MASTER OF SCIENCE BY RESEARCH

Acute tolerance to the diminishing effects of alcohol and the influence of alcohol outcome expectancies

Eastwood, Andy

Award date:
2018

Awarding institution:
Coventry University

Acute Tolerance to the Diminishing Effects of Alcohol and the Influence of Alcohol Outcome Expectancies

by

Andy Eastwood

Submitted in fulfilment of the Degree of Masters by Research

2018

Centre for Research in Psychology, Behaviour and Achievement
Coventry University
Priory Street
Coventry, United Kingdom
CV1 5FB

Supervisors: Dr Nigel Wilson, Dr Rebecca Jenks and Dr Miguel Farias

Certificate of Ethical Approval

Applicant:

Andy Eastwood

Project Title:

Acute tolerance to the impairing effects of alcohol

This is to certify that the above named applicant has completed the Coventry University Ethical Approval process and their project has been confirmed and approved as Medium Risk

Date of approval:

16 July 2015

Project Reference Number:

P31903

Certificate of Ethical Approval

Applicant:

Andy Eastwood

Project Title:

The role of outcome expectancies and the development of acute tolerance to the impairing effects of alcohol.

This is to certify that the above named applicant has completed the Coventry University Ethical Approval process and their project has been confirmed and approved as Medium Risk

Date of approval:

27 June 2016

Project Reference Number:

P44178

ABSTRACT

Acute alcohol tolerance refers to diminished alcohol induced impairment on the descending compared to the ascending limb of the blood alcohol concentration (BAC) curve, at comparable BACs. Research has demonstrated that this rapid tolerance develops for behavioural activation, psychomotor ability and self-report feelings of intoxication. No acute recovery of inhibitory control has been found. Alcohol outcome expectancies are beliefs about the anticipated outcomes associated with alcohol consumption; positive expectations increase consumption whilst negative expectations decrease consumption. To date, the influence that these pre-existing expectancies have on the development of acute alcohol tolerance remains unexplored. Using an adapted version of the BAC curve experimental procedure, the present thesis initially aimed to identify whether past literature demonstrating the development of acute alcohol tolerance is replicable. The primary objective was to then investigate whether alcohol outcome expectancies predict the magnitude of acute tolerance development. It was anticipated that the development of acute alcohol tolerance would be replicated and that outcome expectancies, particularly negative, would predict the magnitude of tolerance development due to the increased influence of compensatory mechanisms. Firstly, results indicate that acute alcohol tolerance developed for measures of psychomotor ability and subjective intoxication. However, no tolerance developed for both aspects of behavioural control (response activation and inhibitory control). This prolonged impairment of inhibitory control on the descending compared to the ascending limb may function to promote excessive consumption during a single drinking session. Secondly, results indicate that positive and negative expectancies do not influence the development of acute alcohol tolerance. Potential explanations surrounding this are discussed and suggestions for future research are outlined.

DECLARATION OF ORIGINALITY

Candidate's Name: Andy Eastwood

Thesis Title: Acute tolerance to the diminishing effects of alcohol and the influence of alcohol outcome expectancies

Department/Faculty: Centre for Research in Psychology, Behaviour and Achievement

Student Number: 3794681

Name of Director of Studies: Dr Nigel Wilson

Name of Supervisor(s): Dr Rebecca Jenks and Dr Miguel Farias

.....

This thesis of work are the results of my own efforts. Any data or text resulting from the work of other authors are fully identified in text and in the bibliography. This thesis has not been submitted in whole or in part for any other academic degree or professional qualification. I agree that the University has the right to submit my work to the plagiarism detection service TurnitinUK for originality checks.

Signature: Some materials have been removed due to 3rd party copyright. The unabridged version can be viewed in Lancester

Date: 14/01/2018

.....

TABLE OF CONTENTS

Chapter 1: Alcohol Tolerance and Outcome Expectancies

1.1 INTRODUCTION

Alcohol is a central nervous system depressant widely known for its acute diminishing effect on behavioural control, psychomotor ability and decision-making (Fillmore, Marczinski and Bowman 2005). According to the World Health Organisation (WHO; 2014), two and a half billion people consume alcohol worldwide. Excessive consumption and problematic drinking behaviours are responsible for 3.3 million deaths every year. Alcohol misuse refers to a recurring use of the substance despite the consumer experiencing negative consequences and is one of the leading preventable causes of death. This misuse is also associated with an increased risk of avoidable injury, an increased risk of cancer development and an increased susceptibility to the development of chronic diseases, including chronic kidney disease, type-two diabetes and cardiovascular disease (Beaglehole and Bonita 2009; Rehm 2003). Despite the overwhelming negative impact alcohol misuse can have, an alarming number of individuals continue to abuse it. One specific problematic drinking pattern, known as binge-drinking, is defined as excessive consumption of alcohol during a short amount of time, followed by a period of abstinence (Crego et al. 2009). There is debate as to what defines 'excessive consumption of alcohol', but typically it equates to the consumption of more than 8 standard UK units of alcohol within a single drinking session for men and 6 for women (Health and Social Care Information Centre UK 2013; NHS Choices 2016); one standard UK unit equals one 25 ml measure of spirit (alcohol by volume (ABV) 40%), or 250ml of beer (ABV 4%) or 76ml of wine (ABV 13%) (Drinkaware 2017). Home Office (2012) statistics indicate that consumption during a binge drinking occasion accounts for half of the alcohol consumed in the UK. Research also suggest that this shift in drinking behaviour contributes to a substantial proportion of reported alcohol associated injuries and deaths (Courtney and Polich 2009; Naimi et al. 2003).

It is clear that binge drinking is becoming increasingly problematic, particularly in the UK. The subsequent sections in this chapter will focus on binge drinking prevalence within the UK specifically, and the associated economic implications of this excessive drinking behaviour. This chapter will then review the existing alcohol tolerance and outcome expectancies literature and discuss the importance of investigating the interaction between these two mechanisms in relation to binge drinking. The present thesis focuses on changes in alcohol induced impairment during the time course of a single drinking session. Specifically, it aims to investigate the complex interplay between acute alcohol

tolerance and pre-existing outcome expectancies as well as the influence these mechanisms may have on the initiation and maintenance of a binge drinking occasion.

1.2 Drinking Behaviour in the UK

1.2.1 Binge Drinking Prevalence

Drinking excessive amounts of alcohol during a single session (binge drinking) is a major public health concern, particularly amongst young adults. In the UK, 58% of individuals (aged 16 or over) reported that they had consumed alcohol in the week prior to interview; of these individuals 48% were aged 16-24 and 15% were classed as binge drinkers (Office for National Statistics 2016). Further, when these findings are split by gender, 29% of males reported drinking in excess of 8 units and 22% of women reported drinking in excess of 6 units on at least one occasion. This type of drinking occurs far more frequently in individuals who attend university/college than in young adults who do not attend and is most prominent in undergraduate students (Naimi et al. 2003; Slutske 2005). Approximately 34% of all alcohol was sold within licenced premises (i.e., bars and restaurants) in 2014 compared to 47% in 2000. There has been a shift in preference to purchase alcohol in off-trade premises to consume at home (i.e., supermarkets and licensed shops) (Sheen 2013). This is likely due to the fact that purchasing alcohol from off-trade premises is cheaper which in turn may increase the amount of alcohol consumed. As such, 'pre-drinking' has become a common feature of nights out for both younger and older drinkers (Ally et al. 2016) and this cheaper option may be an influential factor in the increase in binge drinking culture.

It is clear that there is a shift towards binge drinking within UK drinking culture. It is important to consider the social construct of this heavy episodic drinking behaviour. Typically, binge drinking is most prevalent on a Friday or Saturday evening (Taylor et al. 2010). Hughes et al. (2011) interviewed people from several European countries, including the UK, during a typical drinking occasion (outside popular bars and nightclubs) and found that 82.5% of these participants reported that they expected to drink excessively. These prior expectations may increase the likelihood of alcohol consumption and increase the probability of initiating and maintaining a binge-drinking occasion. It is generally supported that positive outcome expectancies increase the likelihood of initiating drinking (Natvigaas et al. 1998), consuming greater volumes of alcohol per drinking occasion (Fromme and D'Amico 2000), and reporting more frequent heavy drinking episodes (Greenfield, Harford and Tam 1991). It is clear that alcohol expectancies may play a functional role in drinking behaviour and this will be explored in greater depth in the subsequent sections of this chapter. As binge drinking is becoming increasingly

prevalent in the UK, it is important to consider the impact this excessive drinking behaviour has on the UK economy.

1.2.2 Economics of Binge Drinking

The increase in binge drinking prevalence, particularly on a Friday and Saturday evening, has a detrimental financial impact on the economy. It is estimated that excessive alcohol consumption within a single session costs the UK economy £4.86 billion per year, which is equivalent to £77 per year, per taxpayer (Francesconi and James 2015). This expense is associated with risk-taking behaviours such as driving whilst intoxicated, engaging in risky sexual behaviour, unintentional physical injuries, sexual crime, reduced academic performance and increased small crime convictions (Hingson, Zha and Weitzman 2009). These risk-taking behaviours have a costly impact on the national health service (NHS). Alcohol consumed during a binge drinking occasion increases the amount of minor injury daily admissions to Accident and Emergency (A & E) departments by approximately 8% (Francesconi and James 2015). This increase is equivalent to an additional 2,504 daily admissions as a result of excessive alcohol consumption; with attending A & E costs approximately £114 per individual per visit (Department of Health 2013). As well as this, binge drinking also has a negative impact on policing in the UK. This drinking behaviour alone increases the amount of alcohol-related arrests by 45%, which is equivalent to 786 additional arrests per day. Road accidents are increased by 17% (equivalent to an additional 82 road accidents a day) as a direct result of binge drinking and fatal car crashes due to intoxication increase by 50%. It has also been identified that policing levels have increased by approximately 30% as a direct result of increased problematic binge drinking. This equates to an approximate increase of 3.2 police offers on duty for every 10,000 people in the country at the weekend (Francesconi and James 2015).

It is clear that the increased prevalence of binge-drinking in the UK, coupled with the apparent risk of injury and likelihood of being involved in crime is likely to be responsible for the negative impact on the UK economy. To better understand this, it is important to consider the pharmacological action of alcohol involved in driving these maladaptive behaviours. The next section summarises how alcohol intoxication occurs by exploring the the pharmacodynamic action of the substance. It will focus specifically on the absorption and elimination rates of alcohol to outline how an individual becomes increasingly intoxicated once consumption has been initiated.

1.3 PHARMACOLOGY OF ALCOHOL

Alcohol is a psychoactive substance that contains ethyl alcohol, or ethanol and is produced naturally as a by-product of yeast fermentation (Lyons et al. 1995). The pharmacokinetics of alcohol are responsible for the change in blood alcohol concentration (BAC) following consumption over time (Parrott, Morinan and Moss 2004). When consumed, alcohol is rapidly absorbed into the blood resulting in an increase in BAC (Ramchandani, Bosron & Li 2001). The rate of this absorption is variable between individuals, but typically peak alcohol intoxication is reached approximately one-hour post ingestion. Absorption relies on several factors including the amount of time alcohol spends in the stomach. BAC increases when the rate of absorption is greater than the rate of elimination. Metabolism accounts for approximately 90% of alcohol eliminated from the body and is catalysed by the enzyme alcohol dehydrogenase. The longer that alcohol stays in the stomach the more exposure it has to alcohol dehydrogenase which results in more alcohol being metabolised and consequently reduces the rate of absorption (McKim & Hancock 2012). Alcohol absorption is quicker when the consumer has fasted, this is because the ingestion of solid foods prolongs the duration alcohol is exposed to alcohol dehydrogenase by increasing the amount of time it spends in the stomach (Watkins & Adler 1993). Fasting therefore results in an increased BAC as greater concentrations of alcohol are absorbed into the blood on an empty stomach. Gender has also been identified as a factor that influences the rate of alcohol absorption as women typically have lower amounts of alcohol dehydrogenase than men. As a result, the same dose of alcohol produces higher BAC's in female, compared to male consumers (Frezza et al. 1990). Alcohol is absorbed much faster when the drink consumed has a higher alcohol content. This is due to high alcohol concentrations increasing the rate of diffusion, compared to lower concentrations (McKim & Hancock 2012).

Alcohol is eliminated from the body by excretion and metabolism. BAC declines when the rate of elimination is greater than the rate of absorption. The metabolic process firstly involves converting alcohol to acetaldehyde (catalysed by the enzyme alcohol dehydrogenase). The rate of this relies on the amount of alcohol dehydrogenase available. The second process involves the conversion of acetaldehyde to acetyl coenzyme A (acetyl- CoA; catalysed by aldehyde dehydrogenase). Following this, a chemical reaction known as the citric acid cycle converts acetyl-CoA to water and carbon dioxide which is then excreted; during this reaction useful energy is released which is then used in several bodily functions (such as the production of steroids and fatty acids) (Li et al. 1998; Lieber 2004). In line with this, previous research has shown that the metabolism of alcohol can significantly alter the chemistry of the human body (McKim & Hancock 2012). Another less frequent metabolic process that converts alcohol to acetaldehyde is the microsomal ethanol oxidizing system (MEOS; Lieber 2004;

Lieber & DeCarli 1968). This process is catalysed by the enzyme cytochrome P450 2E1 (CYP2E1) and is typically responsible for metabolising 5-10% of alcohol present in the body following consumption. However, the MEOS system becomes increasingly active as the concentration of alcohol in the body increases. As a result, alcohol is metabolised (and eliminated) at a much faster rate in heavier drinkers due to the increased activity of the MEOS system. This may explain why heavy episodic drinkers develop chronic alcohol tolerance as large concentrations of the substance are metabolised faster by the increased activity of the MEOS system. This section has described the pharmacokinetics of alcohol (i.e., absorption and elimination) and explained how alcohol intoxication occurs. The following section addresses the influence intoxication has on behaviour and cognition. Specifically, it considers the underlying cognitive mechanisms that may be compromised by alcohol and consequently increase the likelihood of poor decision making and risk-taking behaviour whilst intoxicated.

1.4 Acute Effects of Alcohol on Cognition

It has been suggested that alcohol induced psychomotor and behavioural control impairment are implicated in the maintenance of a binge drinking occasion (Fillmore, Marczinski and Bowman 2005; Ostling and Fillmore 2010; Reynolds, Richards and de Wit 2006; Weafer and Fillmore 2012). Research has demonstrated that acute alcohol consumption impairs psychomotor ability; this refers to the coordination of a motor activity (e.g. movement) and cognition (Tiplady et al. 2001). Psychomotor ability is primarily measured in terms of speed and accuracy of performance on motor activity tasks. Typically, in sober individuals, psychomotor tasks are completed with either greater speed but less accuracy or with less speed and greater accuracy (Wickelgren 1977). Research has investigated the influence alcohol has on psychomotor speed and accuracy using several motor ability tracking tasks. It has identified that psychomotor speed (time taken to complete the task) is not significantly impaired by alcohol whereas accuracy (amount of errors made during the task) is significantly impaired (Tiplady et al. 2004). This suggests that speed of response remains constant when comparing sober and intoxicated individuals but more errors are made as BAC increases. In addition, Brumback, Cao and King (2007) explored whether this differed between heavy social drinkers and light social drinkers. In this study, heavy social drinkers were defined using a combination of consumption frequency and quantity including the frequent occurrence of weekly binge-drinking. Light social drinkers were defined as infrequent binge-drinkers that consume 6 drinks or less per week. Both heavy and light social drinkers demonstrated psychomotor performance impairment following a 0.8g of alcohol/kg of body mass dose of alcohol despite heavy social drinkers reporting feeling less intoxicated. They discussed that this apparent alcohol induced impairment of psychomotor ability in

heavy drinkers paired with the belief that they are less intoxicated may increase the likelihood of these individuals engaging in dangerous activities, such as drink driving. It is clear from this that acute alcohol impairment of psychomotor ability can lead to negative consequences.

Similarly, the impairing effects of alcohol have been explored on aspects of behavioural control (response activation and inhibition). Response activation is defined as the commencement of a behaviour when presented with a stimuli and response inhibition refers to an individual's ability to inhibit an already initiated behaviour (Logan and Cowan 1984). These specific behavioural mechanisms determine when and where a particular behaviour is expressed (Miller and Fillmore 2014). In a practical sense, this ability to inhibit behavioural responses is vital when an individual must demonstrate self-control. As such, an impairment of inhibitory control may result in individuals engaging in riskier and more impulsive behaviours (Weafer and Fillmore 2012). Inhibitory control and behavioural activation impairment are associated with acute alcohol consumption and are significant predictors of negative consequences (Marczinski et al. 2005). Laboratory studies outline a diminishing effect on both aspects of behavioural control (Fillmore, Marczinski and Bowman 2005; Ostling and Fillmore 2010; Reynolds, Richards and de Wit 2006; Weafer and Fillmore 2012). These studies have shown this effect using the cued go/no-go task that measures the speed at which an individual responds to a 'go signal' and the accuracy of inhibiting a response to an already instigated 'no-go signal' (Logan 1994; Miller, Schaffer and Hackley 1991). Individuals completing this task take longer to respond to 'go-signals' whilst making more incorrect responses (i.e., errors) when presented with 'no-go signals' following alcohol consumption; thus demonstrating impaired behavioural activation and response inhibition, respectively. However, acute alcohol consumption does not impair response inhibition and behavioural activation similarly, as dissociation between these two mechanisms of behavioural control has been identified. Evidence suggests that inhibitory control is more sensitive to the impairing effects of alcohol when compared to behavioural activation impairment (Abroms, Fillmore and Marczinski 2003; Marczinski and Fillmore 2003). This is likely to influence the maintenance of a binge drinking occasion as it is likely that alcohol consumption will be re-initiated due to this increased sensitivity to inhibitory control impairment (and comparatively reduced sensitivity to behavioural activation impairment). As these mechanisms are impaired differently following an acute dose of alcohol, it is important to consider how this alcohol induced impairment changes during a single drinking session. Since inhibitory control is more sensitive to alcohol induced impairment, it is reasonable to speculate that this mechanism will remain impaired for the duration of a single drinking session whilst the ability to activate a behaviour recovers. Impairment recovery during a single drinking session, despite BAC remaining equivalent, is referred to as acute alcohol tolerance development (Martin and Moss 1993; Mellanby 1919). This will be explored in greater detail

in the next section as well as the role it plays in the initiation and maintenance of a binge drinking occasion.

1.5 Acute Alcohol Tolerance

Research suggests that alcohol tolerance plays a fundamental role in the development of problematic drinking behaviour. In pharmacology, chronic alcohol tolerance refers to a decrease in drug effect when consumption remains constant over time, resulting in the individual requiring larger amounts of the substance to achieve the same desired effect (American Psychiatric Association 2000). The development of tolerance within a single drinking session, referred to as acute tolerance, has also received a lot of research attention. BAC rises when alcohol is absorbed faster than it is eliminated following an acute dose of alcohol. It then falls when the rate of elimination is greater than the absorption rate (McKim and Hancock 2012). These ascending and descending phases of the BAC curve are referred to as the ascending and descending limbs (Parrott, Morinan and Moss 2004). A comparison of behavioural measures at comparable BACs on the ascending and descending limbs allows the investigation of acute tolerance development. Acute tolerance is defined as a single dose exposure to alcohol resulting in decreased impairment at a given BAC on the descending limb when compared to that on the ascending limb of the BAC curve (Martin and Moss 1993; Mellanby 1919). Behavioural measures demonstrate less impaired performance when alcohol intoxication falls; this is due to the acute development of tolerance (Fillmore, Marczinski and Bowman 2005). Recent research has focused on the acute development of tolerance to measures of psychomotor performance and behavioural control. The next section will review the current evidence surrounding this development of tolerance.

1.5.1 Acute Tolerance Development to Psychomotor Performance and Behavioural Control

Research indicates that acute alcohol tolerance develops to measures of psychomotor performance and self-reported subjective intoxication within a single drinking session. Hiltunen (1997a) compared psychomotor performance on the ascending and descending limb at equal BACs in moderate and light drinkers. In this study, moderate drinkers consumed an average of 45.5g of alcohol per week (approximately 6 units) and light drinkers consumed 13.3g per week (approximately 2 units). Psychomotor performance was measured using a computerised pursuit rotor task following moderate (0.5 grams of alcohol/kg of body mass; g/kg) and high doses (1.0g/kg) of alcohol. The light drinkers demonstrated acute tolerance to psychomotor performance at both moderate and high alcohol doses

whereas moderate drinkers only demonstrated this tolerance development at the higher dose. In fact, the evidence also suggested that individuals with greater drinking experiences and past alcohol exposure (moderate drinkers) demonstrated no psychomotor impairment on the ascending limb resulting in no acute alcohol tolerance development (as there was no initial impairment). This may be due to the fact the moderate drinkers used in the study may have greater chronic tolerance to alcohol meaning that they require a greater dose of alcohol to achieve the same impairing effect on the ascending limb. This may also be explained by the simplicity of the task as the low rpm may have made the fixation point easy to track despite consuming 0.5g/kg. More demanding psychomotor tasks may demonstrate greater impairment on the ascending limb and will show whether a significant recovery on the descending limb occurs.

More recent studies have attempted to determine whether aspects of behavioural control (behavioural activation and inhibition) develop acute alcohol tolerance. As previously mentioned, alcohol impairs both behavioural activation and inhibition on the ascending limb (Fillmore, Marczinski and Bowman 2005; Ostling and Fillmore 2010; Reynolds, Richards and de Wit 2006). Weafer and Fillmore (2012) investigated the effects of alcohol consumption on cognitive mechanisms, specifically behavioural control, that are closely associated with driving. They specifically explored alcohol induced behavioural control impairment across the time course of a single drinking session focusing on the descending limb of the intoxication curve. In the context of driving, it is important to understand the effect alcohol has on inhibitory control during a single drinking session, as it is an important cognitive mechanism that implicitly results in the individual not engaging in risky driving behaviours (e.g. sporadic lane changing; Barry 1973; Fillmore, Blackburn and Harrison 2008). Social drinkers were used in the investigation and participants were excluded if they demonstrated alcohol dependence or problematic drinking habits. Behavioural control was measured using the cued go/no-go task. This task has also been used regularly in similar investigations surrounding the disinhibiting effects of alcohol and appears to be an accurate measure of behavioural inhibition and activation (Fillmore, Marczinski and Bowman 2005; Marczinski and Fillmore 2003). It has been validated as a robust measure for detecting impulse control impairment in clinical population of young adults with ADHD (Derefinko et al. 2008) as well as in cocaine abusers (Fillmore and Rush 2005). Psychomotor performance and subjective intoxication were also measured using the grooved pegboard task and an intoxication questionnaire, respectively. Again, as previously mentioned, alcohol consumption significantly impairs psychomotor performance and participants report feeling significantly more intoxicated on the ascending limb compared too sober (Brumback, Cao and King 2007; Tiplady et al. 2001). Performances on all tasks were tested under two conditions including a control condition in which participants received a placebo (i.e., no alcohol) and an experimental condition in which

participants received 0.65g/kg of alcohol (40% ABV). The alcohol condition produced a peak BAC of 90mg of alcohol/100ml of blood (90mg/100ml) and testing was conducted at comparable BACs on the ascending and descending limbs of the BAC curve (i.e., 70mg/100ml).

The results indicated that psychomotor performance and subjective ratings of intoxication recovered on the descending limb. Specifically, participants in this investigation demonstrated greater speed on the psychomotor task and reported feeling less intoxicated on the descending compared to the ascending limb (at equivalent BACs). In addition, participants' ability to activate a response recovered on the descending limb. More interestingly, results show no recovery of inhibitory control on the descending limb. As previously outlined, inhibitory control is an important driving ability that allows individuals to inhibit inappropriate driving behaviours (Barry 1973; Fillmore, Blackburn and Harrison 2008). Therefore, this lack of recovery may result in individuals acting more impulsively and engaging in riskier behaviours. It is also important to consider the fact that subjective intoxication measures did demonstrate acute tolerance; participants reported feeling less intoxicated on the descending limb. As well as this, the ability to execute a behaviour recovered on the descending limb. Taken together, these findings suggest that risky behaviours are likely to be executed due to the recovery of subjective intoxication and behavioural activation alongside the prolonged impairment of inhibition on the descending limb. This may also explain why the drinking behaviour is re-initiated within a binge drinking occasion. It is clear that alcohol induced impairment of psychomotor ability, subjective intoxication and behavioural activation are likely to recover at comparable BACs on the descending compared to the ascending limb. It is also clear that inhibitory control is likely to remain impaired. Past drinking experience and prior exposure to alcohol, resulting in chronic tolerance, is likely to influence the development of acute tolerance during a single drinking session. Therefore, the next section reviews the evidence surrounding acute tolerance development and past drinking exposure.

1.5.2 Prior Exposure to Alcohol and Acute Tolerance

Chronic alcohol tolerance development is greater in frequent, heavy drinkers. These habitual drinkers tend to display less behavioural and cognitive impairment to alcohol than less frequent, lighter drinkers (Brumback, Cao and King 2007; Holdstock, King and de Wit 2000; Townshend and Duka 2005). Fillmore and Weafer (2012) aimed to establish whether this difference in drinking behaviour influenced the development of acute tolerance by attempting to identify whether alcohol induced impairment recovery is more prominent in more frequent drinkers. A total of 40 adult drinkers were used in the study. Participants were classified as either at-risk, heavy drinkers or low risk, light drinkers (Babor, Kranzler and Lauerman 1989) using the alcohol use disorder identification test (AUDIT);

resulting in 20 participants per condition. Again, like the investigation by Weafer and Fillmore (2012), one key aspect of the project focused on alcohol induced inhibitory control impairment (measured using the cued go/no-go task). This task also measured behavioural activation represented by the participant's reaction time to a 'go signal'. Psychomotor performance and subjective intoxication measures were also included as well as the addition of a personal drinking habits questionnaire. The experimental procedure was similar to that of Weafer and Fillmore's (2012) study, in that testing was conducted at comparable BACs on the ascending and descending limb of the intoxication.

The results indicated that heavier drinkers develop a greater tolerance to alcohol induced psychomotor and subjective intoxication impairment on the descending limb compared to low risk, lighter drinkers. In reality, the impairment recovery in heavy drinkers on the descending limb produced almost placebo level scores on psychomotor and subjective intoxication measures despite BAC remaining elevated (above 50mg/100ml). Focusing still on heavy drinkers, the results also indicated that acute alcohol tolerance developed for behavioural activation. The alcohol-induced impairment of behavioural activation was reduced on the descending limb of the alcohol intoxication meaning that the heavy drinking participants were able to react faster to a 'go signal' on the task. Regarding the light, less frequent drinkers, it is reasonable that the reduced development of acute tolerance to measures of psychomotor ability and behavioural control was due to the lack of prior exposure to alcohol; and consequentially the reduced chronic tolerance development to the substance. Consistent with Weafer and Fillmore (2012), no participants developed acute tolerance to alcohol induced impairment of behavioural inhibition. Heavy drinkers clearly demonstrated a favoured behavioural activation recovery which, coupled with this prolonged impairment of inhibitory control, could lead to engagement in more impulsive behaviours (such as the continuation and increased consumption of alcohol leading to binge drinking). This is vitally important in attempting to understand the specific mechanisms behind the development of problematic alcohol use. The findings also indicated that prior exposure to alcohol may contribute to the development of acute alcohol tolerance i.e., heavy drinkers develop greater acute tolerance to behavioural activation, psychomotor performance and subjective intoxication measures. However, as with all cross-sectional studies, a causal relationship between heavy drinkers and increased tolerance cannot be established. However, based on this evidence it is reasonable to conclude that this prior exposure to the substance may lead to greater acute tolerance to alcohol. Therefore, the present thesis will recruit only frequent alcohol consumers to test the development of acute tolerance (and will exclude light, in frequent drinkers). Similar to the investigation by Fillmore and Weafer (2012), the AUDIT scale will be used to test drinking habits and to exclude participants that do not regularly consume alcohol. Whilst considering prior drinking exposure is of apparent importance when investigating acute tolerance development, the

exact nature of acute tolerance development remains unclear. It is likely that this tolerance develops as a function of the experienced sedative and stimulant effects of alcohol (Hendershot et al. 2015). Therefore, it is also necessary to consider changes in stimulant and sedative effects of alcohol during a drinking session. The next section will explore how individuals experience these biphasic effects during a single drinking session focusing specifically on whether they are most influential on the ascending or descending limbs.

1.5.3 Acute Tolerance and Biphasic Alcohol Effects

Alcohol induced impairment, including psychomotor ability and behavioural control, is thought to reflect both stimulant and sedative effects of alcohol. These effects are often measured using the 14-item self-report Biphasic Alcohol Effects Scale (BAES; Martin et al., 1993). Stimulant effects are categorised as an increased state of elation and excitability whereas sedative effects are associated with sluggishness and inactivity (Hendler et al. 2013). Typically, stimulant effects of alcohol are considered to be more positive as they are associated with the feelings of euphoria experienced whilst intoxicated and consequently contribute to increased alcohol consumption (Corbin, Gearhardt and Fromme 2008). Sedative effects are responsible for depressed feelings and are generally considered more negative, often resulting in reduced consumption. However, some sedative effects, including reduced anxiety and stress, are considered to be positive and also promote alcohol consumption (Morean and Corbin 2010).

What remains unclear is the role these biphasic alcohol effects have on alcohol impairment and acute tolerance development. It is reasonable to speculate that sedative effects are associated with increased alcohol impairment, particularly behaviour and motor control, due to the specific characteristics of inactivity and sluggishness. The opposite is likely for stimulant effects. However, Hendershot et al. (2015) demonstrated that stimulant effects were greater on the ascending limb whereas sedative effects were significantly lower. In this investigation, the administration of alcohol resulted in a decrease in stimulant effects and an increase in sedative effects over time. This means that stimulant effects are more active on the ascending limb of the BAC curve compared to the descending limb. Whilst sedative effects are more active on the descending compared to the ascending limb (at comparable BACs). These finding coupled with the fact that alcohol induced psychomotor and behavioural control impairment is greater on the ascending compared to the descending suggest that these biphasic effects play a functional role in the development of acute alcohol tolerance. That is, stimulant effects are associated with increased impairment and sedative effects are associated with reduced impairment, on the ascending and descending limbs, respectively.

The exact reasons for these associations remain elusive. Therefore, understanding the influence of biphasic alcohol effects across a single drinking session and the development of acute alcohol tolerance to psychomotor ability and behavioural control is of importance. The present thesis will address this by investigating whether the findings of Hendershot et al. (2015) are replicable. It is anticipated that greater stimulant effects will be acting on the ascending limb compared to sedative effects, and greater sedative effects will be acting on the descending limb compared to stimulant effects. It has been argued in this section that there may be several factors that contribute to the development of acute alcohol tolerance. These include the associations between the biphasic effects of alcohol and impairment, as well as prior exposure to alcohol and drinking habits. Prior drinking experience shape alcohol outcome expectancies (Reich, Below and Goldman 2010), and it is likely that these expectancies play a functional role in the development of acute alcohol tolerance. The subsequent section will explore alcohol outcome expectancies in greater depth and discuss the role they may play in tolerance development during a single drinking session.

1.6 Alcohol Outcome Expectancies

The social learning perspective has been one of the most influential approaches in explaining cognitive processes that function to promote heavy episodic alcohol consumption (Pabst et al. 2014). One specific cognitive mechanism, outcome expectancy, is a social learning construct that involves individuals becoming increasingly motivated to engage in a particular behaviour due to the anticipated outcomes of completing that behaviour (Bandura 1977). Within the context of alcohol research, outcome expectancies are pre-existing, implicit and explicit beliefs about the likely result of consuming alcohol (Reich, Below and Goldman 2010). Research suggests that individuals acquire alcohol outcome expectancies through direct alcohol related experiences and vicarious learning through observation of alcohol behaviour (Palfai and Wood 2001). They are long term memory structures that have a fundamental role in decision making processes related to consumption (Goldman et al. 1991; Jones, Corbin and Fromme 2001). As a result, variation exists in the type of outcome expectancies held by specific individuals due to varying differences in past alcohol consumption and histories. It is this feature that is thought to contribute to the observable variability in consumption. Typically, individuals with positive alcohol-related experiences will develop positive outcome expectancies (such as ‘I will feel like more of a happy-go-lucky person when I drink’) (Brown, Christiansen and Goldman 1987) whereas individuals that have experienced negative alcohol related consequence will develop negative expectancies (such as ‘I will become argumentative when I drink’) (McMahon and Jones 1993a; McMahon and Jones 1993b). A large body of research has explored the interplay between

alcohol outcome expectancies and drinking behaviour. It is generally supported that positive expectancies increase the likelihood of initiating drinking (Natvigaas et al. 1998), consuming greater volumes of alcohol per drinking occasion (Fromme and D'Amico 2000), and reporting more frequent heavy drinking episodes (Greenfield, Harford and Tam 1991). Similarly, negative outcome expectancies have been linked with reduced alcohol consumption frequency and volume during a single drinking session (Lee, Greely and Oei 1999). The present thesis aims to explore the role of these alcohol consumption outcome expectancies in the development of acute tolerance. It is likely that prior drinking experience and consequential expectations will influence the magnitude of tolerance development during a single drinking occasion. This influence is also likely to differ between individuals that have positive versus individuals that have negative alcohol outcome expectations. Positive and negative outcome expectancies will be explored in the next section.

1.6.1 Positive and Negative Expectancies

The association between alcohol outcome expectancies and drinking behaviour has received a lot of research attention. It is widely accepted that outcome expectancies predict problematic alcohol use. Outcome expectancies have been suggested to predict problematic binge drinking behaviour (Derby 2011; LaBrie, Grant and Hummer 2011; Wardell and Read 2013; Zamboanga 2010) and the occurrence of subsequent binge drinking occasions (Blume, Schmaling and Marlatt 2003). It has also been suggested that they are associated with regrettable drinking-related social behaviours (Dunne and Katz 2015), and various alcohol consumption related consequences (Blume and Guttu 2015; Pabst et al. 2014). One aspect of problematic binge drinking behaviour that has been investigated is involvement in preloading and drinking games. Preloading is described as a risky drinking practise that involves consuming large quantities of alcohol prior to a primary social gathering or event (Foster and Ferguson 2013). Drinking games facilitate this excessive consumption and although they typically occur during preloading they can and often are played any time during a drinking occasion (Borsari 2007). The primary goal of preloading is to get the drinking individual intoxicated before the primary drinking event occurs (Borsari 2004). A study by Zamboanga (2010) aimed to explore whether outcome expectancies predict involvement in preloading and drinking games. They hypothesised that positive expectancies would be associated with an increase in the frequency of preloading, participation in drinking games and hazardous drinking. As well as this, they hypothesised that negative expectancies would be associated with a reduction in these drinking behaviours. Self-report measures were used to explore alcohol outcome expectancies, involvement in preloading and drinking games, and hazardous alcohol use. Results indicated that 98.2% of the 1327 college participants that took part in the investigation reported preloading at least once in the month

prior to assessment and 59.3% of participants reported playing drinking games. This simple demographic data highlights a preference for this type of drinking behaviour in university and college students. It was reported that positive, but not negative outcome expectancies were significantly associated with preloading and drinking game involvement. Therefore, participants that expect positive outcomes as a result of drinking (such as increased sociability) would be more likely to involve themselves in preloading and drinking games. These findings are consistent with expectancy theory and with similar investigations surrounding the influence of outcome expectancies on drinking game and preloading involvement (Zamboanga et al. 2005). This investigation offers support for the notion that alcohol outcome expectancies are associated with increased risky consumption behaviours and greater alcohol related risks. It is also clear that outcome expectancies in a binge-drinking context are associated with the amount of alcohol consumed during the drinking occasion.

There is quite clearly an abundance of compelling evidence to support the association between positive outcome expectancies and increased alcohol consumption (e.g. Fromme and D'Amico 2000; Greenfield, Harford and Tam 1991; Natvigaas et al. 1998). Negative expectancies have also been explored within a similar context (Lee, Greely and Oei 1999) however there are some inconsistencies in alcohol research exploring this association with consumption behaviour. Typically, research has identified that negative expectancies predict lower alcohol consumption (Sharkansky and Finn 1998) and less frequent excessive drinking episodes (Amodeo and Kurtz 1990). However, in other studies, a positive association has been reported between high negative expectancies and increased alcohol consumption behaviour (McMahon, Jones and O'Donnell 1994) whilst Fromme, Stroot and Kaplan (1993) suggested that there is no significant association between consumption and negative expectancies. One possible explanation for this discrepancy is the time-course of the anticipated negative outcomes. Proximal negative outcomes refer to the anticipation of negative consequences on the same day of consumption whereas distal negative outcomes refer to the next day consequences (McMahon, Jones and O'Donnell 1994). McMahon and colleagues report that negative proximal expectancies are less likely to reduce alcohol consumption whereas distal expectancies are more likely to reduce consumption. One possible theory that may explain how negative expectancies curtail alcohol consumption is the influence of behaviour compensation (i.e. negative expectations may exert compensatory mechanisms that reduce the level of alcohol consumption). This will be explored in the next section.

1.6.2 Compensatory Mechanisms and Alcohol Impairment

It is clear that there is an association between alcohol outcome expectancies, both positive and negative, and drinking behaviour (Blume and Guttu 2015; Pabst et al. 2014; Dunne and Katz 2015).

In addition, it is also apparent that alcohol significantly impairs a wide range of cognitive mechanisms including behavioural control, psychomotor ability and subjective ratings of intoxication (Fillmore, Marczinski and Bowman 2005; Ostling and Fillmore 2010; Reynolds, Richards and de Wit 2006; Tiplady et al. 2004; Weafer and Fillmore 2012). Past literature has demonstrated that this impairment is due to the pharmacologic effects of alcohol (McKim and Hancock 2012). Research has also sought to explore whether the expectation of alcohol contributes to alcohol induced impairment. For example, Testa et al. (2006) outlined that alcohol administration studies involving a placebo manipulation demonstrate that the expectation of alcohol results in compensatory effects. This was established by administering a placebo but informing the participant that they were receiving an active dose of alcohol. The expectation alone reduced alcohol impairment which suggests the anticipated intoxicating effects of alcohol were compensated for. Similarly, there was a decrease in impairment following the administration of alcohol when participants were told to expect alcohol compared to when they were told to not expect alcohol. It is apparent that due to the anticipated outcomes of consuming alcohol, individuals become hyper-vigilant to the impairing effects and compensate accordingly. As previously mentioned, alcohol outcome expectations are shaped by direct past experiences with alcohol and vicarious reinforcement through observation (Palfai and Wood 2001). It is therefore likely that the anticipation of negative alcohol consumption consequences increases the amount of compensation and consequently decreases the magnitude of alcohol impairment. Equally, it is likely that the anticipation of positive outcomes has no influence on the amount of intoxication compensation and therefore no influence on impairment.

With regards to chronic alcohol tolerance development, the anticipated outcomes and subsequent compensatory effects are suggested to counteract some if not all of the impairing effects of alcohol over time (Laberg and Löberg 1989). The conditioned compensatory model has been used to explain tolerance development and suggests that through repeated alcohol consumption and direct drinking experiences, individuals learn to compensate for the impairing effects of alcohol. This subsequently results in the substance having less of an impairing effect. As a result, the individual requires more of the drug to achieve the original desired level of impairment (Newlin 1986). However, the role that these outcome expectations have on the development of tolerance within a single drinking session remains unclear. Specifically, the role that positive and negative outcome expectancies have on the acute development of alcohol tolerance has yet to be explored. The present thesis aims to investigate whether individuals who report having negative alcohol outcome expectancies develop greater acute alcohol tolerance. It is likely that individuals with negative expectancies exert greater compensatory effects and consequently develop greater tolerance within a single drinking session. The opposite is likely for individuals with positive alcohol outcome

expectancies as it is expected that they exert less intoxication compensation. To consider these aims effectively, it is important to consider limitations with the methodology used in past acute alcohol tolerance literature, specifically the BAC curve procedure used (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012). This will be explored in the next section and an alternative procedure will be discussed.

1.7 LIMITATIONS OF THE BAC CURVE EXPERIMENTAL PROCEDURE

To investigate acute tolerance development, many studies implement a BAC curve procedure (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012). This involves administering an acute dose of alcohol that produces a BAC curve. On the ascending limb of this curve at a particular BAC a test battery lasting approximately 20-30 minutes is completed by the participant. At the same BAC on the descending limb, this test battery is completed again (at equivalent BAC). One key limitation with this procedure is the pharmacokinetic individual differences that are observable between participants (i.e., the rate of absorption and elimination of alcohol; McKim and Hancock 2012). When the test battery is administered on the ascending limb, BAC is rising dues to alcohol absorption and on the descending limb BAC is declining due to elimination (McKim and Hancock 2012). The rate of alcohol absorption is faster than the rate of elimination and as a result BAC tends to rise at a much faster rate than decline. Therefore, tasks completed towards the end of the task battery on the ascending limb will be at much higher BACs than the same task at the end of the descending limb task battery. This suggests that past research adopting this approach may not have implemented their test battery at comparable BACs on the ascending and descending limbs (Fillmore, Marczinski and Bowman 2005; Ostling and Fillmore 2010; Reynolds, Richards and de Wit 2006). Despite an overwhelming amount of literature supporting the development of acute alcohol tolerance and its impact on drinking behaviour (specifically the role of behavioural control), there is very little consideration for the specific psychological factors that may contribute to its development. To overcome this, the order of the tasks in the battery in the present thesis will be reversed on the descending limb so that tasks are completed at approximately comparable BACs. Specifically, the first task of the test battery on the ascending limb will be the same as the last task of the test battery on the descending limb. Equally, the last task of the test battery will be completed at the end and the start of the test battery, on the ascending and descending limbs, respectively. This will allow for a more accurate investigation of acute alcohol tolerance development.

1.8 AIMS OF THESIS

1.8.1 RESEARCH OBJECTIVES

The above reviewed evidence suggests that tolerance develops to the impairing effects of alcohol on measures of behavioural activation, psychomotor ability and subjective intoxication within a single drinking episode, but does not develop to measures of inhibition (Hiltunen 1997a; 1997b; Ostling and Fillmore 2010; Weafer and Fillmore 2012). Evidence also suggests that the expectation of alcohol increases compensatory effects, and over time increases the amount of chronic alcohol tolerance (Newlin 1986). To date, the association between these outcome expectancies and the acute development of tolerance is unclear. The present thesis is a two part investigation aimed at testing the replicability of past acute tolerance literature and exploring the specific role of alcohol outcome expectancies in the development of this tolerance. The first phase of the investigation will test the replicability of past acute alcohol tolerance literature and confirm whether acute alcohol tolerance develops to behavioural control, psychomotor ability and subjective intoxication. A revised BAC curve limb comparison procedure will be implemented to ensure cognitive tasks are completed at comparable BACs; this will ensure that the investigation accurately models acute alcohol tolerance. Consistent with past research, impairment recovery on the ascending limb (i.e., acute tolerance development) is expected for measures of behavioural activation, psychomotor performance and subjective intoxication but not for inhibition. In addition, the first phase of this two part investigation will also explore changes in self-reported biphasic effects of alcohol (stimulant and sedative effects) on the ascending and the descending limb of the BAC curve. If findings replicate past literature (I.e., provide evidence for the development of acute alcohol tolerance), the magnitude of tolerance development will be calculated for all measures that demonstrate impairment recovery on the descending compared to the ascending limb of the BAC curve.

The second phase of the present thesis will investigate whether pre-existing alcohol outcome expectancies (i.e., positive and negative outcome expectations) are associated with the magnitude of acute tolerance development. As previously mentioned, anticipating alcohol impairment results in intoxication compensation (Newlin 1986). That is, individuals become hyper vigilant to the impairing effects of alcohol and adjust their behaviour accordingly. Alcohol outcome expectations are shaped by direct past experiences with alcohol and vicarious reinforcement through observation (Palfai and Wood 2001). It is likely that negative expectations exert greater intoxication compensation. As the individual may become more vigilant to negative consequence. It is predicted that negative alcohol outcome expectancies will result in intoxication compensation, whereby participants demonstrate

hyper-vigilance to the impairing effects of alcohol and thus develop an increased behavioural tolerance within a single session. The opposite is expected for positive alcohol outcome expectancies due to no anticipated compensatory effects.

1.8.2 HYPOTHESES

1.8.2.1 Acute tolerance to the Impairing Effects of Alcohol

Phase one of the present investigation aims to establish whether acute alcohol tolerance develops to measures of behaviour control, psychomotor performance and subjective ratings of intoxication. Acute tolerance across all hypotheses refers to the impairment recovery on the descending compared to the ascending limb of the BAC curve, at comparable BAC.

H1. Behavioural activation (i.e., ability to activate a response) will develop acute alcohol tolerance, whereas behavioural inhibition (i.e., the ability to inhibit a prepotent response) will not develop acute alcohol tolerance.

H2. Psychomotor performance (i.e., speed and accuracy) will develop acute alcohol tolerance.

H3. Subjective intoxication (i.e., self-reported levels of intoxication) will develop acute alcohol tolerance.

H4. Participants will report greater feelings of stimulation on the ascending limb (compared to the descending limb) and greater feelings of sedation on the descending limb (compared to the ascending limb).

1.8.2.2 The role of outcome expectancies and the development of acute alcohol tolerance

Phase two of the present investigation aims to investigate whether the magnitude of acute alcohol tolerance development can be predicted by pre-existing alcohol outcome expectancies. The magnitude of tolerance will only be calculated for measures that demonstrate evidence of acute alcohol tolerance development in phase one.

H5. Negative outcome expectancies (i.e., anticipation of negative consequence) will predict greater acute tolerance development. Whereas positive expectancies (i.e., anticipation of positive outcomes) will not predict greater acute tolerance development.

Chapter 2: Methods

2.1 ACUTE TOLERANCE TO THE IMPAIRING EFFECTS OF ALCOHOL

2.1.1 DESIGN

The present investigation used a within participant experimental design. The independent variable was the time course of alcohol intoxication with three levels. These were a zero-alcohol baseline (before alcohol was consumed at zero minutes), the ascending limb (at approximately 20 minutes post alcohol consumption) and the descending limb (approximately 70 minutes post alcohol consumption). Several dependent measures were used including two subjective intoxication measures, two psychomotor performance measures and a behavioural activation/inhibition measure. Psychomotor performance was measured using the Zig-Zag tracking task and the grooved pegboard task. Behavioural control (activation/inhibition) was measured using the Cued go/no go task. Subjective intoxication was measured using a researcher constructed VAS scale and the Biphasic Alcohol Effect Scale.

2.1.2 PARTICIPANTS

2.1.2.1 Participant Demographics

Participants were recruited from the staff and student population of Coventry University, and the general population using the University's internal SONA participant recruitment system, email invitation and social media advertisement. There were 21 participants in total (13 male and 8 female) with a mean age of 24.75 years (SD = 1.68); ages ranged from 19-43 years old. With regards to the occupation of the sample, 76.20% of participants were students, 9.5% were academic researchers of Coventry University and the remaining 14.3% were non-academic workers. The majority of participants (76.20%) were White British, 9.60% were Black British, 4.8% were Indian, 4.8% were Asian and the remaining 4.8% did not report their ethnicity. Smoking habits were also recorded and the majority of the participants (81%) were non-smokers. The remaining 19% of participants reported smoking regularly and consumed at least 5 cigarettes per day. Drinking habits were assessed and all participants were regular drinkers having reported consuming at least 2-4 alcoholic drinks per month. In addition to this, 52.4% of participants reported drinking alcohol 2-3 times per week and 4.8% at least 4 times per week. On a typical drinking occasion, the majority of participants reported consuming 7-9 units of alcohol (38.1%), 23.8% consumed in excess of 10 units, 19.0% consumed 5-6 units, and

the remaining participants consumed fewer than 5 units (19.1%). Finally, binge drinking behaviour was assessed and showed that the majority of participants (90.5%) reported binge drinking at least once per month; this is defined as consuming in excess of 8 units per occasion for males and 6 for females. Of these participants, 42.9% reported binge drinking at least once per week.

2.1.2.2 Inclusion and Exclusion Criteria

A self-report health and medical history questionnaire thoroughly assessed general health, health issues, drug use (medical and recreational) and other demographic information (such as age, gender and ethnicity) (*Appendix 1*). All participants had to be above the age of 18 years old. Participants that self-reported any contraindication to alcohol, impaired cardiovascular functioning, seizure or head trauma were excluded from participation. Participants were also asked about past or present diagnosis of psychiatric disorders as outlined in Axis-I of the DSM- V (American Psychiatric Association 2013). Those that reported a clinical psychiatric disorder were excluded. The Short Michigan Alcohol Screening Test (S-MAST; Seltzer, Vinokur and Van Rooijen 1975) was used to explore alcohol dependence and problematic use. In similar acute tolerance research, participants that scored a score of 5 or greater on the S-MAST scale demonstrated high alcohol dependence or problematic alcohol use and were excluded from participation (Fillmore and Weafer 2012; Weafer and Fillmore 2012). The same cut off point was used in the present investigation to exclude participants that demonstrated problematic dependence and/or alcohol dependence.

2.1.2.3 Recruitment, Remuneration, and Ethics

Participants recruited from the student population of Coventry University received research credits (a requirement of their course) as remunerations and those recruited via social media received a £5 *love2shop* voucher per hour. Remunerations were not used as incentives and did not affect the participant's right to withdraw. Signed informed consent was obtained after participants were informed about the specific requirements of the investigations, their right to terminate the experiment at any time and their right to withdraw their data for up to 3 weeks following the conclusion of the investigation. A full debrief was given to each participant once the experiment had concluded. The study was approved by the Coventry University Ethics Committee and conformed to the British Psychological Society (BPS) ethical consideration guidelines (reference: P31903; Appendix 2).

2.1.3 MEASURES

2.1.3.1 Alcohol Administration and Breath Alcohol Measure

Participants consumed 0.65g/kg of alcohol. The drink consumed consisted of *Tesco Value* vodka (37.5% alcohol by volume; ABV) and *Tesco* summer fruits sparkling water. The drink produced an approximate peak BAC of 90mg/100ml. BACs were measured using the Lion 500 Alcometer (Lion laboratories ltd., Vale of Glamorgan, UK). The Lion 500 Alcometer measures Breath Alcohol Concentration (BrAC) in mg/l. This was converted to BAC (mg/100ml) using the widely accepted UK blood breath ratio (2300: 1) (Jones 1990).

2.1.3.2 Prior Alcohol Consumption and Problematic Alcohol Use

Short Michigan Alcohol Screening Test (S-MAST)

The Short Michigan Alcohol Screening Test (S-MAST; Seltzer, Vinokur and Van Rooijen 1975) is a 13-item questionnaire aimed at measuring alcohol dependence (Appendix 3). The test includes questions such as 'Have you ever gotten into trouble at work because of your drinking?' and 'Have you ever been arrested, even for a few hours, because of other drunken behaviours?' and requires a yes or no answer. Research has demonstrated good internal consistency (α = .84; Hays et al. 1995). Each yes answer is equal to a score of 1 and the S-MAST score is calculated by summing the yes answers. The total scores therefore range from 0-13.

Alcohol Use Disorders Identification Test (AUDIT)

The Alcohol Use Disorders Identification Test (AUDIT) is a widely used alcohol screening instrument developed by the World Health Organisation (WHO; de Meneses-Gaya 2009) and can be seen in Appendix 4. It consists of 10-items measuring the frequency of alcohol consumption (items 1-3), alcohol dependence (items 4-6) and problems associated with alcohol use (items 7-10). Items 1-8 are scored on a 5-point Likert scale (0,1,2,3,4) and items 9-10 on a 3-point Likert scale (0,2,4); total AUDIT scores therefore range from 0-40 (Shelvin and Smith 2007). Research has demonstrated that the AUDIT has good internal consistency (α = .95; Carey, Carey and Chandra 2003) and good test-retest reliability (r = .95; Dybek et al. 2006). The outcome measure of interest for the present investigation was the total AUDIT score. Higher scores reflect greater alcohol use whereas lower scores reflect lower alcohol use.

2.1.3.3 Behavioural Control

Cued Go/No-go task (CGNGT)

The cued go/no-go (CGNGT) reaction time task measures behavioural activation and inhibition and is widely used in past alcohol research (e.g. Fillmore, Marczinski and Bowman 2005; Marczinski and Fillmore 2003). The task is operated by E-prime experiment software and requires roughly 10-12 minutes to complete (Schneider, Eschman and Zuccolotto 2002). Each trial within the task begins with a fixation point (+) for 800ms, followed by a blank screen for 500ms. A blank rectangle shaped cue is then presented either vertically or horizontally on the screen followed by either a go or no-go signal (green or blue coloured, respectively). If a go signal is presented (green) the participant is required to press the assigned computer response key (/) as quickly as possible. If a no-go signal is presented (blue) the participant was required to avoid pressing the assigned computer key (/). The orientation of the initial colourless rectangle signalled the probability of a go or no-go signal following. For example, a vertically positioned rectangle indicates that a go-signal (green) should follow; 80% of vertically positioned rectangles are followed by a go signal. A horizontally positioned rectangle indicates a no-go signal (blue) should follow; 80% of horizontally positioned rectangles are followed by a no-go signal. Two outcome measures of interest taken from the cued go/ no-go task were used in the present investigation; behavioural activation and inhibition. Reaction time scores (in milliseconds) were determined by the speed at which participants responded to go-signals (green) in no-go cued trials (horizontal rectangle). Faster reaction times to go signals in no-go trials reflects behavioural activation as this demonstrates a greater ability to activate a response when cued to suppress it. Trials on which participants responded to these go-cues in less than 100ms and greater than 1000ms were excluded (less than 2% of trials). The second measure of interest was behavioural inhibition. The frequency with which participants responded to no-go signals (blue) in a go-cued trial (vertical rectangle) was taken to reflect behavioural inhibition (Miller and Fillmore 2014; Ostling and Fillmore 2010).

2.1.3.4 Psychomotor Performance

Grooved Pegboard (GPB)

The Grooved Pegboard Task (GPB) is a measure of psychomotor co-ordination and dexterity (Klove 1963; Lafayette Instruments 1989). It is widely used in healthcare professions (Causby et al. 2014) and in past alcohol research (Miller and Fillmore 2014; Fillmore and Weafer 2012; Weafer and Fillmore 2012). The pegboard consists of 25 peg holes organised in a 5x5 grid. The peg holes are key shaped with a grooved edge and a rounded top. All holes are identical but are arranged so no adjacent key hole is pointing in the same direction (they are slightly rotated). Participants are equipped with

identical pegs that match the holes on the pegboard (all pegs are equal in shape, size and weight). The aim of the task was to put the pegs into the peg holes in the fastest time possible. Participants self-reported whether they were right-hand dominant or left-hand dominant. Right handed participants started with the top left hole and worked left to right ending on the bottom right hole. Left handed individuals started top right of the grid and worked right to left ending on the bottom right hole. The measure of interest was the speed at which participants completed the pegboard task. Scores were calculated based on 4 trials; mean time (seconds) was calculated for each participant once all trials concluded (Levine et al. 2004).

Zig-Zag Tracking Task (ZZTR)

The Zig-Zag Tracking Task (ZZTR) is a pen and paper task measuring psychomotor accuracy and speed (Tiplady et al., 2004; Appendix 5). Participants are required to navigate their way from start to finish along a light grey zig-zag track whilst avoiding black circle obstacles. The ZZTR test includes 20 different, but equally difficult, variants of the obstacle layout (I.e., black circle obstacles were position differently on each difference variant). This ensured that repeated completion of the task and consequentially increasing practise had very little impact on performance (Tiplady et al. 2004). The present study utilises two outcome scores from this task (speed and accuracy). The time taken to reach the end of the track, measured in seconds, reflects psychomotor speed. The psychomotor error score is calculated by adding up the frequency of errors. One error point is scored for touching the black obstacle or edge of the track and two error points are scored when the participant goes through the obstacle or completely leaves the track.

2.1.3.5 Self-report Intoxication

Subjective Intoxication Scale (SI)

The self-report level of intoxication questionnaire was an adapted version of the subjective feelings scale developed by Bond and Lader (1974). It was used to measure how intoxicated each participant subjectively felt throughout the experiment (Appendix 6). It asked a single question ('How drunk do you currently feel?') and participants were required to score how intoxicated they felt on a 100mm visual analogue scale (VAS); 0 (completely sober) to 100 (highly intoxicated). Higher scores on the VAS indicated greater levels of self-reported intoxication and lower scores indicated lower levels of intoxication.

Biphasic Alcohol Effects Scale (BAES)

The Biphasic Alcohol Effects Scale (BAES; Martin et al., 1993) is a 14-item measure that captures self-reported sedative (7-items) and stimulant (7-items) effects of alcohol (Appendix 7). Each

item is an adjective that describes typical feelings an individual might experience after consuming alcohol (Martin et al. 1993). The adjectives that reflect stimulant effects of alcohol include 'elated' and 'excited' and the adjectives that reflect sedative effects include 'down' and 'sluggish'. Participants were required to rate each item on a 10-point scale ranging from 1 (Not at all) to 10 (Extremely). A total for both the sedative and stimulant effects of alcohol was calculated. A high score on each biphasic effect measure equals an increased subjective experience of this effect (e.g. a high stimulant effect score suggests that the participant was experiencing heightened stimulation). Corbin et al. (2015) report that the BAES has strong internal consistency (α values range from .88 to .94 for sedation items and .78 to .88 for stimulant items).

2.1.4 PROCEDURE

2.1.4.1 Preliminary Screening

Participants were required to attend a short screening session. In this session participants completed the AUDIT and S-MAST scales to gain information about their past alcohol use and alcohol dependence. As well as this, each participant completed a health and medical history questionnaire to assess whether they had any health concerns that would prevent them from being able to participate. This questionnaire also obtained basic demographic information including age, gender, ethnicity and religion. Following this, participants were allocated time to practise the battery of tasks used in the experimental paradigm to ensure they were well practised and understood the demands of each task. Participants were informed via email if they did not meet the inclusion criteria and those that did were invited back to the testing session. Signed informed consent was obtained following a thorough health and safety brief.

2.1.4.2 Testing

The testing procedure utilised was an adapted version of the process outlined by Miller and Fillmore (2014). Prior to taking part, participants were asked to fast for 4 hours (no food or caffeinated drinks) and to abstain from using any psychoactive substances 24 hours prior to participating. On arrival, participants were required to provide a breath alcohol reading of 0 mg/100ml. They then completed a counterbalanced test battery prior to receiving alcohol as a baseline performance measure. This battery comprised of the ZZTR, the GPT, the CGNGT, the BAES and the self-report subjective intoxication tasks. Following this, weight was measured in order to calculate the most appropriate dose of alcohol to be administered. Evidence suggests that males metabolise alcohol more efficiently due to the activity of alcohol dehydrogenase. The presence of this enzyme is less active in females resulting in increased BACs compared to males following the consumption of an

identical dose of alcohol (Frezza et al. 1990). The alcohol dose administered was .65g/kg for males and approximately .57 g/kg for females. This difference in dosage was to account for differences in alcohol metabolism identified by gender and aims to achieve the same BACs for males and females. The alcohol was then administered in the ratio of one-part alcohol to three-parts carbonated soda, split into 2 equal drinks. Participants had 3 minutes to consume each drink and they were served 4 minutes apart. BAC was measured using the Lion 500 Alcometer every 10 minutes following the consumption of the second drink until the experiment concluded. This was done to ensure that the ascending limb for each individual participant began at approximately 80mg/100ml and ended at approximately 90mg/100ml. The exact reverse BAC scores were expected for the descending limb. Participants were required to complete the counterbalanced test battery on the ascending and descending limb of the BAC curve which took approximately 30 minutes to complete; the task order was reversed on the descending limb to ensure each individual task was completed at comparable BACs. Prior to testing, the testing procedure was piloted using a small sample of healthy volunteers. This was done to determine the appropriate time to run the task battery on the ascending and descending limbs. It was confirmed that at approximately 20-50 minutes post alcohol administration was the ascending phase and 70-100 minutes was the comparable descending phase of intoxication; peak intoxication was approximately 60 minutes post consumption.

Participants started the tasks on the ascending limb at approximately 20 minutes' post alcohol administration and completed them at approximately 50 minutes' post administration. The mean BACs at these time points were 80.50mg/100ml (SD = 14.26) and 92.00mg/100ml (SD = 12.42), respectively. Peak intoxication was achieved at approximately 60 minutes' post administration. The task battery was completed on the descending limb starting at approximately 70 minutes and finishing 100 minutes' post administration; mean BACs were 91.77mg/100ml (SD = 16.10) and 78.20mg/100ml (SD = 11.50), respectively. Paired samples t-tests reveal that there was no significant difference between BACs when the tasks were started on the ascending limb (M = 80.50mg/100ml, SD = 14.26) and when they ended on the descending limb (78.20mg/100ml, SD = 11.50); $t(20) = .274$, $p = .787$. As well as this, no significant difference was found between BAC scores when tasks concluded on the ascending limb (92.00mg/100ml, SD = 12.42) and when they started on the descending limb (91.77mg/100ml; SD = 16.10); $t(20) = .580$, $p = .568$. This confirmed that reversing the test battery on the descending limb ensured individual tasks were completed at comparable BACs. Once completed and when alcohol intoxication dropped below 20mg/100ml participants were debriefed and allowed to leave the laboratory.

2.2 THE ROLE OF OUTCOME EXPECTANCIES AND THE DEVELOPMENT OF ACUTE ALCOHOL TOLERANCE

2.2.1 DESIGN

A correlational design was used to investigate whether pre-existing positive and negative alcohol outcome expectancies predict the magnitude of acute alcohol tolerance. The acute tolerance outcome variables include measures of behavioural control, psychomotor ability and measures of subjective intoxication; only measures that demonstrate significant acute tolerance development were analysed. The predictor variables included self-reported positive and negative alcohol outcome expectancies.

2.2.2 PARTICIPANTS

The same sample of participants were contacted after they had taken part in the above acute alcohol tolerance investigation (N=21). Please see *2.1.1 Participants* for additional information surrounding participant demographics and recruitment methods. Participants were made aware of their right to terminate their involvement in this follow up at any time and their right to withdraw their data for up to 3 weeks following the conclusion of the investigation. Following this, participant gave informed consent before completing the online questionnaires. A full debrief was given to each participant once the study had concluded. The collection of this additional data in this follow up study was approved by the Coventry University Ethics Committee and conformed to the British Psychological Society (BPS) ethical consideration guidelines (reference: P144178; Appendix 8).

2.2.3 MEASURES AND MATERIALS

2.2.3.1 Behavioural Control

The Cued Go/No-go response data was used. This included reaction time scores (in milliseconds) to go signals in no-go cued trials (behavioural activation measure), and the frequency of responses to no-go signals in go trials (inhibitory control measure) (see Cued Go/No-go task in *2.1.3.3 Behavioural Control*). The difference between descending and ascending limb scores on these measures were used as a proxy measure of the magnitude of acute alcohol tolerance development (i.e., the amount performance recovered on the descending compared to the ascending limb).

2.2.3.2 Psychomotor Performance

The GPB and ZZTR task response data was used. The mean time to complete the GPB task (in seconds) and the mean time to complete (seconds) and error rates (frequency of error) on the ZZTR task were the measures of interest (see GPB and ZZTR task details in 2.1.3.4 Psychomotor Performance). Again, the difference between descending and ascending limb scores on these measures were used as a proxy measure of the magnitude of acute alcohol tolerance development.

2.2.3.3 Self-report Intoxication

Subjective intoxication response data was used from the SI scale and the BAES. The level of intoxication reported by participants on a 0-100 VAS and the self-reported levels of stimulant and sedative alcohol effects were the measures of interest (see SI scale and BAES in 2.1.3.5 Self-report Intoxication). The difference between descending and ascending limb scores on these measures were used as a proxy measure of the magnitude of acute alcohol tolerance development.

2.2.3.4 Alcohol Outcome Expectancies

Alcohol Expectancy Questionnaire (Adult Version; AEQ-III)

The alcohol expectancy questionnaire-III (Brown, Christiansen and Goldman 1987; AEQ-III) is a self-report measure that assesses an individual's positive alcohol outcome expectations based on past drinking experience and prior consumption behaviour (Appendix 9). The adult version used in the present study consist of 90 statements (68 items are scored) that assess 6 individual positive expectancy sub- factors. These are anticipated global positive change (N = 24), sexual enhancement (N = 7), physical/social pleasure (N = 9), social assertiveness (N = 10), tension reduction (N = 9) and arousal (N = 9) (Brown, Christiansen and Goldman 1987). Participants are required to score each statement on a five-point Likert scale ranging from '1- Strongly Disagree' to '5 - Strongly Agree'. Scores were totalled for each sub-factor and the higher the score the greater the expectancy for that. An aggregated total of all the sub-factor scores was used to indicate overall positive outcome expectancies in the present study.

Negative Alcohol Expectancy Questionnaire (NAEQ)

Negative expectations are thought to develop following negative drinking experiences. The negative expectancy questionnaire (McMahon and Jones 1993a; McMahon and Jones 1993b; NAEQ) is a 60 item self-report scale that assesses these anticipated negative outcomes (Appendix 10). Each item is a statement that reflects negative consequences of consuming alcohol. Participants were required to score each statement on a five-point Likert scale ranging from '1 Strongly Disagree – 5

Strongly Agree'. There are 3 sub-categories within the NAEQ that assess same day (N = 21), next day (N = 18) and continued alcohol consumption (N = 21) negative expectancies. Response scores were totalled for each sub-category and the aggregated total of these categories was used to as the total negative expectancy in the present study.

2.2.4 Follow up Procedure

The first phase of this thesis was to explore whether acute alcohol tolerance developed following a single dose exposure to the substance and to assess whether past findings of this were replicable (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012) for behavioural control, psychomotor ability and subjective ratings of intoxication. The second phase of the investigation aimed to explore the interplay between pre-existing alcohol outcome expectancies and the development of acute alcohol tolerance. This was due to the fact that it was deemed likely that compensatory mechanisms associated with outcome expectancies (Newlin 1986) would influence the development of tolerance during a single drinking session. Data for this second phase was collected retrospectively (approximately 3 months after the initial acute tolerance investigation). Evidence suggests that priming alcohol outcome expectancies can influence drinking behaviour (Friedman et al., 2009). It was therefore deemed appropriate to collect expectancy data after the completion of the acute alcohol tolerance experimental study to avoid any undue influence of priming. This retrospective data collection would not have likely influenced the results of the study as evidence suggests that alcohol outcome expectancies are internalised beliefs shaped from drinking experiences that remain constant over time (Jones, Corbin and Fromme 2001). As a result, all participants were contacted after the completion of the acute tolerance investigation and were asked to complete the AEQ-III and the NAEQ. These measures collected data on each participant's self-report alcohol outcome expectancies based on their past drinking experiences and prior alcohol consumption behaviour. These questionnaires were made available on Bristol Online Survey and took approximately 20 minutes to complete.

Chapter 3: Results

3.1 Acute tolerance to the impairing effects of alcohol

3.1.1 Statistical Analysis

One-way repeated measures ANOVAs were used to test the development of acute alcohol tolerance for measures of behavioural control (i.e., behavioural activation and inhibition), psychomotor performance (i.e., speed and accuracy) and subjective ratings of intoxication (i.e., self-reported intoxication and biphasic effects). As there are several dependent measures (*see section 2.1.1*) MANOVA was considered as a method of statistical analysis. However, MANOVA requires a strong conceptual link between dependent measures (Field 2013). As the dependent measures in the present investigation do not have a strong conceptual link, separate one-way ANOVAs were preferred. Data were first screened for outliers and normal distribution which revealed minor assumption violations (*see Appendix 11.1 and Appendix 11.2, respectively*). Mauchly's test was used to assess the assumption of sphericity (*Appendix* 11.3); sphericity was assumed unless otherwise stated. Repeated measures Analysis of Variance (ANOVA) is robust to minor violation (Field 2013; Tabachnick and Fidell 2012) and was therefore used to explore task performance differences between baseline, ascending and descending limb scores for all dependent measures (CGNGT, ZZTR, GPB, SI, BAES). In addition, 2 a priori planned (repeated) contrasts were conducted to explore differences between baseline and ascending limb scores as well as differences between ascending and descending limb scores. Lower performance scores on the ascending limb compared to the baseline recording reflects greater alcohol impairment and higher scores on the descending compared to the ascending limb reflects greater acute alcohol tolerance. Effect size estimates are Omega-squared (ω^2). This was preferred as it offers a less biased estimate of effect size than eta-squared (n^2) in smaller samples (Keselman 1975).

3.1.2 Descriptive Statistics

Table 1 summarises the mean (standard deviation) scores for all dependent measures at all time points. It appears that alcohol impaired behavioural control, psychomotor ability and self-report subjective intoxication as poorer performance is demonstrated on the ascending limb compared to baseline. The mean performance score on the descending limb appear to be in the expected direction with the exception of error frequency on the CGNGT (i.e., demonstrating alcohol impairment recovery). Participants appeared to respond quicker to go-signals in no-go trials on the CGNGT suggesting that behavioural activation develops acute alcohol tolerance. They also appeared to

complete the ZZTR task faster with fewer errors, and took less time to complete the GPB. This suggests that alcohol induced psychomotor ability impairment (both speed and accuracy) appears to recover on the descending compared to the ascending limb. Finally, the mean ratings of intoxication appear to be lower on the descending compared to the ascending limb. This suggests that participants felt less intoxicated on the descending limb demonstrating acute alcohol tolerance. Participants also reported experiencing greater stimulant effects of alcohol on the ascending limb and greater sedative effects on the descending limb.

Table 1: Mean (Standard Deviation) values for each dependent measure (CGNGT, ZZTR, GPB, SI, BAES) at baseline, ascending and descending limb time points.

N=21	Mean (Standard Deviation)		
	Baseline	Ascending Limb	Descending Limb
CGNGT (Error Frequency)	.11 (.13)	.18 (.18)	.19 (.21)
CGNGT (RT; Milliseconds)	342.45 (33.48)	361.47 (33.91)	358.03 (33.91)
ZZTR Speed (Seconds)	77.22 (25.52)	71.93 (23.89)	63.07 (19.07)
ZZTR Accuracy (Error total)	13.63 (10.13)	24.39 (11.67)	20.07 (11.45)
GPB Speed	63.05 (11.09)	64.50 (9.82)	60.89 (8.90)
SI	.76 (1.55)	56.76 (19.47)	37.33 (22.56)
BAES Sedative Effects	14.38 (13.16)	10.81 (6.83)	20.10 (16.36)
BAES Stimulant Effects	26.57 (11.41)	39.57 (12.97)	26.52 (14.15)

3.1.3 Behavioural Control

3.1.3.1 Cued Go/No-go task

To address the first hypothesis (H1), two repeated measures ANOVA were used to explore differences in the CGNGT error frequency scores (behavioural inhibition measure) and response RT (behavioural activation measure) between the baseline, ascending and descending limb time points (mean (SD) scores can be seen in *Table 1*). Analysis showed that both CGNGT error frequency and response RT significantly differed across time points, F (2, 40) = 4.83, p = .013, ω^2 = .03 and F (2, 40) = 6.01, p = .005, ω^2 = .05, respectively. Planned contrast analysis comparing baseline and ascending limb

error frequency scores showed that participants made significantly more errors on the ascending limb compared to the baseline and showed that this observed difference was moderate-large, F (1, 20) = 8.04, p = .010, r = .54. Response RT was significantly slower on the ascending limb compared to the baseline and this observed difference was also moderate-large, F (1,20) = 8.93, p = .007, r = .56. This demonstrates that alcohol significantly impairs both the ability to inhibit a response and the ability to activate one. As well as this, planned contrast analyses comparing ascending and descending limb scores showed that there was no significant difference between the error frequency scores nor response RT scores at these time points, F (1, 20) = .43, p = .520, r = .14 and F (1,20) = .51, p =.485, r = .16, respectively. This outlines that participants did not develop acute alcohol tolerance to behavioural inhibition or behavioural activation (no significant performance recovery on the descending limb).

Figure 1 plots the mean error frequency scores and response RT's at baseline, ascending and descending limb time points.

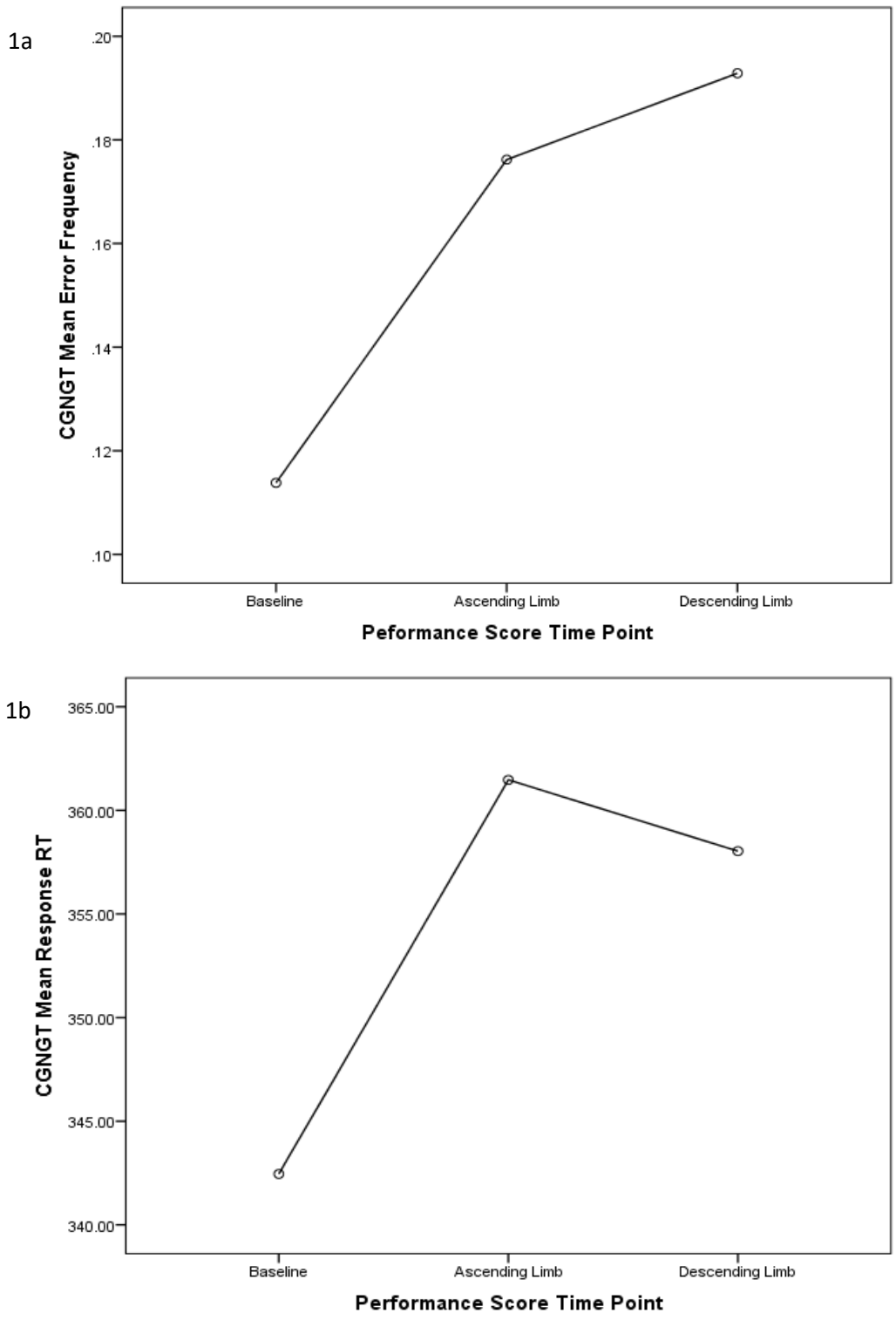


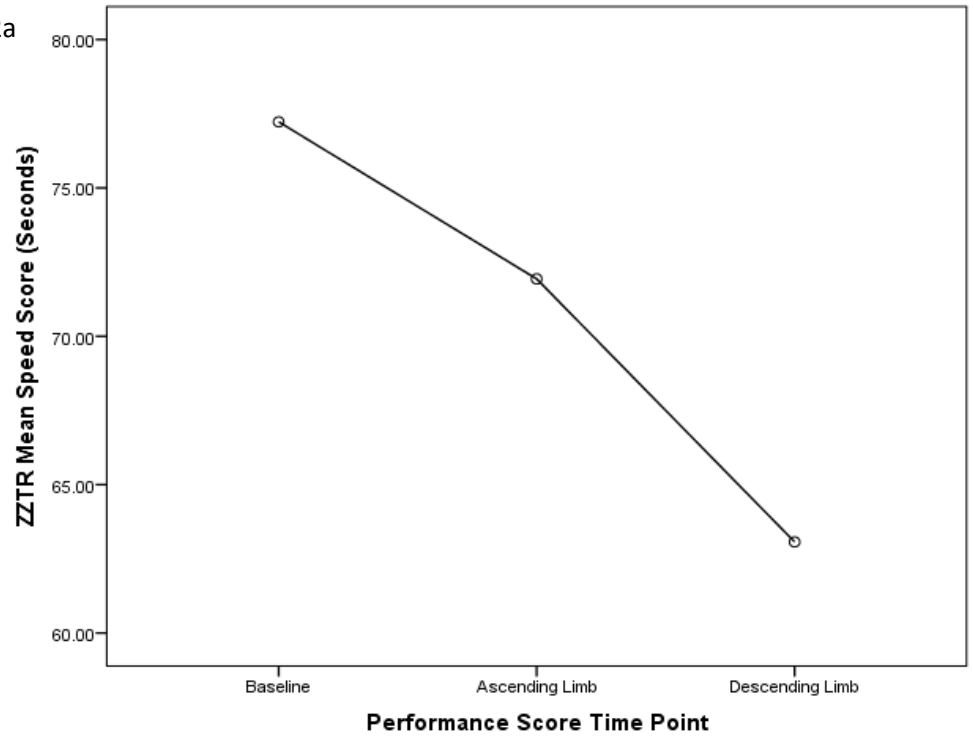
Figure 1: Mean CGNGT Error Frequency Scores (1a) and Mean CGNGT Response RT Scores (1b) at Each Time Point (Baseline, Ascending and Descending Limb).

3.1.4 PSYCHOMOTOR PERFORMANCE

3.1.4.1 Zig-Zag Tracking Task (ZZTR)

To address hypothesis two (H2), repeated measure ANOVAs were used to explore psychomotor speed (ZZTR time in seconds) and accuracy (ZZTR error total) score differences between baseline, ascending and descending limb time points (mean (SD) scores can be seen in *Table 1*). Analysis showed that both psychomotor speed and accuracy as measured by the ZZTR task significantly differed across these time points, F (2, 40) = 20.05, $p < .001$, $\omega^2 = .06$ and F (2, 40) = 40.20, $p < .001$, $\omega^2 = .13$, respectively. Alcohol impairment was explored using planned contrast analyses comparing the baseline and ascending limb psychomotor speed and accuracy scores. These revealed that psychomotor speed significantly and moderately increased on the ascending limb compared to the baseline measurement, F (1, 20) = 6.87, $p = .016$, $r = .51$ whilst psychomotor accuracy significantly and largely decreased, F (1, 20) = 58.99 $p < .001$, $r = .86$ (these trends can be seen in *Figure 2*). This suggests that alcohol impairs psychomotor ability by increasing the speed at which the task is carried out which, in turn, increases the total amount of errors made. Planned contrasts were also used to explore psychomotor speed and accuracy performance score differences on the ascending and descending limb. Analysis demonstrated that the measure of psychomotor speed demonstrated a significant moderate increase on the descending compared to the ascending limb, F (1, 20) = 16.82, $p = .001$, $r = .68$. This suggests that participants took less time to complete the psychomotor task on the descending compared to the ascending limb, at comparable BACs. The measure of psychomotor accuracy showed that participants made significantly fewer errors on the descending compared to the ascending limb, F (1, 20) = 25.16, $p < .001$, $r = .75$ (these trends can be seen in *Figure 2*). Taken together, the time taken to complete the psychomotor task appears to reduce (*Figure 2*). This combined with the decrease in error rate suggests that psychomotor performance recovers on the descending compared to the ascending limb by increasing the speed at which a task is completed whilst making fewer mistakes.

2a



2b

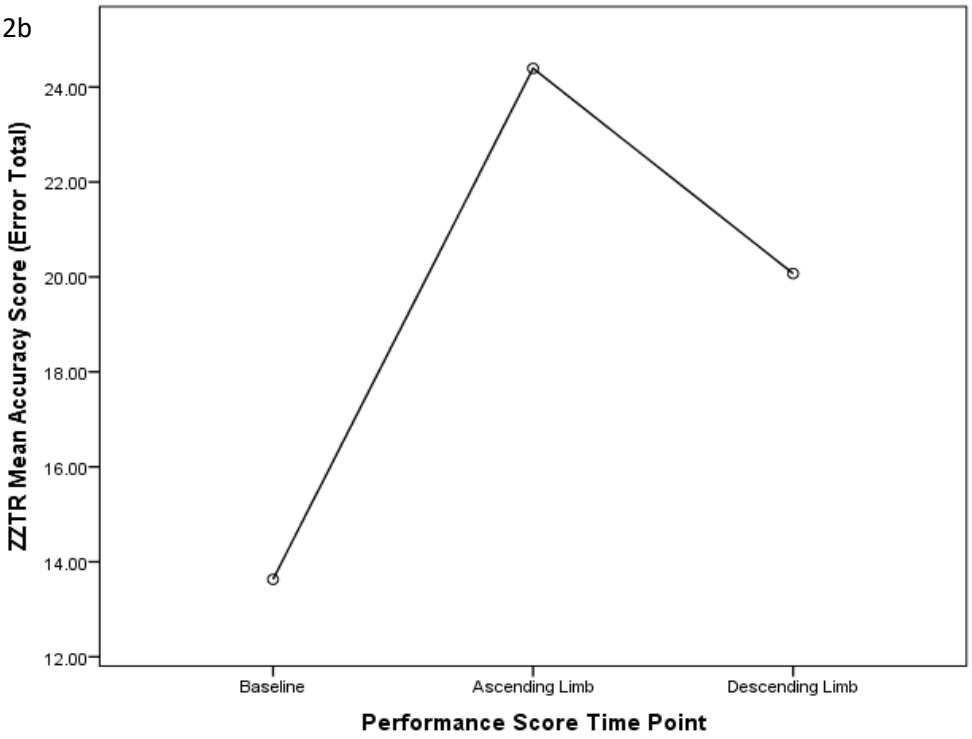


Figure 2: Mean ZZTR Speed Score in Seconds (2a) and Mean ZZTR Total Error Score (2b) at Each Time Point (Baseline, Ascending and Descending Limb).

3.1.4.2 Grooved Pegboard Task (GPB)

Hypothesis two (H2) was also tested using a repeated measures ANOVA to explore psychomotor speed differences (as measured by the GPB) between baseline, ascending and descending limb time points (mean (SD) scores can be seen in *Table 1*). The analysis showed that time taken to complete the GPB task did not significantly differ across these time points, F (2,40) = 2.62, p = .085, ω^2 = .01. However, mean differences between baseline, ascending and descending limb time points were in the anticipated direction and these differences appeared to be approaching statistical significance. Planned contrasts were conducted to explore whether alcohol impaired psychomotor speed (baseline vs ascending limb comparison) and if any impairment recovered on the descending compared to the ascending limb, at comparable BACs. Planned contrasts exploring whether psychomotor speed was impaired by the acute administration of alcohol (baseline vs ascending limb time points) revealed no significant difference, F (1, 20) = 1.05, p = .318, r = .22. This suggests that alcohol did not significantly impair psychomotor speed. Planned contrasts were also used to explore whether psychomotor speed performance increased on the descending compared to the ascending limb of the BAC curve. Analysis showed that participants performed significantly quicker on the descending compared to the ascending limb, F (1, 20) = 5.49, p = .030, r = .46 (this trend can be seen in *Figure 3)*.

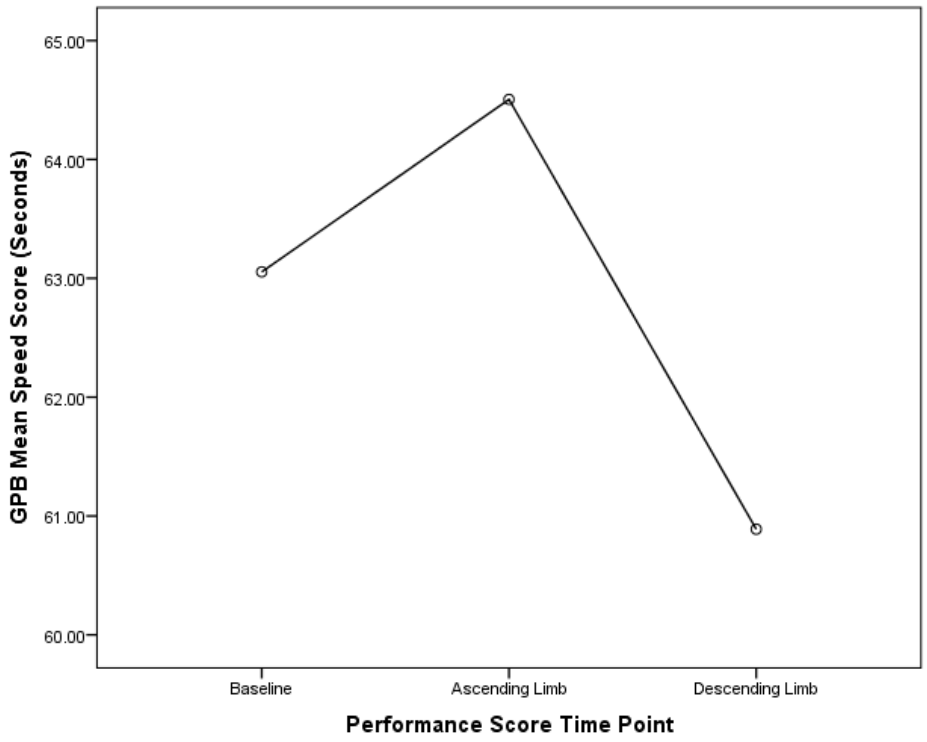


Figure 3: Mean GPB Speed Score in Seconds at Each Time Point (Baseline, Ascending and Descending Limb).

3.1.5 Self-Report Intoxication

3.1.5.1 Subjective Intoxication (SI)

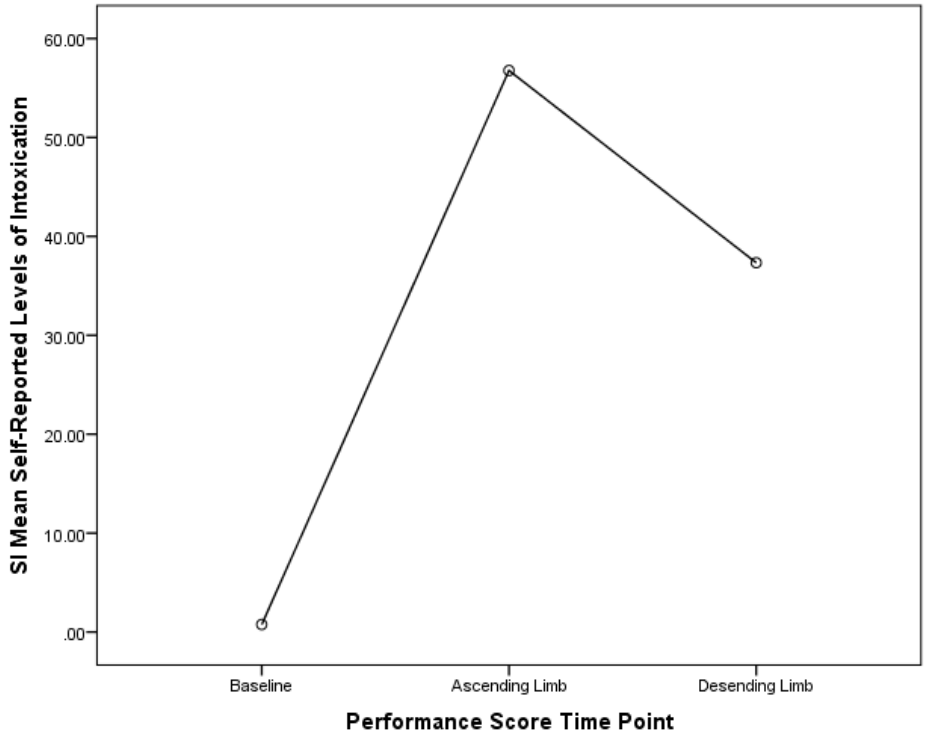
Hypothesis three (H3) was tested using a repeated measure ANOVA to explore whether there were differences between levels of self-reported subjective intoxication (SI) between baseline, ascending and descending limb time points. The analysis showed that there was a significant difference in levels of self-report intoxication across these time points, F (2, 40) = 75.43, p < .001, ω^2 = .64. Mean (SD) SI values can be seen in *Table 1.* Planned contrasts were used to explore whether participants reported feeling more intoxicated on the ascending limb compared to the descending limb and whether they reported feeling less intoxicated on the descending compared to the ascending limb. Participants reported feeling significantly more intoxicated on the ascending limb compared to baseline, F (1, 20) = 179.40, p < .001, r = .95 and significantly less intoxicated on the descending compared to the ascending limb, F (1, 20) = 15.92, p = .001, r = .67 (these trends can be seen in *Figure 4*). This outlines that participants develop acute alcohol tolerance as they report feeling less intoxicated on the descending limb compared to the ascending limb, at comparable BACs.

3.1.5.2 Biphasic Alcohol Effects Scales (BAES)

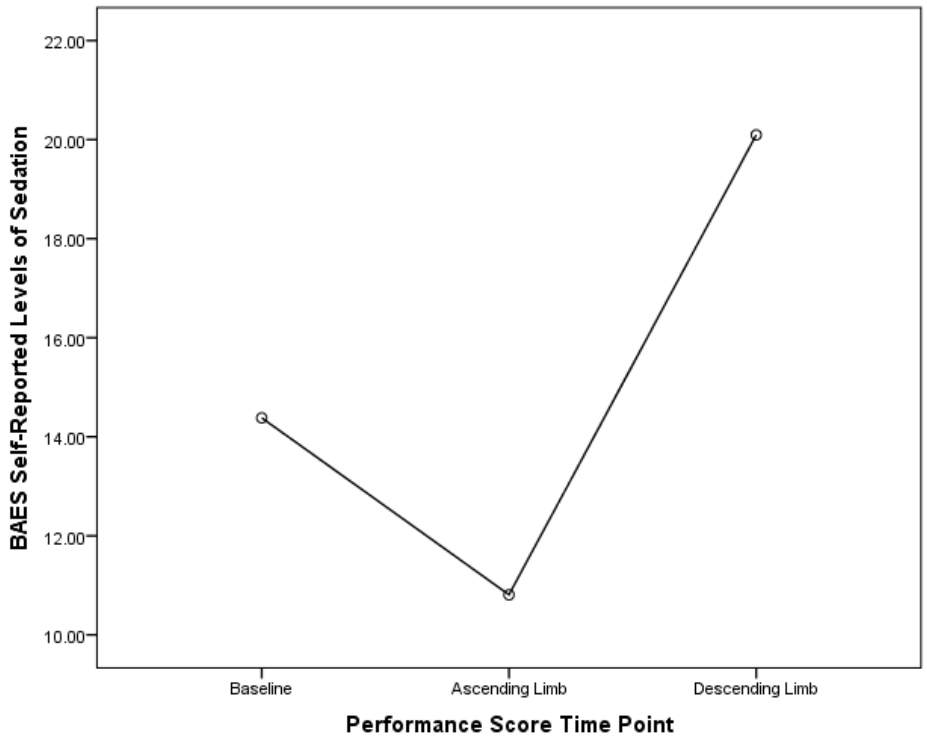
To address hypothesis four (H4), two repeated measures ANOVAs were used to explore whether there was a difference in self-reported levels of alcohol sedation and stimulation (as measured by the BAES) between baseline, ascending and descending limb time points. Analysis showed that self-reported stimulant effects significantly differed between these time points, F (2, 40) = 10.88, p < .001, ω^2 = .17. Planned contrasts were used to explore whether these self-reported stimulant effects differed between baseline and ascending limb time points, as well as ascending and descending limb time points. This showed that participants reported feeling significantly more stimulated on the ascending limb compared to the baseline, F (1, 20) = 18.49, p < .001, r = .69 and reported feeling significantly less stimulated on the descending compared to the ascending limb, F (1, 20) = 19.05, p < .001, r = .70 (these trends can be seen in *Figure 4).* These findings demonstrate that participants develop acute tolerance to self-report levels of alcohol stimulation. Mauchly's test outlined that the assumption of sphericity was violated (see Appendix X) for self-reported sedation across the three aforementioned time points, therefore, Greenhouse-Geisser corrected tests are reported (ε = .67; Appendix 11.3). The ANOVA showed no significant difference in self-reported sedative effects between baseline, ascending and descending limb time points, F (1.35, 26.93) = 3.58, p = .058, ω^2 = .06. As the ANOVA statistic was approaching significance, planned contrast analyses were conducted to explore whether there was a difference in self-reported sedation between baseline and ascending limb time points, as well as between ascending and descending limb time points. This

analysis showed no significant difference between the baseline and ascending limb time points, F (1, 20) = 2.92, p = .103, r = .36 but did show that participants reported feeling significantly more sedated on the descending compared to the ascending limb, F (1, 20) = 6.43, p = .020, r = .49 (trends can be seen in *Figure 4*). This outlines that participants develop acute sensitisation to self-reported sedative effects of alcohol. In sum, these findings demonstrate that self-reported stimulant effects of alcohol decline on the descending compared to the ascending limb (acute tolerance), at comparable BACs, whilst self-reported sedative effects increase (acute sensitisation).

4a



4b



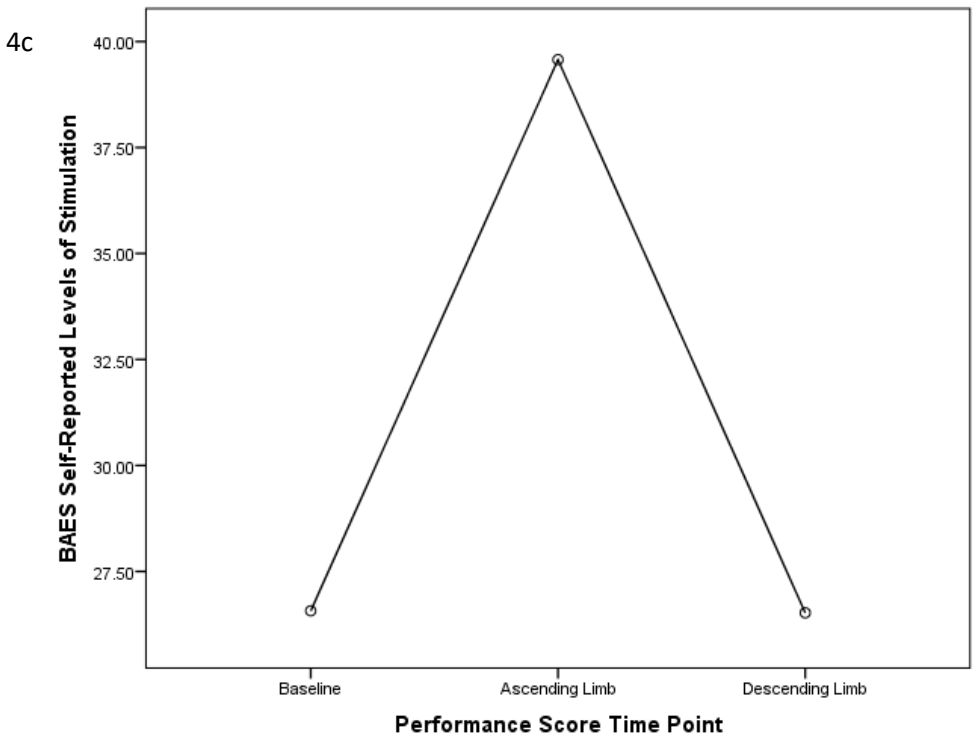


Figure 4: Mean Self-Reported Levels of Intoxication (4a), Mean Self-Reported Levels of Alcohol Sedation (4b) and Mean Self-Reported Levels of Alcohol Stimulation (4c) at Each Time Point (Baseline, Ascending and Descending Limb).

3.2 THE ROLE OF OUTCOME EXPECTANCIES AND THE DEVELOPMENT OF ACUTE ALCOHOL TOLERANCE

3.2.1 STATISTICAL ANALYSIS

To test the fifth hypothesis (H5), a series of multiple regression (MR) analyses were used to examine the degree to which positive and negative alcohol outcomes predict the magnitude of acute tolerance development. The difference between ascending and descending limb scores was calculated and was used as a proxy measure of the magnitude of acute alcohol tolerance. Only measures that demonstrated significant alcohol impairment recovery on the descending compared to the ascending limb were used. A large mean difference between these time points suggests greater tolerance and a smaller difference suggests less tolerance. Regression diagnostics and assumption tests can be seen in *Appendix 12*. The enter method was used meaning positive and negative expectancy scores were simultaneously entered in to each regression model to explore the amount of variance they each explain.

3.2.2 DESCRIPTIVE STATISTICS

Means scores can be seen in *Table 2* for each predictor variable (i.e., positive and negative outcome expectancy scores) and for each outcome variable (i.e., ZZTR speed & accuracy, GPB speed, subjective intoxication and BAES tolerance scores).

Table 2: Mean (SD) scores for each predictor variable (i.e., positive and negative outcome expectancy scores) and each outcome variable (i.e., ZZTR speed & accuracy, GPB speed, subjective intoxication and BAES tolerance scores).

Variable	Mean (Standard Deviation)
Positive Outcome Expectancy	221.24 (42.22)
Negative Outcome Expectancy	114.10 (35.41)
ZZTR Speed (Seconds) Tolerance	8.86 (9.90)
ZZTR Accuracy (Error Total) Tolerance	4.32 (3.95)
GPB Speed Tolerance	3.62 (7.07)
SI Tolerance	19.43 (22.32)
Tolerance to Stimulant Effects	13.05 (13.70)
Sensitisation to Sedative Effects	-9.29 (16.79)*

*NOTE: *Acute sensitisation developed (i.e., the effect increased on the descending compared to the ascending limb of the BAC curve.*

3.2.3 PSYCHOMOTOR PERFORMANCE

3.2.3.1 Zig-Zag Tracking Task (ZZTR)

Multiple regression analyses were used to explore the amount of variance in acute alcohol tolerance development for measures of psychomotor speed and accuracy (as measured by the ZZTR task) that is attributable to self-report positive and negative alcohol outcome expectancies. Results show that these expectancies do not significantly predict the magnitude of acute alcohol tolerance development for measures of psychomotor speed, adjusted R^2 = -.07, F (2, 18) = .35, p = .708. Similarly, these expectancies do not predict the magnitude of acute alcohol tolerance development for measures of psychomotor accuracy, adjusted R^2 = -.02, F (2, 18) = .85, p = .445. These findings suggest that positive and negative outcome expectancies do not predict the magnitude of the acute alcohol tolerance development for measures of psychomotor ability. *Table 3* shows the standardised and

unstandardised regression coefficients for each individual predictor variable (positive and negative expectancy).

Table 3: Linear models of predictors (positive and negative expectancy) of both psychomotor speed and accuracy (ZZTR) tolerance.

	ZZTR Speed (Seconds) Tolerance				ZZTR Accuracy (Error Total) Tolerance			
	b	SE B	β	p	b	SE B	β	p
Positive Outcome Expectancy	-.01	.06	-.03	.917	.00	.02	.01	.956
Negative Outcome Expectancy	-.05	.07	-.18	.496	.03	.03	.29	.271

3.2.3.2 Grooved Pegboard Task (GPB)

A multiple regression analysis was used to explore whether positive and negative alcohol outcome expectancies predict the magnitude of acute alcohol tolerance development with regards to the impairing effects of alcohol on psychomotor speed (as measured by the GPB task). The analysis showed that alcohol outcome expectancies do not significantly predict the magnitude of acute alcohol tolerance development to measures of psychomotor speed, adjusted R^2 = -.40, F (2, 18) = 1.42, p = .268. *Table 4* shows the standardised and unstandardised regression coefficients for each of the individual predictor variables.

Table 4: Linear model of predictors (positive and negative expectancy) of psychomotor speed (GPB) tolerance.

	GPB Speed Tolerance			
	b	SE B	β	p
Positive Outcome Expectancy	.06	.04	.35	.172
Negative Outcome Expectancy	.01	.05	.04	.874

3.2.4 Self-Report Intoxication

3.2.4.1 Subjective Intoxication (SI)

Multiple regression showed that alcohol expectancies do not significantly predict the magnitude of acute alcohol tolerance with respect to alcohol induced subjective intoxication impairment and recovery, adjusted R^2 = -.01, F (2, 18) = .96, p = .402. *Table 5* shows the standardised and unstandardised regression coefficients for each predictor variable.

Table 5: Linear model of predictors (positive and negative expectancy) of self-reported subjective intoxication tolerance.

	SI Tolerance			
	b	SE B	β	p
Positive Outcome Expectancy	.18	.13	.343	.189
Negative Outcome Expectancy	-.13	.16	-.21	.419

3.2.4.2 Biphasic Alcohol Effects Scale

Multiple regression analysis was used to explore whether positive and negative alcohol outcome expectancies predict the magnitude of the development of alcohol tolerance with respect to self-reported alcohol stimulation during a single drinking session. Analysis showed that both positive and negative outcome expectations did not predict this magnitude of tolerance development, adjusted R^2 =-.08, F (2, 18) = .26, p = .771. As well as this, multiple regression was used to explore whether these expectancies (both positive and negative) predict the magnitude of sensitisation to sedation. Analysis similarly showed that the overall model did not achieve statistical significance, adjusted R^2 =.13, F (2, 18) = 2.48, p = .112 (*Table 6* shows the standardised and unstandardised regression coefficients for each predictor variable on each outcome variable). However, an increase in negative outcome expectancies did significantly predict an increase in magnitude of sensitisation to sedation (β = .52, p = .041). This suggests that individuals with greater negative expectations surrounding alcohol consumption will experience greater feeling of sedation on the descending compared to the ascending limb of the BAC (at comparable BACs).

Table 6: Linear models of predictors (positive and negative expectancy) of tolerance to alcohol induced stimulant effects and sensitisation to sedative effects (BAES).

	Tolerance to Stimulant Effects				Sensitisation to Sedative Effects			
	b	SE B	β	p	b	SE B	β	p
Positive Outcome Expectancy	.06	.08	.19	.477	-.12	.09	-.30	.219
Negative Outcome Expectancy	-.03	.10	-.08	.762	.24	.11	.52	.041

Chapter 4: General Discussion

4.1 Alcohol Induced Impairment

The present investigation aimed to firstly test whether measures of behavioural control, psychomotor ability and self-report intoxication demonstrate acute alcohol tolerance within a single session exposure. For tolerance to develop, there first needs to be clear evidence for alcohol impairment (i.e., poorer performance on the ascending limb compared to baseline). Therefore, alcohol induced impairment was first investigated to establish whether task performance across these measures diminished following a moderate dose of alcohol. As expected, results indicate that participants reported feeling significantly more impaired on the ascending limb when compared to baseline measures. This is consistent with past literature (Brumback, Cao and King 2007; Tiplady et al. 2001). It is clear that a moderate dose of alcohol increases subjective ratings of intoxication which may enhance behavioural impairment. Results also indicate that behavioural activation and inhibition were both impaired by the administration of alcohol; ascending limb response activation and inhibition scores were significantly worse than when participants were sober. This replicated the findings of past literature that similarly suggest that acute alcohol administration impairs both aspects of behavioural control (Fillmore, Marczinski and Bowman 2005; Marczinski and Fillmore 2003; Reynolds, Richards and de Wit 2006). Taken together, these findings confirm that both the ability to initiate a behavioural response and the ability to stop this response once it has been initiated are impaired by the consumption of a moderate dose of alcohol. These findings are important as alcohol induced deficits in behavioural control have been implicated in several maladaptive behaviours (Miller and Fillmore 2014). Fillmore (2003) described inhibitory control as a primary mechanism involved in the expression of impulsive behaviour. Therefore, impairment of behavioural control may lead to poor decision making and increase the likelihood of risk taking whilst intoxicated. It is also apparent that frequent acute impairment of impulse control may lead to the development of problematic drinking behaviour (i.e., excessive binge drinking) (Fillmore 2007; Lyvers 2000). In support of this, Marczinski, Combs, and Fillmore (2007) concluded that poor inhibitory control increased alcohol consumption during a binge drinking occasion again emphasising the clear implications inhibitory control impairment has on consumption.

Similarly, ZZTR task performance results indicate that alcohol had an impairing effect on psychomotor performance (speed and accuracy). The findings suggest that psychomotor speed increased on the ascending limb compared to baseline, whilst accuracy decreased; participants were

faster at completing the task but made more errors when intoxicated. Collectively, this suggests that alcohol impairs psychomotor ability by increasing the speed with which participants carry out a cognitive task that involves motor-coordination which in turn results in an increase in errors made. This is somewhat consistent with the past findings of Tiplady et al. (2004) who also used the ZZTR task to measure alcohol induced psychomotor performance impairment. They concluded that alcohol significantly affected psychomotor accuracy by increasing the total amount of errors made but did not affect speed. It is worth noting that although this study (Tiplady et al. 2004) administered higher doses of alcohol (.70g/kg administered to males and .60g/kg to females), the psychomotor task was completed at approximately 75 minutes' post alcohol administration. This may explain why they detected no psychomotor speed impairment as BAC may have already started to decline due to alcohol elimination and the development of acute tolerance.

4.2 Acute Alcohol Tolerance Development

The present investigation aimed to explore whether aspects of behavioural control (activation and inhibition), psychomotor performance (speed and accuracy) and self-reported intoxication develop acute alcohol tolerance following a moderate dose of alcohol. It was hypothesised that acute tolerance would develop to measures of psychomotor performance (H2) and to subjective ratings of intoxication (H3). Results indicate that both psychomotor and accuracy recovered on the descending compared to the ascending limb of the BAC curve. Results also outline that participants reported feeling significantly less intoxicated on the descending compared to the ascending limb and thus demonstrate an acute tolerance effect. These findings offer support to H2 and H3, and successfully replicate past literature that similarly suggest that acute alcohol tolerance develops to psychomotor ability and subjective ratings of intoxication (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012). With regards to behavioural control, there is some degree of discrepancy surrounding the recovery of behavioural activation and inhibition at comparable BACs on the descending compared to the ascending limb of the BAC curve. Acute tolerance investigations that explore this recovery using a BAC curve comparison procedure report improved behavioural activation performance on the descending compared to the ascending limb but no behavioural inhibition impairment recovery (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012). Conversely, investigations using an alcohol clamp procedure (keeping BAC at a constant level) do not demonstrate any behavioural control recovery; both behavioural activation and inhibition remain impaired (Hendershot et al. 2015). It was hypothesised that the findings in the present thesis would replicate past literature using the BAC curve

comparison experimental procedure (H1); behavioural activation would recover whilst inhibition would remain impaired. Descriptive results from the present study suggest that behavioural activation scores improve whilst inhibition scores worsen on the descending compared to the ascending limb of the BAC curve. However, the inferential results from the present investigation outline that these observable differences were not statistically significant meaning that no acute alcohol tolerance nor acute alcohol sensitisation to either measure of behavioural control was demonstrated. These findings lend partial support to H1 and suggest that acute recovery of behavioural control impairment may not occur during a single drinking session and lends support to the conclusions made by Hendershot et al. (2015).

Despite this, there is still a large breadth of acute alcohol tolerance literature using the BAC curve comparison procedure that conclude that behavioural activation but not inhibition develop tolerance within a single session (Fillmore and Weafer 2012; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012). A possible explanation for why the results from the present investigation contradict these conclusions made, surrounds the implementation of the BAC limb comparison procedure. In the previous investigations using the BAC limb comparison procedure, a large task battery (often around 30-minutes in duration) is completed by each participant at approximately 35-65 and 95-125 minutes' post alcohol administration (ascending and descending limb testing phases, respectively). During the ascending limb testing phase, BAC rises due to alcohol absorption and during the descending limb testing phase BAC declines due to elimination (McKim and Hancock 2012). Therefore, theoretically speaking, the task completed at the start of the ascending limb phase at 35 minutes' post alcohol administration would not be completed at a comparable BAC on the descending limb testing phase time point (and vice versa). As a result, the reported behavioural activation recovery on the descending limb may be simply due to the participant being more intoxicated on the ascending compared to the descending limb. To overcome this in the present study, the 30-minute test battery was reversed on the descending limb in an attempt to administer specific tasks (within the test battery) at comparable BACs. The test battery commenced at approximately 20 minutes and concluded at approximately 50 minutes' post alcohol administration (ascending limb testing phase). The test battery commenced again at approximately 70 minutes and concluded at 100 minutes' post alcohol administration (descending limb testing phase). BACs recorded at the start of the ascending limb testing phase and the end of the descending limb testing phase (20 and 100 minutes' post alcohol administration respectively) did not significantly differ. Similarly, BACs recorded at the end of the ascending limb testing phase and the start of the descending limb testing phase (50 and 70 minutes' post alcohol administration, respectively) did not significantly differ. These results

coupled with the reversal of the test battery on the descending limb increase the likelihood of specific tasks within the test battery being completed at comparable BACs.

Despite this procedural adaptation, it is almost impossible to control for the varying speed of alcohol absorption/elimination between participants in the present investigation (and all investigations using the BAC curve comparison paradigm). Therefore, individual differences in the rate of absorption and elimination may limit the conclusion made from the obtained results. Future investigation of acute tolerance should adopt an alcohol time course procedure referred to as a BAC clamp, similar to the procedure outlined by Hendershot et al. (2015). In this study a dose of alcohol was administered via an indwelling catheter and the BAC was pseudo-clamped at 80mg/100ml. It took approximately 20 minutes to achieve a BAC of 80mg/100ml and this level was kept constant for a further 80 minutes (100 minutes in total). Alcohol impairment was measured at 40 and 90 minutes post alcohol infusion which demonstrated acute alcohol tolerance; impairment was significantly reduced at 90 compared to 40 minutes post infusion. This procedural adaptation overcomes the differences in BAC identified between participants due to differing rates of alcohol absorption and elimination on the ascending limb and descending limb.

One consistent agreement between the present study and past acute alcohol tolerance literature is that impaired psychomotor ability and subjective ratings of intoxication recover during a single drinking session, whereas, behavioural inhibition does not. Taken together, this evidence may explain why individuals engage in risk taking and make poor decisions within a drinking session. Continued impairment of inhibition increases the likelihood of individuals demonstrating risk taking as they are less likely to inhibit these behaviours. In conjunction, these individuals report feeling less intoxicated at this time point and have also developed tolerance to the diminishing effects that alcohol has on motor ability, which may increase their capacity to carry out these behaviours. Weafer and Fillmore (2012) support these conclusions, as they identified that individuals demonstrate a greater willingness to drive on the descending limb of the BAC curve. Findings from the present study also suggest why repeated consumption occurs within a single drinking occasion. Due to the prolonged impairment of behavioural control and the recovered self-report intoxication, there is an increased likelihood that individuals will seek more alcohol to achieve the desired effects within a single drinking session. This may explain why individuals continue to consume alcohol and may predict the likelihood of binge drinking being initiated and maintained. Similar evidence shows that individuals self-administer higher quantities of alcohol on the descending limb in comparison to the ascending limb, lending support to the conclusions made in the present study (Hendershot et al. 2015; Weafer and Fillmore 2012). This outlines the importance of understanding the acute development of tolerance

within a single drinking session, and how this relates to the initiation and maintenance of, as well as poor decision making within, a binge drinking occasion.

With regards to the biphasic effects of alcohol, it was hypothesised that self-report stimulant effects would be most dominant on the ascending limb (compared to the descending limb) and sedative effects would be more dominant on the descending limb (compared to the ascending limb)(H4). Stimulant effects appear to be more dominant on the ascending limb of the BAC curve and on the descending limb these stimulant effects significantly diminish. In contrast, the sedative effects of alcohol appear to be more dominant on the descending limb and significantly less so on the ascending limb. These findings lend support to H4. As previously discussed, the results from the present study outline greater alcohol impairment of psychomotor ability and subjective ratings of intoxication on the ascending limb and reduced impairment on the descending limb due to the acute development of tolerance. It would therefore appear that stimulant effects of alcohol are associated with an increase in alcohol impairment and sedative effects are associated with a reduction in impairment. These findings are consistent with past literature that similarly outline this trend (Hendershot et al. 2015). Notably, Morzorati et al. (2002) demonstrated that the sedative effects were dominant for 3 hours post alcohol administration. Collectively, the present study and similar past literature demonstrate a relatively short adaptation to stimulant effects of alcohol and a long sensitisation to the sedative effects. It is therefore likely that these effects play an important role in the recovery from alcohol impairment within a single drinking occasion.

4.3 Alcohol Outcome Expectancy

The present investigation also sought to explore whether positive and negative alcohol outcome expectancies were associated with acute alcohol tolerance development. Specifically, it aimed to identify whether outcome expectancies predict the magnitude of tolerance developed within a single drinking session when a moderate dose of alcohol was administered. Since the present study only identified that tolerance developed to measures of psychomotor ability and self-report subjective intoxication, the role of expectancy was only explored for these measures. It was hypothesised that anticipating negative consequences as a result of consuming alcohol would exert impairment compensation (H5). This conditioned response has been identified to counteract the impairing effects of alcohol (Laberg and Löberg 1989) and so this compensatory effect would in turn increase the amount of tolerance displayed within a single drinking session. However, no significant associations were identified between outcome expectancies and the magnitude of tolerance development. This was observed for both measures of psychomotor ability and subjective intoxication. These results lend

no support to H5 and suggest that following consumption of a moderate dose of alcohol, anticipating negative alcohol related outcomes does not predict greater amounts of acute tolerance to psychomotor ability nor subjective intoxication ratings. Similarly, these results also suggest that expecting positive outcomes does not predict tolerance. However, it was discovered that negative expectancies did predict changes in self-reported feelings of alcohol sedation. As previously mentioned, it was discovered that participants reported greater feelings of sedation on the descending compared to the ascending limb (acute sensitisation). This was found to be enhanced by greater pre-existing negative outcome expectancies (i.e., greater negative expectancies predicted greater sedation sensitisation). This is of particular interest as sedative effects of alcohol are associated with reduced cognitive impairment whilst stimulant effects are associated with increased impairment (Vogel-Sprott and Fillmore 1993). It is therefore likely that increasing negative outcome expectancies in individuals demonstrating problematic drinking behaviours (i.e., binge drinking) would increase the development of sensitisation to these sedative effects and consequentially reduce alcohol induced impairment at a faster rate.

As the acute tolerance development for measures of psychomotor performance and subjective intoxication was found to not be influenced by outcome expectancies, it is important to consider potential explanations for this. In the present study, positive and negative expectancies were represented by self-reported scores on the AEQ-III and NAEQ. Data was collected using these measures after the initial acute alcohol tolerance investigation concluded. This was due to the fact that it was later decided that prior expectations were likely to influence the development of acute tolerance as they have been previously found to exert compensatory effects (Testa et al. 2006). This approach was considered appropriate, as positive and negative outcome expectancies are beliefs individuals develop surrounding the likely outcomes of their drinking behaviour. These beliefs become internalised and remain constant over time (Reich, Below and Goldman 2010). Therefore, collecting this additional data retrospectively is unlikely to have influenced results. However, these expectancy measures depend on participants self-reflecting on these past drinking experiences and their prior exposure to alcohol. As all participants involved in the present investigation were regular social drinkers, there may be a lack of variability in the types of outcome expectancies held. This potentially limiting factor could be overcome by experimentally manipulating expectation. Past literature has identified that when positive outcome expectancies are primed, participants consume more alcohol than when negative expectancies are primed (Carter et al. 1998). By priming these beliefs, this study was able to establish the amount of variability in alcohol consumption that is attributable to positive expectancies and the amount attributable to negative expectancies. Future studies surrounding acute

alcohol tolerance and the influence of outcome expectancies could prime these anticipated outcomes and explore whether a difference in magnitude of tolerance is apparent.

A second possible limiting factor not considered by the present investigation, surrounds the association between prior drinking behaviour and outcome expectancies. As previously mentioned, participants in the present investigation shared similar drinking behaviours and reported consuming similar quantities of alcohol per occasion. These individuals were classified as regular alcohol consumers and are likely to share similar outcome expectancies. Individuals classified as being alcohol dependent have been identified to have greater negative expectancies compared to regular non-problematic drinkers (Finn et al. 2005). This suggests that type of drinking behaviour predicts the type of outcome expectancies held by an individual. It has also been identified that problematic drinkers (classified as 'heavy episodic drinkers') demonstrate greater cross-sessional tolerance (Brumback, Cao and King 2007) and greater acute tolerance (Fillmore and Weafer 2012). It is therefore possible that alcohol dependent individuals that expect greater negative consequence as a result of consuming alcohol will develop greater acute alcohol tolerance. However, due to ethical considerations, it was not possible to include alcohol dependent individuals in the present investigation. Future research could explore these associations by including both non-problematic and problematic drinkers. This would establish the extent to which prior drinking experience and behaviour predicts the specific type of expectation held as well as exploring the extent to which these expectations predict the magnitude of acute tolerance development.

Another possible limitation surrounds the conditioned compensatory response model and the experimental manipulation used in the present study. As previously mentioned, the compensatory response model suggests that anticipating alcohol impairment results in greater impairment compensation (Testa et al. 2006). Testa et al. (2006) also discuss balanced-placebo manipulation studies that administer both alcohol and a placebo in addition to instructing participants that they are in fact receiving alcohol or are receiving a placebo. This experimental manipulation allows the researcher to compare the effects of alcohol that are a combination of expectation and pharmacology (receive alcohol and expect alcohol condition), purely pharmacological (receive alcohol and not expect alcohol) and purely expectation (receive placebo and expect alcohol). Research supporting the conditioned compensatory model describes that when individuals expect to receive alcohol compared to when they do not, they compensate for the anticipated impairing effects (Newlin 1986). All participants in the present study received a moderate dose of alcohol and were expecting to receive alcohol and so only the collective influence of expectancy and the pharmacology of alcohol could be explored. Due to this, it was difficult to distinguish whether the anticipation of alcohol impairment, whether it be positive or negative expectations, influenced the acute development of tolerance.

Therefore, the lack of a placebo controlled condition limits the results of the present study as the effects of expectation alone on the development of acute alcohol tolerance could not be fully identified. This limitation could be addressed in future research by experimentally manipulating expectancy using a balanced placebo design to explore the influence it has on tolerance development within a single drinking session. It is clear from the finding of the present study and the findings of similar past literature (Fillmore and Weafer 2012; Hendershot et al. 2015; Miller and Fillmore 2014; Ostling and Fillmore 2010; Weafer and Fillmore 2012), that administering alcohol and instructing the participants to expect alcohol results in the acute development of tolerance. What is not clear is the influence the expectation of alcohol is having on the development of this alcohol tolerance. The balanced placebo design would allow for a thorough investigation into the amount of tolerance explained by the pharmacology of alcohol and the amount explained by expectation. This would clarify the specific role expectation plays in the development of acute alcohol tolerance.

4.4 FUTURE RESEARCH

In the present thesis, the role that positive and negative outcome expectancies play in the development of acute alcohol tolerance was explored. It was identified that self-reported alcohol outcome expectations (positive and negative) did not predict the magnitude of alcohol impairment recovery within a single drinking session across all measures that demonstrated this tolerance. It must therefore be concluded that there is no association between pre-existing alcohol outcome expectations and acute tolerance development. To confirm this conclusion, future research should implement the balanced placebo design (Testa et al. 2006). As discussed above, this would make it possible to identify the influence of both alcohol expectation and pharmacology on the development of acute tolerance, individually as well as collectively. This would allow the specific influence that each factor has to be confirmed. Future research could also address the issue surrounding shared expectations due to similar alcohol experience and drinking behaviours. The majority of participants used in this thesis were undergraduate students that were likely to share similar positive expectancies due to their shared drinking experience. To address this issue positive and negative outcome expectations could be primed. Carter et al. (1998) identified that when positive outcome expectancies are primed, participants consume more alcohol than when negative expectancies are primed. Therefore, by manipulating the type of outcome expectancies individuals have, their influence on tolerance could be explored.

Acute tolerance research to date, including the present investigation, has only focused on inhibitory mechanisms associated with one aspect of behavioural control (the ability to inhibit

behavioural responses). What is not clear are the disruptive effects of alcohol on inhibitory mechanisms associated with attention orientation on the ascending compared to the descending limb. Abroms and Fillmore (2004) investigated the acute effects of alcohol on selective attention, specifically the efficiency of visual search. Visual orientation efficiency to new visual stimuli is reliant on the inhibition of return (IOR) mechanism that prevents attention returning to a previously attended to stimuli. Results from this study indicated that a moderate dose of alcohol impairs the IOR mechanism by shortening its duration of influence (on the ascending limb), which allows attention to return to a previous location. This ultimately reduces the efficiency of visual search. In the context of alcohol consumption and consequential behaviour, a reduction in visual search efficiency may lead to the increased likelihood of negative consequence. In support of this, Olthuis and Klein (2012) similarly conclude that alcohol consumption impairs visual search efficiency. They suggest that failure to inhibit attention returning to previously attended to irrelevant information reduces the ability to complete higher order, behavioural tasks, such as driving a car. In this drink driving example, impaired visual search efficiency would result in the driver missing key and relevant information (e.g., brake lights) whilst returning to previously attended to irrelevant information. Understanding alcohol induced impairment of inhibition mechanisms associated with attentional orientation are of apparent importance and have clear real world implications. What remains unclear is whether the diminished IOR effect on the ascending limb continues to be impaired at a comparable BAC on the descending limb, similar to behavioural inhibition. Future research should test this by comparing attentional orientation and visual search on the ascending and descending limbs of the intoxication. Since it has been established that behavioural inhibition remains impaired on the descending limb, it would be interesting to explore whether IOR impairment similarly remains impaired on the descending compared to the ascending limb (at comparable BACs) or whether impairment recovers. It is likely that IOR performance will remain impaired as many cognitive and perceptual processes, such as inhibitory influences, operate in a similar manner (Barkley 1997; McClelland and Rumelhart 1981).

Attentional bias has received a lot of research attention and has also been found to promote alcohol consumption. This theory suggests that the desire and motivation to consume alcohol becomes paired with alcohol cues resulting in the cues alone triggering the alcohol seeking behaviour (i.e., *incentive sensitization theory*; Robinson and Berridge 2001). A wealth of evidence has demonstrated that, in sober individuals, increased attention to alcohol cues is associated with problematic consumption and increased alcohol seeking behaviours (Fadardi and Cox 2008; Field and Eastwood 2005); these cues may contribute to the initiation of a binge-drinking occasion. Weafer and Fillmore (2012) tested the magnitude of alcohol cue attentional bias in sober individuals and again following the acute administration of alcohol, as expected, and similar to past literature, the authors

found that sober individuals displayed greater bias to alcohol stimuli. However, following the administration of alcohol this bias was diminished in heavy drinkers. One possible explanation for this is that once drinking has been initiated the alcohol seeking behaviour and the resulting motivation to consume alcohol has been rewarded which results in the decrease in attentional orientation to alcohol stimuli. Again, this effect was demonstrated on the ascending limb of the BAC intoxication curve. Future investigations could determine whether the diminished attentional bias demonstrated on the ascending limb recovers on the descending limb due to acute tolerance. If attentional bias recovers in this manner, heavy drinkers will show an increase in preference for alcohol cues on the descending limb, which would in turn re-initiate alcohol consumption within the single session. This exploration would offer an insight as to how attentional orientation during a drinking session might contribute to repeated alcohol consumption and what ultimately contributes to binge-drinking behaviour.

4.5 CONCLUSION

The present thesis explored whether alcohol induced impairment of behavioural control (both activation and inhibition of a response) recovered on the descending limb compared to the ascending limb at comparable BACs (acute tolerance development). It also sought to confirm whether this acute tolerance development was consistent with past literature that demonstrated impairment recovery to measures of psychomotor ability and subjective intoxication. It can be concluded that acute alcohol tolerance developed to measures of psychomotor ability, both speed and accuracy, and subjective intoxication. These findings were consistent with past acute alcohol tolerance literature. With regards to both response activation and inhibition impairment, no acute alcohol tolerance was observed. This prolonged impairment of behavioural inhibition on the descending limb was also consistent with past literature. However, the prolonged impairment of behavioural activation and therefore the lack of acute tolerance development observed in the present study contradicts findings of past literature adopting a similar BAC curve comparison procedure. This observed discrepancy could be a result of between subject differences in past alcohol consumption behaviour, rate of alcohol absorption/elimination and methodological flaws (comparing ascending and descending limb performance using the BAC curve procedure). Future investigations should explore acute tolerance development using an alcohol clamp procedure to overcome these limitations.

The present thesis also aimed to address whether pre-existing alcohol outcome expectancies influenced the development of acute alcohol tolerance. It was found that the magnitude of tolerance development for measures of subjective intoxication and psychomotor performance was not significantly predicted by positive and negative outcome expectancies. It must therefore be concluded

that outcome expectancies do not exert influence on the development of acute alcohol tolerance. However, negative outcome expectancies were found to predict the magnitude of sedation sensitisation (i.e., the increased experience of sedation on the descending compared to the ascending limb, at comparable BACs). Negative beliefs surrounding the likely outcomes of consuming alcohol predicted greater acute sensitisation to the sedative effects of alcohol on the descending compared to the ascending limb, at comparable BACs. This is of particular interest as increasing negative outcome expectancies in individuals demonstrating problematic drinking behaviours (i.e. binge drinking) should therefore increase the development of sensitisation to these sedative effects. Coupled with the fact that sedative effects of alcohol have been found to be associated with less cognitive impairment, this may result in reducing alcohol induced impairment at a faster rate.

With regards to implications, the present thesis does offer some insight as to how a binge drinking session may be initiated and maintained as well as explaining why risk taking may occur within the drinking session. The prolonged impairment of behavioural inhibition, coupled with the acute recovery of subjective intoxication and motor performance impairment, suggests that individuals are less likely to inhibit a particular behaviour and have the capacity to carry out this behaviour. In the context of a drinking session, this could mean that individuals are more likely to initiate further alcohol consumption as a result of feeling less intoxicated (due to tolerance) whilst their ability to inhibit this consumption behaviour remains impaired. This could also explain why individuals make poor choices and engage in risk taking during the drinking occasion. By understanding the specific mechanisms that function to promote binge drinking and poor decision making, it is possible that interventions could be developed to reduce these problematic drinking behaviours. Web-based inhibitory control training has been found to reduce alcohol consumption (Jones et al. 2014). Similar training could be adapted to address the issue associated with prolonged impulse control during a single drinking session. To date, alcohol research has primarily focused on behavioural adaptation during a single drinking session. Future research could explore whether acute tolerance develops to attentional orientation. Specifically, research could determine whether a preference for alcohol cues is apparent on both the ascending and descending limb of the BAC curve. This would develop a better understanding of the complex interplay between tolerance, expectation, behavioural control and attentional orientation and the subsequent influence on binge-drinking and decision making.

To conclude, the present thesis offers a useful insight surrounding the complex mechanisms that may function to promote alcohol consumption within a single drinking session and influence intoxicated behaviour. By using an adapted version of the BAC curve procedure, it was discovered that tolerance develops to measures of psychomotor ability and subjective intoxication but not to any aspect of behavioural control (response activation and inhibition) during a single drinking occasion.

This replicates a wealth of literature that concludes that behavioural inhibition does not recover on the descending limb of the BAC curve, but psychomotor performance and subjective ratings of intoxication do. Taken together, this is likely to promote alcohol consumption and risk taking due to the lack of impulse control paired with the recovery of motor ability and beliefs surrounding intoxication. Findings surrounding acute alcohol tolerance development and behavioural activation are inconsistent. Future research should consider using a blood alcohol clamp paradigm to test this discrepancy. Conclusions can also be drawn surrounding the influence alcohol outcome expectancies have on the development of acute tolerance. This thesis concludes that pre-existing expectancies exert no influence on the development of tolerance. However, further research adopting a balanced placebo design is needed to test these conclusions.

References

Abroms, B.D. and Fillmore, M.T. (2004) 'Alcohol-Induced Impairment of Inhibitory Mechanisms Involved in Visual Search.' *Experimental and Clinical Psychopharmacology* 12 (4), 243–250

Abroms, B.D., Fillmore, M.T. and Marczinski, C.A. (2003) 'Alcohol-induced impairment of behavioral control: effects on alteration and suppression of prepotent responses.' *Journal of Studies on Alcohol and Drugs* 64:687–695

Ally, A.K., Lovatt, M., Meier, P.S., Brennan, A. and Holmes, J. (2016) 'Developing a social practice-based typology of British drinking culture in 2009–2011: implications for alcohol policy analysis' *Addiction* 111 (9), 1568-1579

American Psychiatric Association (2000) *Diagnostic and Statistical Manual of Mental Disorders, 4th edn, text revision.* Washington, DC: American Psychiatric Association

American Psychiatric Association. (2013) *Diagnostic and statistical manual of mental disorders (5th ed.).* Arlington, VA: American Psychiatric Publishing

Amodeo, M. and Kurtz, N. (1990) 'Cognitive processes and abstinence in a treated alcoholic population.' *International Journal of the Addictions* 25: 983–1009

Babor, T.F., Kranzler, H.R. and Lauerman, R.J. (1989) 'Early detection of harmful alcohol consumption: Comparison of clinical, laboratory, and self-report screening procedures'. *Addictive Behaviors* 14, 139-157

Bandura, A. (1977) *Social Learning Theory*. New York: General Learning Press

Barkley, R. A. (1997) 'Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD.' *Psychological Bulletin* 121, 61–94

Barnard, E.A., Skolnick, P., Olsen, R.W., Mohler, H., Sieghart, W., Biggio, G., Braestrup, C., Bateson, A.N. and Langer, S.Z. (1998) 'International Union of Pharmacology. XV. Subtypes of gamma-aminobutyric acidA receptors: classification on the basis of subunit structure and receptor function' *Pharmacological Rev*iews 50(2), 291-313

Barry, H. (1973) 'Motivational and cognitive effects of alcohol'. *Journal of Safety Research* 200–221

Beaglehole, R. and Bonita, R. (2009) 'Alcohol: A Global Health Priority.' *The Lancet* 373(9682): 2173–2174

Beirness, D. and Vogel-Sprott, M. (1984) 'The development of alcohol tolerance: acute recovery as a predictor'. *Psychopharmacology* 85, 398-401

Blume, A. W. and Guttu, B. L. (2015) 'Categories of alcohol outcome expectancies and their relationships to alcohol related consequences'. *Addictive Behaviors Reports* 1, 64–67

Blume, A. W., Schmaling, K. B., and Marlatt, A. G. (2003) 'Predictors of change in binge drinking over a 3-month period' *Addictive Behaviors* 28 (5), 1007–1012

Blume, A.W., Schmaling, K.B. and Marlatt, G.A. (2005) 'Memory, executive function, and readiness to change drinking behavior'. *Addictive Behaviors* 30, 301-314

Bond, A. and Lader, M. (1974) 'The use of analogue scales in rating subjective feelings.' *British Journal of Medical Psychology* 47, 211 – 218

Bond, J. C., Roberts, S. C., Greenfield, T. K., Korcha, R., Yu, Y., and Nayak, M. B. (2010) 'Gender differences in public and pri- vate drinking contexts: A multi-level GENACIS analysis.' *International Journal of Environmental Research and Public Health*, 7, 2136–2160

Borjesson, W. I., and Dunn, M. E. (2001) 'Alcohol expectancies of women and men in relation to alcohol use and perceptions of the effects of alcohol on the opposite sex.' *Addictive Behaviors*, 26, 707–719

Borsari, B. (2004) 'Drinking games in the college environment: A review.' *Journal of Alcohol and Drug Education*, 48, 29–51

Borsari, B., Boyle, K. E., Hustad, J. T. P., Barnett, N. P., O'Leary Tevyaw, T., and Kahler, C. W. (2007) 'Drinking before drinking: Pregaming and in drinking games in mandated students.' *Addictive Behaviors* 32, 2694–2705

Brown, S.A., Christiansen, B.A. and Goldman, M.S. (1987) 'The alcohol expectancy questionnaire: An instrument for the assessment of adolescent and adult alcohol expectancies.' *Journal of Studies on Alcohol* 48 (5), 483–491

Brumback, T., Cao, D. and King, A. (2007) 'Effects of alcohol on psychomotor performance and perceived impairment in heavy binge social drinkers'. *Drug and Alcohol Dependence* 91 (1), 10-17

Carey, K.B., Carey, M.P. and Chandra, P.S. (2003) 'Psychometric evaluation of the alcohol use disorders identification test and short drug abuse screening test with psychiatric patients in India'. *Journal of Clinical Psychiatry* 64, 767-774

Carter, J.A., McNair, L.D., Corbin, W.R. and Black, D.H. (1998) 'Effects of priming positive and negative outcomes on drinking responses.' *Experimental and Clinical Psychopharmacology* 6, 399-405

Causby, R., Reed, L., McDonnell, M., and Hillier, S. (2014) 'Use of Objective Psychomotor Tests in Health Professionals'. *Perceptual and Motor Skills* 118 (3), 765-804

Cole-Harding, S. and Michels, V. (2006) 'Does expectancy affect alcohol absorption?'. *Addictive behaviors* 32 (1), 194–8

Cook, R. D., and Weisberg, S. (1982) *Residuals and influence in regression*. New York, NY: Chapman and Hall

Corbin, W. R., Gearhardt, A., and Fromme, K. (2008) 'Stimulant alcohol effects prime within session drinking behavior'. *Psychopharmacology* 197 (2), 327–337

Corbin, W.R., Scott, C., Boyd, S.J., Menary, K.R. and Enders, C.K. (2015) 'Contextual Influences on Subjective and Behavioral Responses to Alcohol'. *Experimental and Clinical Psychopharmacology* 23 (1), 59-70

Courtney, K.E. and Polich, J. (2009) 'Binge Drinking in Young Adults: Data, Definitions, and Determinants.' *Psychological Bulletin* 135(1): 142–156

Crego, A., Holguin, S.R., Parada, M., Mota, N., Corral, M. and Cadaveira, F. (2009) 'Binge drinking affects attentional and visual working memory processing in young university students.' *Alcohol Clinical Experimental Research* 33 (11), 1-10

Davies, M. (2003) 'The role of GABAA receptors in mediating the effects of alcohol in the central nervous system.' *Journal of Psychiatry and Neuroscience* 28(4), 263-274

de Meneses-Gaya, C., Zuardi, A.W., Loureiro, S.R. and Crippa, J.A.S. (2009) 'Alcohol use Disorders Identification Test (AUDIT): An Updated Systematic Review of Psychometric Properties'. *Psychology and Neuroscience* 2 (1), 83-97

Department of Health (2013) Department of Health Reference Costs 2012-13. Available at <https://www.gov.uk/government/uploads/system/uploads/attachment-data/file/261154/nhs-reference-costs-2012-13-acc.pdf>

Derby, D.C. (2011) 'Men's alcohol expectancies at selected community colleges.' *Community College Journal of Research and Practice* 35: 791–801

Derefinko, K., Adams, Z., Milich, R., Fillmore, M., Lorch, E. and Lynam, D. (2008) "Response Style Differences In The Inattentive And Combined Subtypes Of Attention-Deficit/Hyperactivity Disorder". *Journal of Abnormal Child Psychology 36* (5), 745-758

Drinkaware (2017) *What is an alcohol unit?*. [online] Available at: <https://www.drinkaware.co.uk/alcohol-facts/alcoholic-drinks-units/what-is-an-alcohol-unit/>

Dry, M.J., Burns, N.R., Nettelbeck, T., Farquharson, A.L. and White, J.M. (2012) 'Dose-Related Effects of Alcohol on Cognitive Functioning'. *PLoS ONE* 7 (11)

Dunn, M. E. and Goldman, M. S. (1998) 'Age and drinking-related differences in the memory organiza- tion of alcohol expectancies in 3rd-, 6th-, 9th-, and 12th-grade children' *Journal of Consulting and Clinical Psychology* 66, 579–58

Dunne, E. M. and Katz, E. C. (2015) 'Alcohol outcome expectancies and regrettable drinking-related social behaviors'. *Alcohol and Alcoholism* 50 (4), 393–398

Dybek, I., Bischof, G., Grothues, J., Reinhardt, S., Meyer, C., Hapke, U., John, U., Broocks, A., Hohagen, C., and Rumpf, H.J. (2006) 'The Reliability and Validity of the Alcohol Use Disorders Identification Test (AUDIT) in a German General Practice Population Sample'. *Journal of Studies on Alcohol* 67, 473-481

Erblich, J., Earleywine, M., Erblich, B., and Bovbjerg, D.H. (2003) 'Biphasic stimulant and sedative effects of ethanol: Are children of alcoholics really different?' *Addictive Behaviors 28,* 1129–1139

Fadardi, J. S., and Cox, W. M. (2008) 'Alcohol-attentional bias and motivational structure as independent predictors of social drinkers' alcohol consumption' *Drug and Alcohol Dependence* 97, 247-256

Field, A. P. (2013) *Discovering statistics using IBM SPSS statistics and sex and drugs and rock 'n' roll*. 4th edn. Thousand Oaks: Sage Publications

Field, M., and Eastwood, B. (2005) 'Experimental manipulation of attentional bias increases the motivation to drink alcohol.' *Psychopharmacology* 183, 350-357.

Fillmore, M.T. (2003) 'Drug abuse as a problem of impaired control: current approaches and findings.' *Behavioral and Cognitive Neuroscience Reviews* 2, 179-197

Fillmore, M.T. (2007) "Acute Alcohol-Induced Impairment Of Cognitive Functions: Past And Present Findings". *International Journal on Disability and Human Development* 6 (2)

Fillmore, M.T., Blackburn, J.S. and Harrison, E.L. (2008) 'Acute disinhibiting effects of alcohol as a factor in risky driving behavior'. *Drug and Alcohol Dependence* 95, 97-106

Fillmore, M.T., Marczinski, C.A. and Bowman, A.M. (2005) 'Acute Tolerance to Alcohol Effects on Inhibitory and Activational Mechanisms of Behavioral Control'. *Journal of Studies on Alcohol and Drugs* 66 (5), 663-672

Fillmore, M. and Rush, C. (2005) "Polydrug Abusers Display Impaired Discrimination-Reversal Learning In A Model Of Behavioural Control". *Journal of Psychopharmacology* 20 (1), 24-32

Fillmore, M.T. and Weafer, J. (2012) ‘Acute Tolerance to Alcohol in At-risk Binge Drinkers’. *Psychology of Addictive Behaviors* 26 (4), 693-702

Finn, P. R., Bobova, L., Wehner, E., Fargo, S., and Rickert, M. E. (2005) ‘Alcohol expectancies, conduct disorder and early-onset alcoholism: Negative alcohol expectancies are associated with less drinking in non-impulsive versus impulsive subjects.’ *Addiction* 100 (7), 953-962

Finnigan, F., Hammersley, R. and Cooper, T. (1998) ‘An examination of next-day hangover effects after a 100 mg/100 ml dose of alcohol in heavy social drinkers’. *Addiction* 93, 1829-1838

Foster, J. and Ferguson, C. (2013) "Alcohol 'Pre-Loading': A Review Of The Literature". *Alcohol and Alcoholism* 49 (2), 213-226

Francesconi, M. and James, J. (2015) *The Cost of Binge Drinking* [online] Bath Economics Research Papers: Bath University, Department of Economics. available from <http://www.bath.ac.uk/economics/research/working-papers/2015-papers/cost-binge-drinking.pdf> [20 April 2016]

Frezza, M., Padova, di, Pozzato, G., Terpin, M., Baraona, E., and Lieber, C. (1990) 'High blood alcohol levels in women. The role of decreased gastric alcohol dehydrogenase activity and first-pass metabolism'. *The New England Journal of Medicine* 322 (2), 95–9

Friedman, R., McCarthy, D., Pedersen, S. and Hicks, J. (2009). ‘Alcohol expectancy priming and drinking behavior: The role of compatibility between prime and expectancy content.’ *Psychology of Addictive Behaviors*, 23(2), pp.329-333.

Fromme, K., and D’Amico, E. J. (2000) ‘Measuring adolescent alcohol outcome expectancies.’ *Psychology of Addictive Behaviors*, 14, 206–212

Fromme, K., Stroot, E.A. and Kaplan, D. (1993) ‘Comprehensive effects of alcohol: development and psychometric assessment of a new expectancy questionnaire.’ *Psychological Assessment* 5: 19–26

Goldman, M. S., Del Boca, F. and Darkes, J. (1999) Alcohol expectancy theory. In K. E. Leonard and H. T. Blane (eds) Psychological theories of drinking and alcoholism, 2nd ed Guilford NY 203-246

Goldman, M.S., Brown, S.A., Christiansen, B.A., and Smith, G.T. (1991) ‘Alcoholism and memory: Broadening the scope of alcohol expectancy research.’ *Psychological Bulletin*, 110, 137–146

Greenfield, T. K., Harford, T. C., and Tam, T. W. (2009) ‘Modeling cogni- tive influences on drinking and alcohol problems.’ *Journal of Studies on Alcohol and Drugs*, 70, 78–86

Health and Social Care Information Centre UK (2013) Statistics on Alcohol: England, 2013[online] available from <http://www.hscic.gov.uk/catalogue/PUB10932/alc-eng-2013-rep.pdf> [1 February 2016]

Hendershot, C. S., Wardell, J. D., Strang, N. M., Markovich, M. S. D., Claus, E. D., and Ramchandani, V. A. (2015) 'Application of an alcohol clamp paradigm to examine inhibitory control, subjective responses, and acute tolerance in late adolescence' *Experimental and Clinical Psychopharmacology* 23 (3), 147–158

Hendler, R., Ramchandani, V., Gilman, J., and Hommer, D. (2013) 'Stimulant and sedative effects of alcohol'. *Current Topics in Behavioral Neurosciences* 13, 489–509

Hiltunen, A.J. (1997a) ‘Acute alcohol tolerance in cognitive and psychomotor performance: influence of the alcohol dose and prior alcohol experience.’ *Alcohol* 14(2), 125–130

Hiltunen, A. J. (1997b) 'Acute Alcohol Tolerance in Social Drinkers: Changes in Subjective Effects Dependent on the Alcohol Dose and Prior Alcohol Experience'. *Alcohol* 14 (4), 373-378

Hingson, R.W., Zha, W. and Weitzman, E.R. (2009) 'Magnitude of and trends in alcohol-related mortality and morbidity among U.S. college students ages 18-24, 1998-2005.' *Journal of Studies on Alcohol and Drugs* 16, 12-20

Holdstock, L., King, A.C. and de Wit, H. (2000). 'Subjective and objective responses to ethanol in moderate/heavy and light social drinkers'. *Alcoholism: Clinical and Experimental Research* 24, 789-794

Home Office (2012) The Government's Alcohol Strategy [online] available from <https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/224075/alcohol-strategy.pdf> [3 February 2016]

Hughes, K., Quigg, Z., Bellis, M.A., van Hasselt, N., Calafat, A., Kosir, M., Juan, M., Duch, M., and Voorham, L. (2011) 'Drinking Behaviours and Blood Alcohol Concentration in Four European Drinking Environments: A Cross-Sectional Study'. *BMC Public Health* 11 (1), 918

Jones, A.W. (1990) 'Dilemma of a constant blood/breath ratio of alcohol in chemical test evidence of intoxication'. in *Proceedings of the 11th International Conference on Alcohol, Drugs and Traffic Safety*. by Perrine, M. (ed.) Chicago: National Safety Council

Jones, B. T., Corbin, W., and Fromme, K. (2001) 'A review of expectancy theory and alcohol consumption'. *Addiction* 96 (1), 57–72

Jones, A., McGrath, E., Houben, K., Nederkoorn, C., Robinson, E. and Field, M. (2014) 'A Comparison of Three Types Of Web-Based Inhibition Training For The Reduction Of Alcohol Consumption In Problem Drinkers: Study Protocol'. *BMC Public Health* 14 (1)

Keselman, H. (1975) 'A Monte Carlo investigation of three estimates of treatment magnitude: Epsilon squared, eta squared, and omega squared.' *Canadian Psychological Review*, 16, 44–48

King, A. C., Houle, T., de Wit, H., Holdstock, L. and Schuster, A. (2002) 'Biphasic alcohol response differs in heavy versus light drinkers'. *Alcoholism: Clinical and Experimental Research, 26,* 827–835

Klein, R.M. (2000) 'Inhibition of return.' *Trends in Cognitive Science* 4, 138 –147

Klove, H. (1963). Clinical neuropsychology. In F. M. Foster (Ed.), The medical clinics of North America. New York, NY: Saunders.

Kruisselbrink, L.D., Martin, K.L., Megeney, M., Fowles, J.R. and Murphy, R.J. (2006) 'Physical and psychomotor functioning of females the morning after consuming low to moderate quantities of beer'. *Journal of Studies on Alcohol* 67, 416-420

Laberg, J. and Löberg, T. (1989) 'Expectancy and tolerance: A study of acute alcohol intoxication using the balanced placebo design'. *Journal of Studies on Alcohol* 50 (5), 448–455

LaBrie, J.W., Grant, S. and Hummer, J.F. (2011) '"This would be better drunk": alcohol expectancies become more positive while drinking in the college social environment.' *Addictive Behaviors* 36:890–893

Lafayette Instruments. (1989). Grooved pegboard test user instructions. Lafayette, IN: Lafayette Instrument Company, Inc.

Lee, N. K., Greely, J., and Oei, T. P. (1999) 'The relationship of positive and negative alcohol expectancies to patterns of consumption of alcohol in social drinkers.' *Addictive Behaviors*, 24, 359–369

Leeman, R.F., Toll, B.A., Taylor, L.A., and Volpicelli, J.R. (2009) 'Alcohol-induced disinhibition expectancies and impaired control as protective predictors of problem drinking in undergraduates.' *Psychology of Addictive Behaviors* 23(4), 553–563

Levine, A., Miller, E. N., Becker, I. T., Selnes, O. A., and Cohen, B. A. (2004). 'Normative data for determining significance of test-retest differences on eight common neuropsychological instruments.' *The Clinical Neuropsychologist* 18, 373-384

Li, T.-K., Beard, J. D., Orr, W. E., Kwo, P. Y., and Ramchandani, V. A. (1998) Gender and ethnic differences in alcohol metabolism ' *Alcoholism: Clinical and Experimental Research* 22 (3), 771–772

Lieber C. S. and DeCarli L. M. (1968) 'Ethanol oxidation by hepatic microsomes: adaptive increase after ethanol feeding. *Science* 162: 417–418

Lieber, C. S. (2004) 'The discovery of the microsomal ethanol Oxidizing system and its physiologic and pathologic role'. *Drug Metabolism Reviews* 36 (3-4), 511–529

Lobo, I.A. and Harris, A.R. (2008) 'GABAA receptors and alcohol.' *Pharmacology Biochemistry and Behavior* 90(1), 90–94.

Logan, G.D. and Cowan, W.B. (1984). 'On the ability to inhibit thought and action: a theory of an act of control'. *Psychological Review* 91, 295–327

Logan, G.D. (1994) On the ability to inhibit thought and action: A user's guide to the stop signal paradigm. In D. Dagenbach, and T. H. Carr (Eds.), Inhibitory processes in attention, memory, and language (pp. 189–239). San Diego, CA: Academic Press

Lupianez, J., Milliken, B., Solano, C., Weaver, B., and Tipper, S. P. (2001) 'On the strategic modulation of the time course of facilitation and inhibition of return.' *The Quarterly Journal of Experimental Psychology* 54A, 753–773

Lyons, T., Kelsall, D. and Murtagh, J. (1995) The Alcohol Textbook. Thrumpton: Nottingham University Press

Lyvers, M. (2000) '"Loss Of Control" In Alcoholism And Drug Addiction: A Neuroscientific Interpretation.' *Experimental and Clinical Psychopharmacology* 8 (2), 225-249

Marczinski, C., Abroms, B., Van Selst, M. and Fillmore, M. (2005) 'Alcohol-Induced Impairment Of Behavioral Control: Differential Effects On Engaging Vs. Disengaging Responses'. *Psychopharmacology* 182 (3), 452-459

Marczinski, C., Combs, S. and Fillmore, M. (2007) 'Increased Sensitivity To The Disinhibiting Effects Of Alcohol In Binge Drinkers.'. *Psychology of Addictive Behaviors* 21 (3), 346-354

Marczinski, C.A. and Fillmore, M.T. (2003) 'Preresponse cues reduce the impairing effects of alcohol on the execution and suppression of responses'. *Experimental and Clinical Psychopharmacology* 11, 110-117

Marczinski, C.A. and Fillmore, M.T. (2005) 'Compensating for Alcohol-Induced Impairment of Control: Effects on Inhibition and Activation of Behavior' *Psychopharmacology* 181, 337–346.

Martin C.S. and Moss, H.B. (1993) 'Measurement of Acute Tolerance to Alcohol in Human Subjects'. *Alcoholism: Clinical and Experimental Research* 7(2), 211–216

Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M. W., and Swift R. M. (1993) 'Development and validation of the Biphasic Alcohol Effects Scale'. *Alcoholism: Clinical and Experimental Research 17*(1), 140–146

McClelland, J. L., and Rumelhart, D. E. (1981) 'An interactive activation model of context effects in letter perception: Part 1. An account of basic findings.' *Psychological Review* 88, 375– 407

McKim, W. A. and Hancock, S. (2012) *Drugs and behavior: An introduction to behavioral pharmacology*. United States: Pearson

McMahon, J. and Jones, B.T. (1993a) 'The Negative Alcohol Expectancy Questionnaire.' *Journal of the Association of Nurses on Substance Abuse*, 12, 17.

McMahon, J. and Jones, B.T. (1993b) 'Negative expectancy and motivation.' *Addiction Research*, 1, 145-155

McMahon, J., Jones, B.T. and O'Donnell, P. (1994) 'Comparing positive and negative alcohol expectancies in male and female social drinkers.' *Addiction Research* 1: 349–365

Mellanby E. (1919) *Alcohol: its absorption into and disappearance from the blood under different conditions.* Special Report Series Monograph. London: Medical Research Committee

Miller, J., Schaffer, R. and Hackley, S.A. (1991) 'Effects of preliminary information in a go versus no-go task'. *Acta Psychologica* 76, 241-292.

Miller, M.A. and Fillmore, M.T. (2014) 'Protracted Impairment of Impulse Control Under an Acute Dose of Alcohol: A Time-Course Analysis'. *Addictive Behaviors* 39 (11), 1589-1596

Miller, P. M., Smith, G. T. and Goldman, M. S. (1990) 'Emergence of alcohol expectancies in child- hood: a possible critical period' *Journal of Studies on Alcohol* 51, 343–349

Monk, R.L. and Heim, D. (2013) 'A Critical Systematic Review of Alcohol-Related Outcome Expectancies'. *Substance Use and Misuse* [online] 48, 539–557

Morean, M.E. and Corbin, W.R. (2010) 'Subjective response to alcohol: a critical review of the literature.' *Alcoholism: Clinical and Experimental Research* 34(3), 385–395

Morzorati, S. L., Ramchandani, V. A., Flury, L., Li, T. K., and O'Connor, S. (2002) 'Self-reported subjective perception of intoxication reflects family history of alcoholism when breath alcohol levels are constant.' *Alcoholism: Clinical and Experimental Research* 26, 1299 –1306

Nagy, J. (2008) 'Alcohol related changes in regulation of NMDA receptor functions.' *Current Neuropharmacology* 6 (1), 39-54

Naimi, T.S., Brewer, R.D., Mokdad, A., Denny, C., Serdula, M.K. and Marks, J.S. (2003) 'Binge Drinking Among US Adults.' *Journal of the American Medical Association* 289(1): 70–75

Natvigaas, H., Leigh, B. C., Anderssen, N., and Jakobsen, R. (1998) 'Two-year longitudinal study of alcohol expectancies and drinking among Norwegian adolescents.' *Addiction*, 93, 373–384

Newlin, D. (1986) 'Conditioned compensatory response to alcohol placebo in humans' *Psychopharmacology* 88 (2), 247–51

NHS Choices, N. (2016) Binge Drinking - Live Well - NHS Choices [online] available from <http://www.nhs.uk/Livewell/alcohol/Pages/Bingedrinking.aspx> [1 February 2016]

Nutt, D.J. and Stahl, S.M. (2010) 'Searching for perfect sleep: The continuing evolution of GABAA receptor modulators as hypnotics'. *Journal of Psychopharmacology* 24 (11), 1601–1612

O'Brien, R. G. and Kaiser, M. K. (1985) 'MANOVA method for analyzing repeated measures designs: An extensive primer' *Psychological Bulletin* 97 (2), 316–333

Office for National Statistics (2016) Opinions and Lifestyles Survey: adult drinking habits in Great Britain, 2014. [Online] Available at <http://www.ons.gov.uk/peoplepopulationandcommunity/healthandsocialcare/drugusealcoholandsmoking/datasets/adultdrinkinghabit>

Olthuis, J. and Klein, R. (2012). On the measurement of the effects of alcohol and illicit substances on inhibition of return. *Psychopharmacology*, 221(4), pp.541-550

Ostling, E. and Fillmore, M. (2010) 'Tolerance to the Impairing Effects of Alcohol on the Inhibition and Activation of Behavior'. *Psychopharmacology* 212 (4), 465-473

Pabst, A., Baumeister, S.E., and Kraus, L. (2010) 'Alcohol-expectancy dimensions and alcohol consumption at different ages in the general population.' *Journal of Studies on Alcohol and Drugs* 71(1), 46–53

Pabst, A., Kraus, L., Piontek, D., Mueller, S., and Demmel, R. (2014) 'Direct and indirect effects of alcohol expectancies on alcohol-related problems'. *Psychology of Addictive Behaviors* 28 (1), 20–30

Palfai, T. and Wood, M. D. (2001) 'Positive alcohol expectancies and drinking behavior: The influence of expectancy strength and memory accessibility'. *Psychology of Addictive Behaviors* 15 (1), 60–67

Parrott, A., Morinan, A., and Moss, M. (2004) *Understanding drugs and behaviour.* United States: Wiley, John and Sons

Paul, S.M. (2006) 'Alcohol-sensitive GABA receptors and alcohol antagonists'. *Proceedings of the National Academy of Sciences* 103 (22), 8307–8308

Prat, G., Adan, A., Perez-Pamies, M., and Sanchez-Turet, M. (2008) 'Neurocognitive effects of alcohol hangover'. *Addictive Behaviors* 33, 15-23

Ramchandani, V. A., Bosron, W. F., and Li, T. K. (2001) 'Research advances in ethanol metabolism'. *Pathologie Biologie* 49 (9), 676–682

Read, J.P.,Wood, M.D., Lejuez, C.W., Palfai, T. P., and Slack, M. (2004) 'Gender, alcohol consumption, and differing alcohol ex- pectancy dimensions in college drinkers.' *Experimental and Clinical Psychopharmacology*, 12, 298–308

Rehm, J., Room, R., Graham, K., Monteiro, M., Gmel, G., and Sempos, C.T. (2003) 'The Relationship of Average Volume of Alcohol Consumption and Patterns of Drinking to Burden of Disease: An Overview.' *Addiction* 98(10): 1209–1228

Reich, R. R., Below, M. C., and Goldman, M. S. (2010) 'Explicit and implicit measures of expectancy and related alcohol cognitions: A meta-analytic comparison.' *Psychology of Addictive Behaviors, 24*, 13–25

Reynolds, B., Richards, J.B. and de Wit, H. (2006) 'Acute-alcohol effects on the Experiential Discounting Task (EDT) and a question-based measure of delay discounting'. *Pharmacology Biochemistry and Behavior* 83, 194-202

Robinson, T. E., and Berridge, K. C. (2001) 'Incentive-sensitization and addiction.' *Addiction* 96, 103-114

Ross, D. and Pihl, R. (1989) 'Modification of the Balanced-Placebo Design for Use at High Blood Alcohol Levels' *Addictive Behaviors* 14 (1), 91–97

Saha, T.D., Stinson, F.S. and Grant, B.F. (2007) 'The role of alcohol consumption in future classifications of alcohol use disorders'. *Drug Alcohol Dependence* 89, 82-92

Saunders, J.B., Aasland, O.G., Babor, T.F., de la Fuente, J.R. and Grant, M. (1993) 'Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption. II.' *Addiction*, 88, 791-804

Schneider, W., Eschman, A. and Zuccolotto, A. (2002) E-Prime user's guide. Psychology Software Tools. Pittsburgh

Seltzer, M.L., Vinokur, A. and Van Rooijen, L. (1975) 'A self-administered Short Michigan Alcoholism Screening Test (SMAST).' *Journal of Studies on Alcohol and Drugs* 36, 117–126

Sharkansky, E.J. and Finn, P.P. (1998) 'Outcome expectancies and Disinhibition on ad-lib alcohol consumption.' *Journal of Studies on Alcohol* 59:198–206

Sheen, D. (2013) Statistical Handbook: a compilation of drinks industry statistics. London: Brewing Publications Limited

Shevlin, M, and Smith, G.W. (2007) 'The factor structure and concurrent validity of the Alcohol Use Disorder Identification Test based on a nationally representative UK sample'. *Alcohol and Alcoholism* 42, 582–587

Slutske WS. (2005) 'Alcohol use disorders among US college students and their non-college-attending peers.' *Archives of General Psychiatry* 62:321–327

Tabachnick, B. G. and Fidell, L. S. (2012) *Using multivariate statistics*. 6th edn. Boston: Pearson Education

Taylor, B., Irving, H.M., Kanteres, F., Room, R., Borges, G., Cherpitel, C., Greenfield, T. and Rehm, J. (2010) 'The More You Drink, The Harder You Fall: A Systematic Review and Meta-Analysis of How Acute Alcohol Consumption and Injury or Collision Risk Increase Together.' *Drug and Alcohol Dependence* 110(1): 108–116

Testa, M., Fillmore, M.T., Norris, J., Abbey, A., Curtin, J.J., Leonard, K.E., Mariano, K.A., Thomas, M.C., Nomensen, K.J., George, W.H., Vanzile-Tamsen, C., Livingston, J.A., Saenz, C., Buck, P.O., Zawacki, T., Parkhill, M.R., Jacques, A.J. and Hayman L.W. Jr. (2006) 'Understanding Alcohol Expectancy Effects: Revisiting the Placebo Condition'. *Alcoholism, Clinical and Experimental Research* 30 (2), 339-348

Tiplady, B., Barton., Dudman, A., Drummond, G. and Wright, P. (2004) *Zig-zag tracking: A test of psychomotor speed and accuracy designed for repeated administration*. British Association for Psychopharmacology: Harrogate

Tiplady, B., Drummond, G., Cameron, E., Gray, E., Hendry, J., Sinclair, W., and Wright, P. (2001) 'Ethanol, errors, and the speed–accuracy trade-off' *Pharmacology Biochemistry and Behavior* 69, 635–641

Tomberg, C. (2010) 'Alcohol Pathophysiology'. *Journal of Psychophysiology* 24 (4), 215–230

Townshend, J.M. and Duka, T. (2005) 'Binge drinking, cognitive performance and mood in a population of young social drinkers'. *Alcoholism: Clinical and Experimental Research* 29, 317-325

Vengeliene, V., Bilbao, A., Molander, A., and Spanagel, R. (2008) 'Neuropharmacology of alcohol addiction' *British Journal of Pharmacology* 154 (2), 299-315

Vogel-Sprott, M. and Fillmore, M.T. (1993) 'Impairment and recovery under repeated doses of alcohol: effects of response-outcomes.' *Pharmacology Biochemistry and Behavior* 45(1), 59–63

Wardell, J.D. and Read, J.P. (2013) ‘Alcohol expectancies, perceived norms, and drinking behavior among college students: examining the reciprocal determinism hypothesis.’ *Psychology of Addictive Behaviors* 27: 191–196

Watkins, R. and Adler, E. (1993) 'The effect of food on alcohol absorption and elimination patterns'. *Journal of Forensic Sciences* 38 (2), 285–91.

Watson, D., Clark, L.A. and Tellegen, A. (1988). Development and Validation of Brief Measures of Positive and Negative Affect: The PANAS Scales. *Journal of Personality and Social Psychology*, 54, 1063-1070

Weafer, J. and Fillmore, M.T. (2012) ‘Acute tolerance to alcohol impairment of behavioural and cognitive mechanisms related to driving: drinking and driving on the descending limb’. *Psychopharmacology* 220, 697-706

Weafer, J. and Fillmore, M.T. (2013) ‘Acute Alcohol Effects on Attentional Bias in Heavy and Moderate Drinkers’. *Psychology of Addictive Behaviors* 27 (1), 32–41

Weafer, J., Baggott, M. J., and de Wit, H. (2013) 'Test–retest Reliability of Behavioral Measures of Impulsive Choice, Impulsive Action, and Inattention'. *Experimental and Clinical Psychopharmacology* 21 (6), 475-481

Wickelgren, W.A. (1977) ‘Speed-accuracy tradeoff and information processing dynamics’. *Acta Psychologica*, 41, 67-85

Wilcockson, T.D.W. and Pothos, E.M. (2015) 'Measuring Inhibitory Processes for Alcohol-Related Attentional Biases: Introducing a Novel Attentional Bias Measure'. *Addictive Behaviors* 44, 88-93

World Health Organization (2014) *Global Status Report on Alcohol and Health 2014*. Geneva: World Health Organization

Zamboanga, B. L., Bean, J. L., Pietras, A. C., and Pabon, L. C. (2005) ‘Subjective evaluations of alcohol expectancies and their relevance to drinking games involvement in female college students.’ *The Journal of Adolescent Health* 37, 77–80

Zamboanga, B.L., Schwartz, S.J., Ham, L.S., Borsari, B., and Van Tyne, K. (2010) ‘Alcohol expectancies, pregaming, drinking games, and hazardous alcohol use in a multiethnic sample of college students.’ *Cognitive Therapy and Research* 34: 124–133

Appendices

APPENDIX 1: MEDICAL HEALTH HISTORY QUESTIONNAIRE

Health History Questionnaire

Name:	Phone:		Email:
Ethnicity:	Age:	Height:	Weight:
Religion:	Occupation:		Gender (M/F):
	Date of Birth:		
Past Medical History			
Significant Illnesses: Cancer, Diabetes, Hepatitis, High Blood Pressure, Heart Disease, Rheumatic Fever, Thyroid Disease, Seizures, Venereal Disease			
Significant Trauma (auto accidents, falls, etc.)			
Allergies (drugs, chemicals, foods):			

Family Medical History
☐ Diabetes ☐ Cancer ☐ High Blood Pressure ☐ Seizures ☐ Asthma ☐ Allergies ☐ Stroke ☐ Heart Disease
Medicines taken within the last two months (Include vitamins, over-the-counter drugs, herbs, etc)
How many cigarettes a day do you smoke?_____ How often do you drink coffee, tea or cola per day/week? Day: _____Week: _____ Please describe any use of drugs for non-medical purposes:

General

☐ Poor Appetite ☐ Poor Sleeping ☐ Fatigue
☐ Fever ☐ Chills ☐ Night Sweats
☐ Sweat Easily ☐ Tremors ☐ Cravings
☐ Localized Weakness ☐ Poor Balance ☐ Change in appetite
☐ Bleed or Bruise Easily ☐ Weight Loss ☐ Weight Gain
☐ Peculiar Tastes or Smells ☐ Strong Thirst (cold or hot drinks)
☐ Sudden Energy Drop (What time of day?) _____

Head, Eyes, Ears, Nose and Throat

☐ Dizziness ☐ Concussions ☐ Migraines
☐ Glasses ☐ Eye Strain ☐ Eye Pain
☐ Poor Vision ☐ Night Blindness ☐ Color Blindness
☐ Cataracts ☐ Blurry Vision ☐ Earaches
☐ Ringing in Ears ☐ Poor Hearing ☐ Spots in Front of Eyes
☐ Sinus Problems ☐ Nose Bleeds ☐ Recurrent Sore Throats
☐ Grinding Teeth ☐ Facial Pain ☐ Sores
☐ Teeth Problems ☐ Jaw Clicks
☐ Headaches (Where and When?) _____
Any other head or neck problems? _____

Cardiovascular

☐ High Blood Pressure ☐ Low Blood Pressure ☐ Chest Pain
☐ Irregular Heartbeat ☐ Dizziness ☐ Fainting
☐ Cold Hands or Feet ☐ Swelling of the Hands ☐ Swelling of the Feet
☐ Blood Clots ☐ Phlebitis ☐ Difficulty in Breathing
Any other heart or blood vessel problems? _____

Respiratory

☐ Cough ☐ Coughing Blood ☐ Chest Pain
☐ Bronchitis ☐ Pneumonia ☐ Pain with a Deep Breath
☐ Difficulty in Breathing when Lying Down
☐ Production of Phlegm (What color?)
Any other lung problems? _____

Gastrointestinal

☐ Nausea ☐ Vomiting ☐ Diarrhea
☐ Constipation ☐ Gas ☐ Belching
☐ Black Stools ☐ Blood in Stools ☐ Indigestion
☐ Bad Breath ☐ Rectal Pain ☐ Hemorrhoids
☐ Abdominal Pain or Cramps
☐ Chronic Laxative Use
Any other problems with your stomach or intestines? _____

Musculoskeletal

☐ Neck Pain ☐ Muscle Pains ☐ Knee Pain
☐ Back Pain ☐ Muscle Weakness ☐ Foot / Ankle Pains
☐ Hand / Wrist Pains ☐ Shoulder Pain ☐ Hip Pain
Any other joint or bone problems? _____

Neuropsychological

☐ Seizures ☐ Dizziness ☐ Loss of Balance
☐ Areas of Numbness ☐ Lack of Coordination ☐ Poor Memory
☐ Concussion ☐ Depression ☐ Anxiety
☐ Bad Temper ☐ Easily Susceptible to Stress
Any other neurological or psychological problems? _____

Comments (any other medical conditions not mentioned above)

APPENDIX 2: ETHICAL APPROVAL CERTIFICATE

Certificate of Ethical Approval

Applicant:

Andy Eastwood

Project Title:

Acute tolerance to the impairing effects of alcohol

This is to certify that the above named applicant has completed the Coventry University Ethical Approval process and their project has been confirmed and approved as Medium Risk

Date of approval:

16 July 2015

Project Reference Number:

P31903

APPENDIX 3: SHORT MICHIGAN ALCOHOL SCREENING TEST (SMAST)

SHORT MICHIGAN ALCOHOL SCREENING TEST (SMAST)

NAME: _____ Date:_____

The following questions concern information about your involvement with alcohol during the past 12 months. Carefully read each countyment and decide if your answer is "YES" or "NO". Then, check the appropriate box beside the question.

Please answer every question. If you have difficulty with a countyment, then choose the response that is mostly right.

These questions refer to the past 12 months only.	**YES**	**NO**
1. Do you feel that you are a normal drinker? (by normal we mean do you drink less than or as much as most other people.)		
2. Does your wife, husband, a parent, or other near relative ever worry or complain about your drinking?		
3. Do you ever feel guilty about your drinking?		
4. Do friends or relatives think you are a normal drinker?		
5. Are you able to stop drinking when you want to?		
6. Have you ever attended a meeting of Alcoholics Anonymous (AA)?		
7. Has your drinking ever created problems between you and your wife, husband, a parent or other near relative?		
8. Have you ever gotten into trouble at work because of your drinking?		
9. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?		
10. Have you ever gone to anyone for help about your drinking?		
11. Have you ever been in a hospital because of drinking?		
12. Have you ever been arrested for drunken driving, driving while intoxicated, or driving under the influence of alcoholic beverages?		
13. Have you ever been arrested, even for a few hours, because of other drunken behaviors?		
*** SMAST Score**		

** See scoring instructions for correct scoring procedures.*

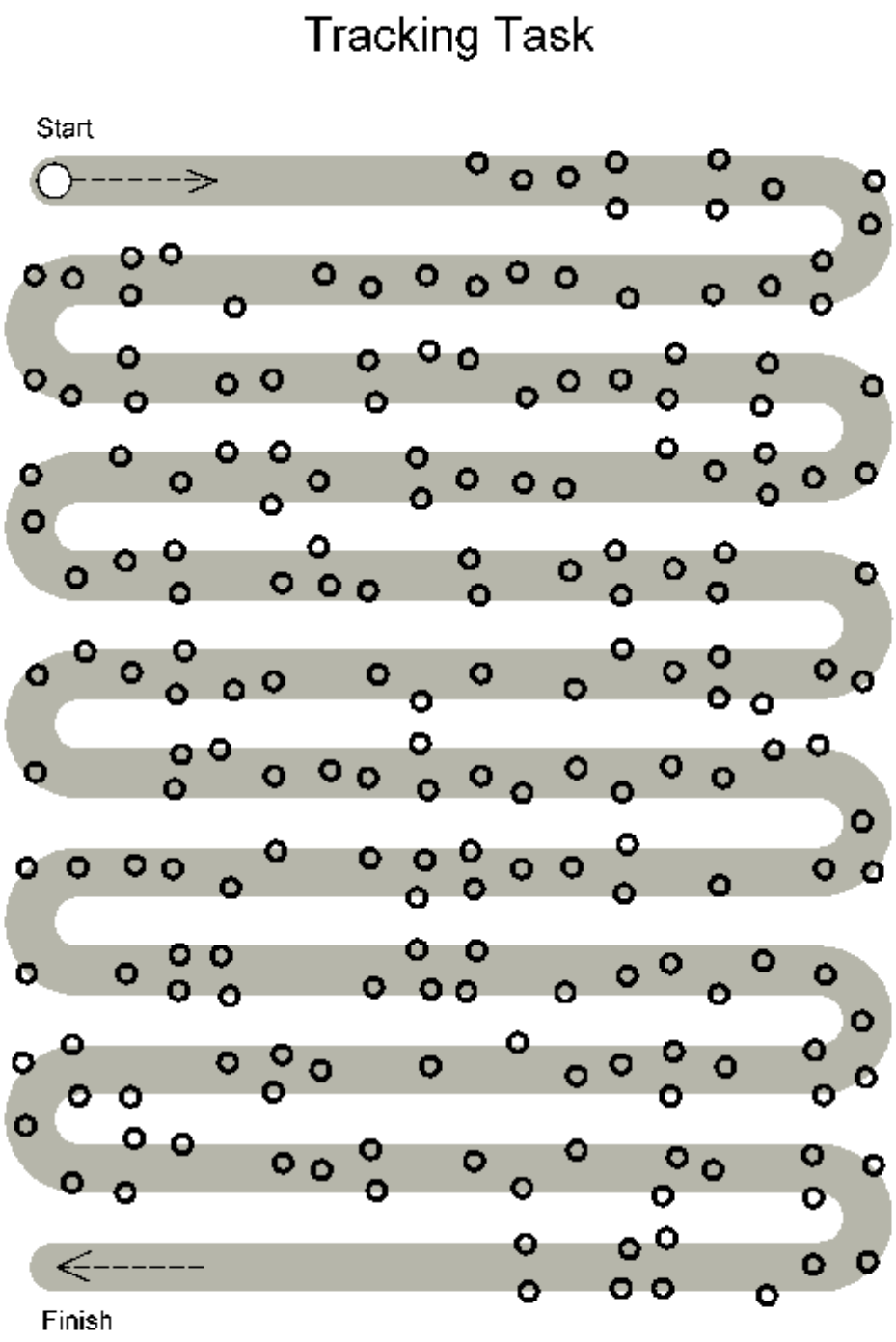
APPENDIX 4: ALCOHOL USE IDENTIFICATION TEST (AUDIT)

This is one unit of alcohol...

AUDIT

Questions	Scoring system					Your score	Sub-Score
	0	1	2	3	4		
How often do you have a drink containing alcohol?	Never	Monthly or less	2 - 4 times per month	2 - 3 times per week	4+ times per week		
How many units of alcohol do you drink on a typical day when you are drinking?	1 -2	3 - 4	5 - 6	7 - 9	10+		
How often have you had 6 or more units if female, or 8 or more if male, on a single occasion in the last year?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily		
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		
How often during the last year have you failed to do what was normally expected from you because of your drinking?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily		
How often during the last year have you needed an alcoholic drink in the morning to get yourself going after a heavy drinking session?	Never	Less than monthly	Monthly	Weekly	Daily or almost daily		
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		
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		
Have you or somebody else been injured as a result of your drinking?	No		Yes, but not in the last year		Yes, during the last year		
Has a relative or friend, doctor or other health worker been concerned about your drinking or suggested that you cut down?	No		Yes, but not in the last year		Yes, during the last year		

APPENDIX 5: ZIG-ZAG TRACKING TASK (ZZTR)



APPENDIX 6: SUBJECTIVE INTOXICATION (SI) SCALE

Participant
Date

Self-reported Intoxication Scale

INSTRUCTIONS

Please answer the following questions. The answers will be treated as strictly confidential.

1. Please rate the way you feel in terms of the dimensions given below.
2. Regard the line as representing the full range of each dimension.
3. Rate your feelings as they are at the moment.
4. Mark clearly and perpendicularly across each line.

1. How drunk do you currently feel?

Baseline

Completely Sober (not drunk) _____ Highly Intoxicated (very drunk)

Ascending

Completely Sober (not drunk) _____ Highly Intoxicated (very drunk)

Descending

Completely Sober (not drunk) _____ Highly Intoxicated (very drunk)

Scale Source: - Bond, A. and Lader, M. (1974) 'The use of analogue scales in rating subjective feelings.' *British Journal of Medical Psychology* 47, 211 – 218

- 1 -

APPENDIX 7: BIPHASIC ALCOHOL EFFECTS SCALE (BAES)

Alcohol Effects Scale (BAES)

Please rate the extent to which these words describe your feelings at the present time (circle the appropriate number for each word)

The following adjectives describe feelings that some people have after drinking alcohol. Please rate the extent to which drinking alcohol has produced these feelings in you at the present time (circle the appropriate number for each word)

		Not at all										Extremely
1	Difficulty concentrating	0	1	2	3	4	5	6	7	8	9	10
2	Down	0	1	2	3	4	5	6	7	8	9	10
3	Elated	0	1	2	3	4	5	6	7	8	9	10
4	Energised	0	1	2	3	4	5	6	7	8	9	10
5	Excited	0	1	2	3	4	5	6	7	8	9	10
6	Heavy head	0	1	2	3	4	5	6	7	8	9	10
7	Inactive	0	1	2	3	4	5	6	7	8	9	10
8	Sedated	0	1	2	3	4	5	6	7	8	9	10
9	Slow thoughts	0	1	2	3	4	5	6	7	8	9	10
10	Sluggish	0	1	2	3	4	5	6	7	8	9	10
11	Stimulated	0	1	2	3	4	5	6	7	8	9	10
12	Talkative	0	1	2	3	4	5	6	7	8	9	10
13	Up	0	1	2	3	4	5	6	7	8	9	10
14	Vigorous	0	1	2	3	4	5	6	7	8	9	10

APPENDIX 8: ETHICAL APPROVAL CERTIFICATE

Certificate of Ethical Approval

Applicant:

Andy Eastwood

Project Title:

The role of outcome expectancies and the development of acute tolerance to the impairing effects of alcohol.

This is to certify that the above named applicant has completed the Coventry University Ethical Approval process and their project has been confirmed and approved as Medium Risk

Date of approval:

27 June 2016

Project Reference Number:

P44178

Appendix 9: Alcohol Expectancy Questionnaire (AEQ-III)

44209

ALCOHOL EXPECTANCY QUESTIONNAIRE, REVISED
[120-item rev.9/94) Page 1
30 YR

(iaq.sav)

Date: ☐☐ / ☐☐ / ☐☐

The following pages contain statements about the effects of alcohol. Read each statement carefully and respond according to your own personal thoughts, feelings and beliefs about alcohol now. We are interested in what you think about alcohol, regardless of what other people might think.

When the statements refer to drinking alcohol, you may think in terms of drinking any alcoholic beverage, such as beer, wine, whiskey, liquor, rum, scotch, vodka, gin, or various alcoholic mixed drinks. Whether or not you have had actual drinking experiences yourself, you are to answer in terms of your beliefs about alcohol. It is important that you respond to every question.

PLEASE BE HONEST. REMEMBER, YOUR ANSWERS ARE CONFIDENTIAL. Please answer every item. RESPOND TO THESE ITEMS ACCORDING TO WHAT YOU PERSONALLY BELIEVE TO BE TRUE ABOUT ALCOHOL. Fill in the circle which shows how much you agree or disagree with each item:

PLEASE USE A BLACK PEN Shade circles like this: ● Not like this: ⊘ ✓	1 DISAGREE STRONGLY	2 DISAGREE SOMEWHAT	3 UNCERTAIN	4 AGREE SOMEWHAT	5 AGREE STRONGLY

	1	2	3	4	5	
iaq1	○	○	○	○	○	1. Alcohol can transform my personality.
	○	○	○	○	○	2. Drinking helps me feel whatever way I want to feel.
	○	○	○	○	○	3. Some alcohol has a pleasant, cleansing, tingly taste.
	○	○	○	○	○	4. Alcohol makes me feel happy.
	○	○	○	○	○	5. Drinking adds a certain warmth to social occasions.
	○	○	○	○	○	6. Sweet, mixed drinks taste good.
	○	○	○	○	○	7. When I am drinking, it is easier to open up and express my feelings.
	○	○	○	○	○	8. Time passes quickly when I am drinking.
	○	○	○	○	○	9. When they drink, women become more sexually relaxed.
	○	○	○	○	○	10. Drinking makes me feel flushed.
	○	○	○	○	○	11. I feel powerful when I drink, as if I can really influence others to do as I want.
	○	○	○	○	○	12. Drinking increases male aggressiveness.
	○	○	○	○	○	13. Alcohol lets my fantasies flow more easily.
	○	○	○	○	○	14. Drinking gives me more confidence in myself.
	○	○	○	○	○	15. Drinking makes me feel good.
	○	○	○	○	○	16. I feel more creative after I have been drinking.
	○	○	○	○	○	17. Having a few drinks is a nice way to celebrate special occasions.
	○	○	○	○	○	18. After a few drinks, it is easier to pick a fight.
	○	○	○	○	○	19. When I am drinking I feel free to be myself and to do whatever I want.
	○	○	○	○	○	20. Drinking makes it easier to concentrate on the good feelings I have at the time.
	○	○	○	○	○	21. Alcohol allows me to be more assertive.
	○	○	○	○	○	22. When I feel "high" from drinking, everything seems to feel better.
	○	○	○	○	○	23. At times, drinking is like permission to forget problems.
	○	○	○	○	○	24. If I am nervous about having sex, alcohol makes me feel better.
iaq25	○	○	○	○	○	25. Drinking relieves boredom.

CONTINUE ON BACK OF PAGE ⟶

0 1 2 3 4 5 6 7 8 9

44209

PLEASE USE A BLACK PEN
Shade circles like this: ●
Not like this: ⊘ ✓

1 DISAGREE STRONGLY	2 DISAGREE SOMEWHAT	3 UNCERTAIN	4 AGREE SOMEWHAT	5 AGREE STRONGLY

	1	2	3	4	5	
iaq26	○	○	○	○	○	26. I find that conversing with members of the opposite sex is easier for me after I have had a few drinks.
	○	○	○	○	○	27. After a few drinks, I feel less sexually inhibited.
	○	○	○	○	○	28. Drinking is pleasurable because it is enjoyable to join in with people who are enjoying themselves.
	○	○	○	○	○	29. I like the taste of some alcoholic beverages.
	○	○	○	○	○	30. If I am feeling restricted in any way, a few drinks make me feel better.
	○	○	○	○	○	31. Men are friendlier when they drink.
	○	○	○	○	○	32. It is easier for me to meet new people if I've been drinking.
	○	○	○	○	○	33. I can discuss or argue a point more forcefully after I have had a drink or two.
	○	○	○	○	○	34. Alcohol can eliminate feelings of inferiority.
	○	○	○	○	○	35. Alcohol makes women more sensuous.
	○	○	○	○	○	36. If I have a couple of drinks, it is easier to express my feelings.
	○	○	○	○	○	37. I feel less bothered by physical ills after a few drinks.
	○	○	○	○	○	38. Alcohol makes me need less attention from others than I usually do.
	○	○	○	○	○	39. A drink or two makes the humorous side of me come out.
	○	○	○	○	○	40. After a few drinks, I feel more self-reliant than usual.
	○	○	○	○	○	41. After a few drinks, I don't worry as much about what other people think of me.
	○	○	○	○	○	42. When drinking, I do not consider myself totally accountable or responsible for my behavior.
	○	○	○	○	○	43. Alcohol enables me to have a better time at parties.
	○	○	○	○	○	44. Anything which requires a relaxed style can be facilitated by alcohol.
	○	○	○	○	○	45. Drinking makes the future seem brighter.
	○	○	○	○	○	46. I am not as tense if I am drinking.
	○	○	○	○	○	47. I often feel sexier after I have had a couple of drinks.
	○	○	○	○	○	48. Having a few drinks helps me relax in a social situation.
	○	○	○	○	○	49. I drink when I am feeling mad.
	○	○	○	○	○	50. Drinking alone or with one other person makes me feel calm and serene.
	○	○	○	○	○	51. After a few drinks, I feel brave and more capable of fighting.
	○	○	○	○	○	52. Drinking can make me more satisfied with myself.
	○	○	○	○	○	53. There is more camaraderie in a group of people who have been drinking.
	○	○	○	○	○	54. My feelings of isolation and alienation decrease when I drink.
	○	○	○	○	○	55. A few drinks make me feel less in touch with what is going on around me.
	○	○	○	○	○	56. Alcohol makes me more tolerant of people I do not enjoy.
	○	○	○	○	○	57. Alcohol helps me sleep better.
	○	○	○	○	○	58. Alcohol makes me more outspoken or opinionated.
	○	○	○	○	○	59. I am a better lover after a few drinks.
iaq60	○	○	○	○	○	60. Women talk more after they have had a few drinks.

NEXT PAGE ⟶

0 1 2 3 4 5 6 7 8 9

PLEASE USE A BLACK PEN
Shade circles like this: ●
Not like this: ⊘ ✓

1 DISAGREE STRONGLY	2 DISAGREE SOMEWHAT	3 UNCERTAIN	4 AGREE SOMEWHAT	5 AGREE STRONGLY

	1	2	3	4	5	
iaq61	○	○	○	○	○	61. Alcohol decreases muscular tension.
	○	○	○	○	○	62. Alcohol makes me worry less.
	○	○	○	○	○	63. A few drinks make it easier to talk to people.
	○	○	○	○	○	64. After a few drinks I am usually in a better mood.
	○	○	○	○	○	65. Alcohol seems like magic.
	○	○	○	○	○	66. Women can have orgasms more easily if they have been drinking.
	○	○	○	○	○	67. Drinking increases female aggressiveness.
	○	○	○	○	○	68. Drinking helps me get out of a depressed mood.
	○	○	○	○	○	69. After I have had a couple of drinks, I feel I am more of a caring, sharing person.
	○	○	○	○	○	70. Alcohol decreases my feeling of guilt about not working.
	○	○	○	○	○	71. I feel more coordinated after I drink.
	○	○	○	○	○	72. Alcohol makes me more interesting.
	○	○	○	○	○	73. A few drinks make me feel less shy.
	○	○	○	○	○	74. If I am tense or anxious, having a few drinks makes me feel better.
	○	○	○	○	○	75. Alcohol enables me to fall asleep more easily.
	○	○	○	○	○	76. If I am feeling afraid, alcohol decreases my fears.
	○	○	○	○	○	77. A couple of drinks makes me more aroused or physiologically excited.
	○	○	○	○	○	78. Alcohol can act as an anesthetic; that is, it can deaden pain.
	○	○	○	○	○	79. I enjoy having sex more if I have had some alcohol.
	○	○	○	○	○	80. I am more romantic when I drink.
	○	○	○	○	○	81. I feel more masculine/feminine after a few drinks.
	○	○	○	○	○	82. When I am feeling antisocial, drinking makes me more gregarious.
	○	○	○	○	○	83. Alcohol makes me feel better physically.
	○	○	○	○	○	84. Sometimes when I drink alone or with one other person it is easy to feel cozy and romantic.
	○	○	○	○	○	85. I feel like more of a happy-go-lucky person when I drink.
	○	○	○	○	○	86. Drinking makes get-togethers more fun.
	○	○	○	○	○	87. Alcohol makes it easier to forget bad feelings.
	○	○	○	○	○	88. After a few drinks, I am more sexually responsive.
	○	○	○	○	○	89. If I am cold, having a few drinks will give me a sense of warmth.
	○	○	○	○	○	90. It is easier to act on my feelings after I have had a few drinks.
	○	○	○	○	○	91. I become lustful when I drink.
	○	○	○	○	○	92. A couple of drinks makes me more outgoing.
	○	○	○	○	○	93. A drink or two can make me feel more wide awake.
	○	○	○	○	○	94. Alcohol makes me feel closer to people.
iaq95	○	○	○	○	○	95. Women are friendlier after they have had a few drinks.

CONTINUE ON BACK OF PAGE ⟶

0	1	2	3	4	5	6	7	8	9
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○

PLEASE USE A BLACK PEN
Shade circles like this: ●
Not like this: ⊘ ✓

1	2	3	4	5
DISAGREE STRONGLY	DISAGREE SOMEWHAT	UNCERTAIN	AGREE SOMEWHAT	AGREE STRONGLY

	1	2	3	4	5	
iaq96	○	○	○	○	○	96. I tend to be less self-critical when I have something alcoholic to drink.
	○	○	○	○	○	97. I find that conversing with members of the opposite sex is easier for me after I have had a few drinks.
	○	○	○	○	○	98. Drinking makes me feel flushed.
	○	○	○	○	○	99. It is easier to remember funny stories or jokes if I have been drinking.
	○	○	○	○	○	100. After a few drinks I am less submissive to those in positions of authority.
	○	○	○	○	○	101. Alcohol makes me more talkative.
	○	○	○	○	○	102. I am more romantic when I drink.
	○	○	○	○	○	103. Men can have orgasms more easily if they have had a drink.
	○	○	○	○	○	104. A drink or two is really refreshing after strenuous physical activity.
	○	○	○	○	○	105. Alcohol enables me to have a better time at parties.
	○	○	○	○	○	106. I can be more persuasive if I have had a few drinks.
	○	○	○	○	○	107. Drinking makes people feel more at ease in social situations.
	○	○	○	○	○	108. Alcohol helps me sleep better.
	○	○	○	○	○	109. After a drink or two, things like muscle aches and pains do not hurt as much.
	○	○	○	○	○	110. Alcohol decreases my hostilities.
	○	○	○	○	○	111. Alcohol makes me worry less.
	○	○	○	○	○	112. Alcohol makes it easier to act impulsively or make decisions quickly.
	○	○	○	○	○	113. Alcohol makes me feel less shy.
	○	○	○	○	○	114. Alcohol makes me more tolerant of people I do not enjoy.
	○	○	○	○	○	115. Alcohol makes me need less attention from others than I usually do.
	○	○	○	○	○	116. A drink or two can slow me down, so I do not feel so rushed or pressured for time.
	○	○	○	○	○	117. I feel more sexual after a few drinks.
	○	○	○	○	○	118. Alcohol makes me feel better physically.
	○	○	○	○	○	119. Having a drink in my hand can make me feel secure in a difficult social situation.
iaq120	○	○	○	○	○	120. Things seem funnier when I have been drinking or at least I laugh more.

0	1	2	3	4	5	6	7	8	9
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○
○	○	○	○	○	○	○	○	○	○

APPENDIX 10: NEGATIVE ALCOHOL EXPECTANCY QUESTIONNAIRE (NAEQ)

Negative Alcohol Expectancy Questionnaire (NAEQ)

Below is a list of things that you might or might not expect to happen to you during or after heavy drinking. Indicate the likelihood of each of these things happening to you if you were to go out and drink heavily. Circle the appropriate number on the 1-2-3-4-5 scale. Please be sure to answer every question.

1 = highly likely 2 = likely 3 = possible 4 = unlikely 5 = highly unlikely

IF I WERE TO GO OUT AND DRINK HEAVILY THIS WEEKEND, THEN…

1.	I would become argumentative	1	2	3	4	5
2.	I would become aggressive	1	2	3	4	5
3.	I would become violent	1	2	3	4	5
4.	I would become anxious	1	2	3	4	5
5.	I would have an accident	1	2	3	4	5
6.	I would become depressed	1	2	3	4	5
7.	I would get drunk	1	2	3	4	5
8.	I would get in a fight	1	2	3	4	5
9.	I would have memory problems	1	2	3	4	5
10.	I would lie about how much I had to drink	1	2	3	4	5
11.	I would end up in jail	1	2	3	4	5
12.	I would argue with my spouse/significant other	1	2	3	4	5
13.	I would have difficulty sleeping	1	2	3	4	5
14.	I would wet the bed	1	2	3	4	5
15.	I would become boastful	1	2	3	4	5
16.	I would borrow money	1	2	3	4	5
17.	I would consider taking other drugs	1	2	3	4	5
18.	I would take other drugs	1	2	3	4	5
19.	I would lose my driving license	1	2	3	4	5
20.	I would drink more than the others with me	1	2	3	4	5
21.	I would have difficulty in stopping drinking	1	2	3	4	5

IF I WERE TO GO OUT AND DRINK HEAVILY THIS WEEKEND, THEN THE NEXT DAY…

22.	I would miss work/school	1	2	3	4	5
23.	I would have ‘the shakes’	1	2	3	4	5
24.	I would have ‘the sweats’	1	2	3	4	5
25.	I would have a hangover	1	2	3	4	5

26	I would feel depressed	1	2	3	4	5
27.	I would have low self-esteem	1	2	3	4	5
28.	I would crave a drink	1	2	3	4	5
29.	I would have difficulty sleeping	1	2	3	4	5
30.	I would feel generally ill	1	2	3	4	5
31.	I would feel frightened	1	2	3	4	5
32.	I would feel guilty	1	2	3	4	5
33.	I would feel remorseful	1	2	3	4	5
34.	I would feel anxious	1	2	3	4	5
35.	I would be shy if meeting people	1	2	3	4	5
36.	I would feel restless	1	2	3	4	5
37.	I would be sick	1	2	3	4	5
38.	I would be unable to eat	1	2	3	4	5
39.	I would go on a binge	1	2	3	4	5

IF I CONTINUED TO DRINK HEAVILY AT MY PRESENT LEVEL, THEN…

40.	I would lose my spouse/significant other	1	2	3	4	5
41.	I would lose my house	1	2	3	4	5
42.	I would lose my job/be forced to leave school	1	2	3	4	5
43.	I would have the DTs (delirium tremors)	1	2	3	4	5
44.	I would have convulsions	1	2	3	4	5
45.	I would lose my friends	1	2	3	4	5
46.	I would get into debt	1	2	3	4	5
47.	I would end up in the hospital	1	2	3	4	5
48.	I would end up sleeping poorly	1	2	3	4	5
49.	I would consider suicide	1	2	3	4	5
50.	I would attempt suicide	1	2	3	4	5
51.	I would feel frightened	1	2	3	4	5
52.	I would feel depressed	1	2	3	4	5
53.	I would feel self-loathing	1	2	3	4	5
54.	I would feel self-pity	1	2	3	4	5
55.	I would lose all respect for myself	1	2	3	4	5
56.	I would end up in jail	1	2	3	4	5
57.	I would damage my liver	1	2	3	4	5
58.	I would feel I was going mad	1	2	3	4	5
59.	I would choke on my own vomit	1	2	3	4	5
60.	I would die	1	2	3	4	5

APPENDIX 11: REPEATED MEASURES ANALYSIS OF VARIANCE ASSUMPTION

11.1 OUTLIERS

On inspection of the boxplots presented in Figure 1, extreme outliers can be seen in the baseline condition for SI rating and GPB performance. No other extreme outliers were present in the data set. Scores for each dependent measure (CGNGT, ZZTR, GPB, SI and BAES) at each time point (baseline, ascending and descending limb) were converted in to z-scores to assess problematic outliers in the data; scores that fall between ± 1.96 were considered normal, scores exceeding ± 1.96 were potential outliers and scores that exceed ± 3.29 were extreme outliers (Fields 2013). Approximately 95% of the data were normal and the remaining ~5% were potential, non-problematic outliers (negligible extreme outliers present in the data).

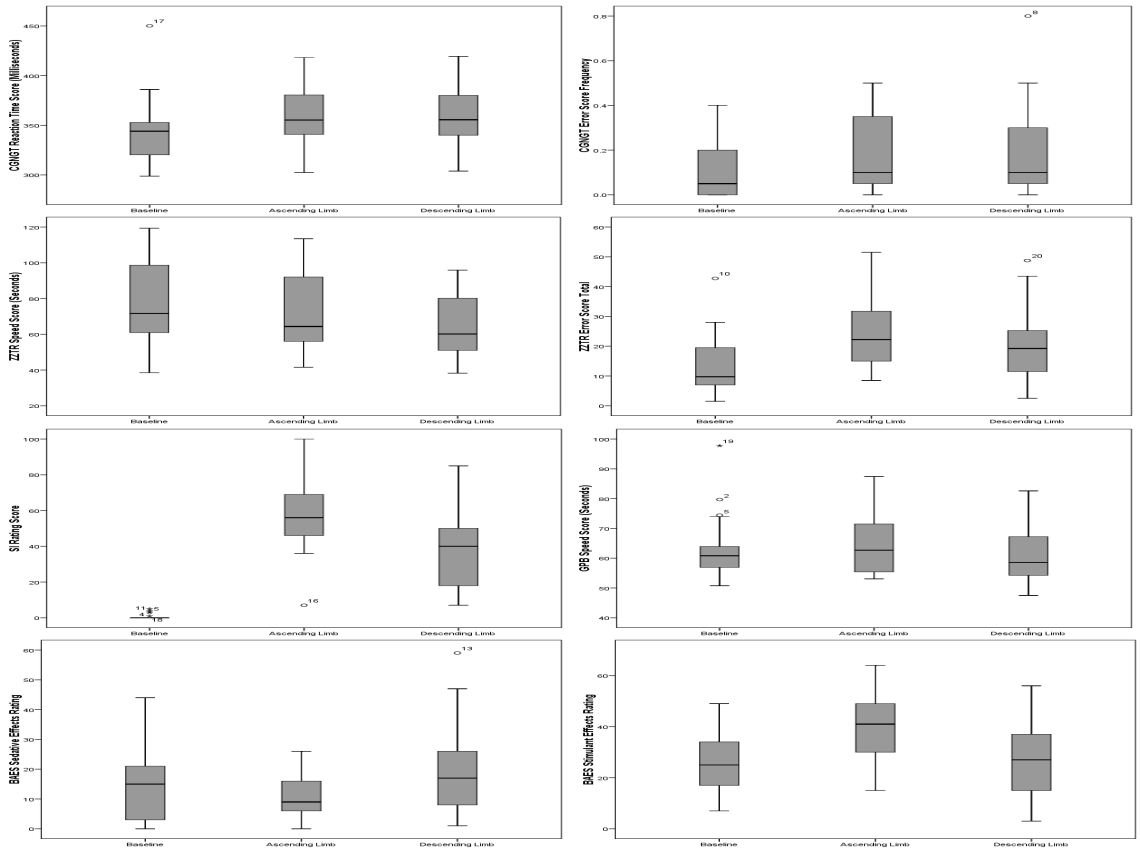


Figure 1: Boxplots for each dependent measure (CGNGT, ZZTR, GPB, SI and BAES) at each time point (baseline, ascending and descending limb).

11.2 Normal Distribution

Normality was further assessed for all dependent measures (CGNGT, ZZTR, GPB, SI and BAES) at each time point (baseline, ascending and descending limb) by calculating skew and kurtosis z-score values (see Table 1); z-scores between the values of ±1.96 reflect no significant skew or kurtosis (Field 2013; Tabachnick and Fidell 2012). Minor violations of normality were apparent. Table 1 shows that the CGNGT error frequency scores on the descending limb and baseline RT scores demonstrated significant positive skew and kurtosis. Baseline GPB, SI and error scores on the ZZTR task also demonstrated significant positive skew and kurtosis. The assumption of normality was maintained for all other levels as the calculated z-scores demonstrated no significant skew/kurtosis. Despite normality violation, data was not transformed as ANOVA is a robust analysis to these minor assumption violations (Field 2013).

Table 1: Skewness and Kurtosis Z-Scores for each dependent measure (CGNGT, ZZTR, GPB, SI and BAES) at each time point (baseline, ascending and descending limb).

N=21	Skewness Z-Score			Kurtosis Z-Score		
	Baseline	Ascending Limb	Descending Limb	Baseline	Ascending Limb	Descending Limb
CGNGT (Error Frequency)	1.73	1.20	3.04*	-0.40	-1.34	2.09*
CGNGT (RT; Milliseconds)	3.24*	0.45	0.68	4.58*	-0.43	-0.77
ZZTR Speed (Seconds)	*0.64*	*0.91*	*0.67*	*-1.09*	*-1.19*	*-1.29*
ZZTR Accuracy (Error total)	*2.76**	*1.89*	*1.98**	*2.09**	*0.5*	*1.09*
GPB Speed	*3.38**	*1.54*	*1.74*	*3.93**	*0.12*	*0.46*
SI	*3.74**	*-0.44*	*0.85*	*2.33**	*1.61*	*-0.58*
BAES Sedative Effects	*1.63*	*0.85*	*1.72*	*0.07*	*-0.29*	*0.05*
BAES Stimulant Effects	*0.55*	*-0.23*	*0.68*	*-0.93*	*-0.61*	*-0.66*

*NOTE: *significant positive skew/kurtosis z-score (+ 1.96) **significant negative skew/kurtosis score (-1.69)*

11.3 SPHERICITY

Table 2: Mauchly's (W) test statistic, approximate χ^2, significance value and Greenhouse-Geisser Correction.

	Mauchly's Test			*Greenhouse-Geisser Correction*
	W	χ^2	*Sig.*	*Sig.*
CGNGT (Error Frequency)	.80	4.21	.12	.
CGNGT (RT; Milliseconds)	.90	2.02	.36	.
ZZTR Speed (Seconds)	.91	1.73	.42	.
ZZTR Error	.75	5.58	.06	.
GPB Speed (Seconds)	.92	1.50	.47	.
SI	.97	.66	.72	.
BAES Sedative Effects	.52	12.62	.002*	.67
BAES Stimulant Effects	.93	1.36	.51	.

*NOTE: *Sphericity violated p<0.05.*

APPENDIX 12: REGRESSION MODEL ASSUMPTIONS

Each regression model assessed below has 2 predictor variables, positive and negative outcome expectancy. These variables were entered into the model simultaneously. The outcome variables were the magnitude of tolerance developed to measures of ZZTR Speed (Seconds), ZZTR Accuracy (Error Total), GPB Speed (Seconds) and SI. Firstly, Cook's d values were calculated to assess whether any data had an undue influence on the regression models. All Cook's d values were below the value of 1 and therefore not considered problematic (Cook and Weisberg 1982).

12.1 INDEPENDENCE OF RESIDUALS

Independence of residuals was assessed by a Durbin Watson statistic (Field 2013). These values demonstrate independence of residuals for each regression model (see *Table 3)*.

Table 3: Durbin Watson Statistics for each Regression Model

Outcome Variable (Magnitude of Acute Alcohol Tolerance Development)	Durbin Watson
ZZTR Speed (Seconds)*	2.082
ZZTR Error (Error Total)*	2.029
GPB Speed (Seconds)*	2.917
SI*	1.897

*NOTE: *Predictors: Positive Expectancy, Negative Expectancy.*

12.2 MULTICOLLINEARITY

The assumption of multicollinearity between the 2 independent variables (positive and negative expectancy was assessed by the tolerance collinearity statistic and the reciprocal Variance Inflation Factor (VIF). There were no issues of collinearity between positive and negative expectancy, tolerance = .80 and VIF = 1.26 (Field 2013).

12.3 NORMALITY

To test normality of residuals, P-P plot's (*Figure 3)* and histogram's (F*igure 4)* were produced for each outcome variable (ZZTR Speed, Accuracy, GPB Speed and SI). These show that the residuals were approximately normally distributed.

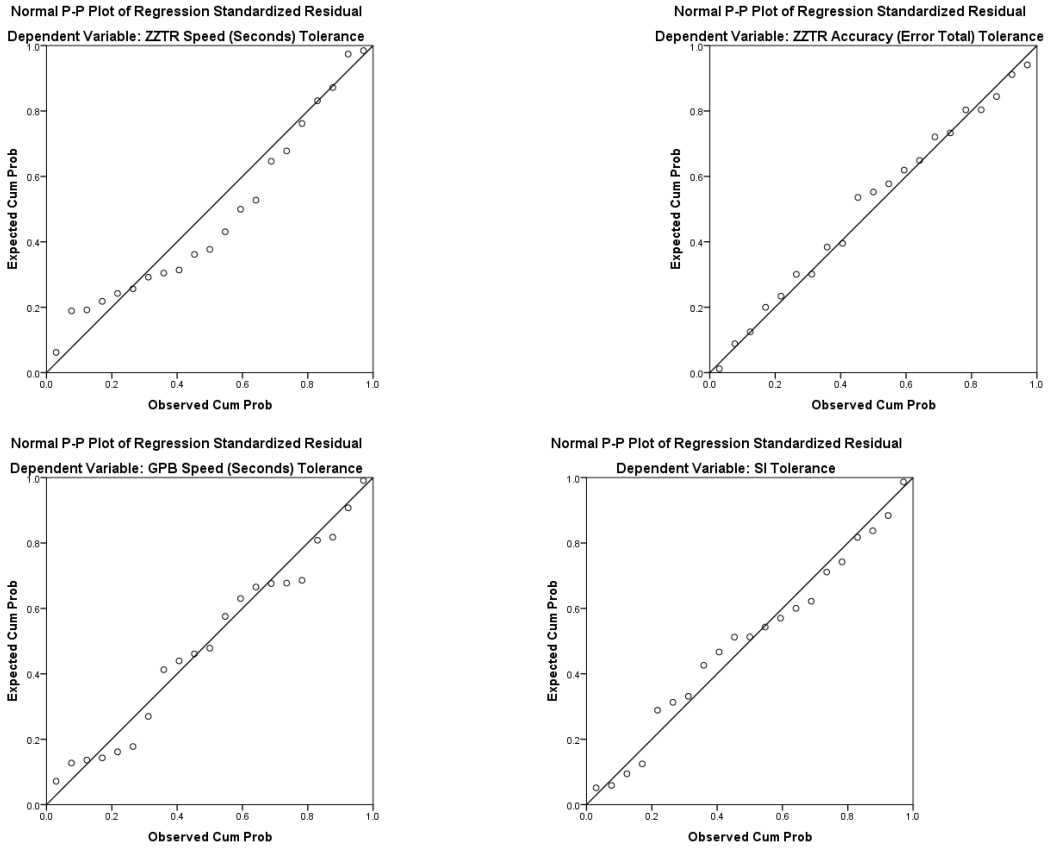
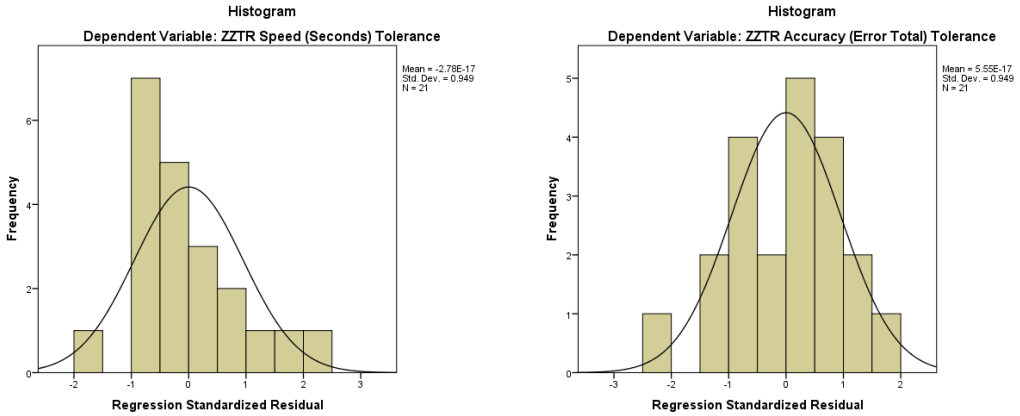


Figure 3: Standardised residual probability plots for each regression model (ZZTR Speed, Accuracy, GPB Speed and SI outcome variables).



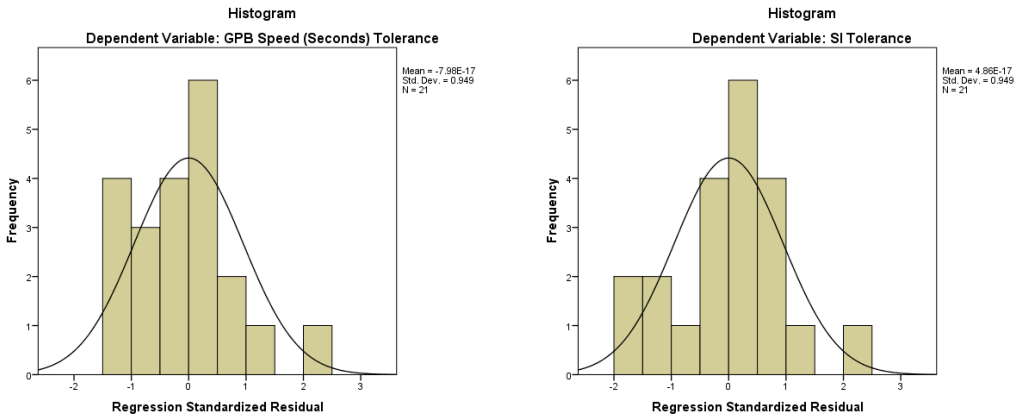


Figure 4: Histogram plots of standardised regression residuals for each regression model (ZZTR Speed, Accuracy, GPB Speed and SI outcome variables).

12.4 LINEARITY AND HOMOSCEDASTICITY

Standardised residual against standardised predicted values plots were produced to assess the relationship between predictor variables (positive and negative outcome expectancy scores) and outcome variables (magnitude of tolerance). The plots were also used to test the assumption of homoscedasticity. These plots (*Figure 2*) show that the relationship between the predictor and outcomes variables in each regression model were approximately linear and that the assumption of homoscedasticity was not violated.

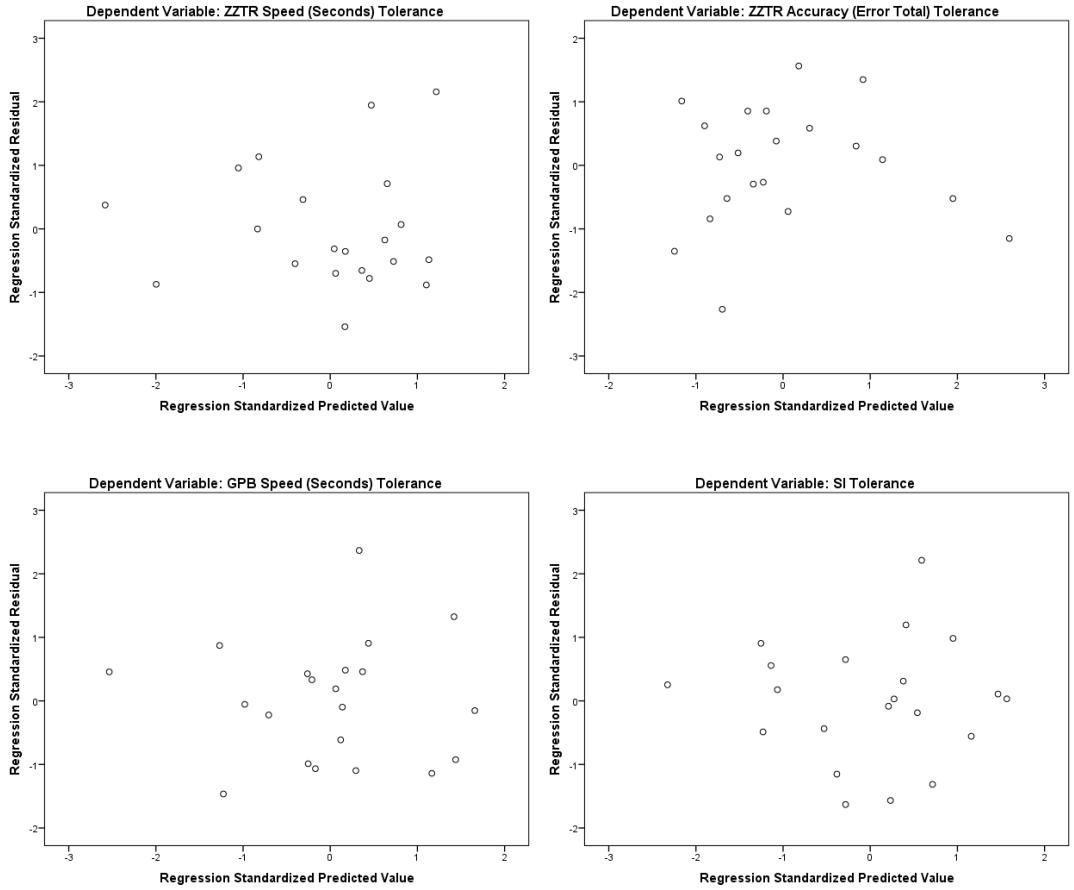


Figure 2: Scatterplots showing the standardised residual values against standardised predicted values for each regression model (ZZTR Speed, Accuracy, GPB Speed and SI outcome variables).