.27	6.43 ±4.90	6.49 ±4.57
TLFB	49.51 ±24.26	51.69 ±26.43	50.60 ±25.61
Binge Drinking Frequency	3.80 ±1.85	3.82 ±2.19	3.81 ±2.01

Experiment 2: Alcohol choice

Females tended to prefer rose wine, males alcohol of choice was beer (see Table 1). There was a significant association between gender and alcohol choice, $\chi^2(3)=13.82$, $p<.001$. Males were 1.5 times more likely to choose beer compared to females. Females were more likely to choose cider (2.6), rose (16) or white (2.75) wine than males.

Table 8 Alcohol choice by gender

	Male	Female	Total
Cider	5	13	18
Beer	9	6	15
White Wine	4	11	15
Rose Wine	0	16	16

*Table 9 Means ±SD of questionnaires, for condition and overall. Italicised measures are subscales, * are second order factors.*

Measure	Total (Mean ±SD) N=64
AUDIT	12.84 ±5.20
AUDIT Consumption	7.18 ±1.49
AUDIT Dependence	1.63 ±1.44
AUDIT Alcohol Related Problem	4.05 ±3.28
Chocolate Stored	4.56 ±6.75

Chocolate Consumed	3.86 ±2.93
Index of Chocolate Use	16.61 ±7.46
BIS	100.82 ±7.68
BIS Attention	12.02 ±1.92
BIS Cognitive Instability	7.86 ±1.40
BIS Motor Impulsivity	17.09 ±2.60
BIS Perseverance	37.53 ±1.54
BIS Self Control	14.16 ±2.27
BIS Cognitive Complexity	12.17 ±2.57
*BIS Attentional**	19.88 ±2.59
*BIS Motor**	54.63 ±3.33
*BIS Non-Planning**	26.33 ±3.66
SCS	36.95 ±7.26
SCS Self-Discipline	25.08 ±5.29
SCS Impulse Control	11.88 ±2.97
TRI	29.33 ±13.63
TRI Govern	8.78 ±5.47
TRI Restrict	9.22 ±4.82
TRI Emotion	11.33 ±5.90
*TRI Concern About Drinking**	5.77 ±4.01
*TRI Cognitive Preoccupation**	5.89 ±4.40
TLFB	44.13 ±16.63
Binge Drinking Frequency	3.28 ±1.83