

**EXAMINING THE RELATIONSHIP BETWEEN ALCOHOL
INTOXICATION, STRESS RESPONSE AND TENSION
REDUCTION ALCOHOL EXPECTANCIES**

by

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Abstract

Stress contributes to both the initiation and maintenance of drug use. Drug intake, specifically alcohol, may be reinforced under stressful conditions by reducing anxiety or tension. The pharmacological effects of alcohol, however, cannot account entirely for the tension-reducing experience of intoxication. This suggests that cognitive factors contribute to the stress-dampening effects of alcohol. This study examined this hypothesis by testing how tension-reduction alcohol expectancies moderate the relationship between stress and alcohol intoxication. Stress response was operationalized as an increase in subjective anxiety and impaired sustained attention. Verbal learning, which was hypothesized to not be impaired by the stressor, was used as a cognitive control. One hundred and nine undergraduate students were randomly assigned to one of five groups (low, medium or high dose alcohol; sober; or placebo). Following beverage consumption, participants completed cognitive tasks before and after the Trier Social Stress Test. Participants completed the State-Trait Anxiety Inventory – State upon arrival in the lab, as well as pre- and post-stressor. They also completed the State-Trait Anxiety Inventory – Trait and the College Drinking Influences Survey, which includes a Stress Reduction scale. Social stress did not hinder cognitive performance, whereas alcohol impaired sustained attention and verbal learning abilities. The stressor evoked a subjective stress response that was reduced by alcohol and the expectancy of alcohol (i.e., placebo). There was no evidence to suggest that tension reduction alcohol expectancies moderated this effect. These findings replicate alcohol's ability to dampen a stress response and, furthermore, demonstrate that the expectancy of alcohol is as effective as

the drug itself in reducing subjective response to stress. This study highlights the need for further research to elucidate which factors modulate the stress-dampening effect of alcohol in undergraduate students. This knowledge, in turn, could present an opportunity for screening and early interventions to circumvent problem drinking as alcohol consumption is used by this population to cope with stress.

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Chapter 1

Introduction

Stress

Stress, defined simply as the body's response to a challenge or 'stressor' (Selye, 1936), may be internal or external to the individual. The stressor, either real or perceived, initiates a complex and highly coordinated set of physiological changes (Boyce & Ellis, 2005; Schlotz et al., 2008). These include increased heart rate (Dobkin & Pihl, 1992), accelerated breathing (Grossman, 1983), dry mouth, and an elevation in circulating hormones (Kirschbaum & Hellhammer, 1994) and immune factors, such as interleukin-6 (Steptoe, Hamer, & Chida, 2007). These physiological changes mobilize the body's "fight or flight" response in the face of a threat or challenge (Cannon, 1915). They are mediated through two major stress pathways: the hypothalamic adrenal pituitary (HPA) axis and the sympathetic-adrenal-medullary (SAM) system. The HPA axis involves the release of cortisol, a glucocorticoid hormone, by the adrenal cortex (Kirschbaum & Hellhammer, 1994), whereas SAM system activation is typically marked by the enzyme, alpha amylase (Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996).

In addition to these physiological changes, the stress response is characterized by altered affective and cognitive states. For example, stress evokes worry, anger and tension (Schlotz et al., 2008), which are typically assessed using self-report measures.

These subjective states overlap with hormonal (e.g., cortisol) responses, suggesting that the two measures tap the same theoretical construct (Neufeld & Davidson, 1974; Schlotz et al., 2008). Stress also disrupts cognitive processes, such as attention and memory, an effect which is attributed, at least in part, to elevated cortisol levels (Bohnen, Houx, Nicolson, & Jolles, 1990; Kirschbaum, Wolf, May, & Wippich, 1996). Impaired performance on some cognitive tasks, therefore, provides an alternative measure of stress responsivity (Zack, Poulos, Aramakis, Khamba, & MacLeod, 2007).

Stress responses may be examined in the laboratory using artificial or naturally-occurring real-life events, which can be physical or social. A common laboratory test of physical stress is the cold pressor test, which involves submerging a participant's hand and forearm in cold (0 – 4 degrees Celsius) water for 1 – 3 minutes (Lovallo, 1975). Social stress is often induced in the lab using the Trier Social Stress Test (Kirschbaum, Pirke, & Hellhammer, 1993). This manipulation produces a robust stress response by asking individuals to deliver an impromptu speech and perform mental arithmetic in front of an affectively neutral audience (Kirschbaum et al., 1993). Interestingly, responses to these two stressors and to others produce comparable somatic and subjective effects (Lewinsohn, 1956; McRae et al., 2006; Schlotz et al., 2008). This generalizability of a stress response to a wide array of stressors is ecologically adaptive: our bodies mobilize physiological resources in response to a perceived risk without assessing the specific nature of the threat.

Stress and Alcohol Abuse

Disrupted responses to stress are associated with a number of psychiatric conditions. For example, dysfunctional HPA axis responses are observed in psychosis (Mondelli & Pariante, 2008), anxiety disorders (Petrowski, Herold, Joraschky, Wittchen, & Kirschbaum, 2010), and major depression (Chopra et al., 2009). HPA axis disturbance is also associated with addictive behaviour, including pathological gambling (Paris, Franco, Sodano, Frye, & Wulfert, 2010) and substance abuse (Koob & Kreek, 2007). Not surprisingly, comorbidity amongst the disorders with HPA axis response dysregulation is common: 20-50% of addicted individuals have disorders related to anxiety and/or depression (Rounsaville, 1982). The connection between stress and addiction is apparent in alcoholics in that the number of stressful life events is positively correlated with alcohol abuse (Linsky, Strauss, & Colby, 1985). Even in healthy populations, stress is associated with drug use: non-pathological alcohol use increases following stressful events, such as divorce and financial hardship (Jose, van Oers, van de Mheen, Garretsen, & Mackenbach, 2000), and the introduction of an artificial stressor increases ethanol consumption in rats (Hansen, Fahlke, Soderpalm, & Hard, 1995) and nonhuman primates (Higley, Hasert, Suomi, & Linnoila, 1991). This combined evidence has led to the suggestion that abnormal stress responses are a risk factor for the establishment and perpetuation of addiction (for review see Kreek, Nielson, Butelman, & LaForge, 2005). Although the link between altered stress responsivity and risk for addiction is well-supported, the relationship between the two and the mechanisms that explain this

connection remain elusive.

One hypothesis to explain the relationship between stress and alcohol misuse relates to the notion that the anxiolytic effects of alcohol are reinforcing because subjective anxiety decreases following alcohol intoxication (Polivy, Schueneman, & Carlson, 1976). This is consistent with Conger's Tension Reduction Hypothesis, which states that alcohol is rewarding in its ability to reduce an internal aversive drive (Conger, 1956). More recently, the Stress Response Dampening (SRD) Theory proposed that the stress-reducing effects of alcohol constitute a rewarding property of the drug that reinforces future use of alcohol following a stressor (Sher & Levenson, 1982). In support of this contention, alcohol use is a commonly reported method for coping with life stressors and is one of the strongest predictors of alcohol abuse (Cooper, Russell, & George, 1988). Both alcoholics and social drinkers report using alcohol for the express purpose of reducing anxiety (Boys, Marsden, & Strang, 2001; Wanberg, 1969). Similarly, college students increase their alcohol consumption on days that they perceive to be more stressful and on days when problem-focused coping is low (Park, Armeli, & Tennen, 2004). Furthermore, alcoholics and problem drinkers report expecting more relaxation and tension reduction effects from alcohol compared to normal drinkers, which is consistent with tension-reduction models of alcoholism (Brown, Goldman, & Christiansen, 1985b). These data demonstrate that some individuals, including university students, seek out alcohol to directly manage the effects of stress.

An assumption of tension-reduction theories is that alcohol reduces tension through a

pharmacological action on biological systems that mediate stress. This idea, however, is difficult to reconcile with findings that stress responses increase, decrease or do not change with alcohol intoxication (Greeley & Oei, 1999). Furthermore, compared to a sober group, intoxicated and placebo groups both show attenuation of self-reported anxiety and stress hormone levels (Balodis, Potenza, & Olmstead, 2009a) and consume more alcohol following a stressor (de Wit, Soderpalm, Nikolayev, & Young, 2003). Thus, the pharmacological effects of alcohol cannot account, entirely, for the stress-dampening effects of the drug. Individual differences in other factors such as a family history of alcoholism (Croissant & Olbrich, 2004) or personality traits (Sher & Levenson, 1982) may contribute to the stress-reducing effects of alcohol. In addition, cognitive factors likely contribute to placebo-induced effects because the relationship between alcohol intoxication and tension reduction involves prior beliefs about the subjective and behavioural effects of alcohol. This is consistent with the basic concept of alcohol expectancy, which suggests that the experience of alcohol intoxication is an interaction between drug effects and the beliefs held about those effects (Campbell & Oei, 2010).

Alcohol Expectancies

Alcohol expectancies are broadly defined as beliefs about the perceived positive and negative effects of alcohol on behaviour, cognitions and emotions (Brown, Goldman, Inn, & Anderson, 1980; Neighbors, Walker, & Larimer, 2003; Sher, Wood, Wood, & Raskin, 1996). Alcohol expectancy effects were clearly demonstrated over three decades ago in a

seminal study assessing the effect of beverage type – alcoholic versus placebo – on *ad libitum* drinking in alcoholics and social drinkers (Marlatt, Demming, & Reid, 1973). The authors proposed that, “if it could be shown that regardless of the actual content of the beverage administered (alcoholic or nonalcoholic), the individual's expectancy of the alcoholic content of the drink is a significant determinant of his drinking rate, doubt would be cast on the theoretical position which accounts for loss of control [sic] drinking primarily as a physiologically mediated effect” (Marlatt et al., 1973). Indeed, the only significant predictor of consumption in this study was the expectancy of beverage content: both alcoholics and social drinkers consumed more if they believed they were receiving alcohol (i.e., both alcohol and placebo groups), compared to a group that was told they would not be receiving alcohol. Since then, the use of a placebo condition has become an important, widely-used and much-discussed (Testa et al., 2006) feature of alcohol studies.

The study of alcohol expectancies now extends beyond the use of placebo groups to include formal assessment of specific alcohol expectancies. A variety of tools have been developed to assess these, including the Alcohol Expectancies Questionnaire (Brown et al., 1980), the Drinking Expectancy Questionnaire and the Comprehensive Effects of Alcohol questionnaire (Fromme, Stroot, & Kaplan, 1993), among others. Of these, the AEQ is arguably the most well-established and widely-used tool to assess alcohol expectancies (Brown et al., 1980). The AEQ is a 90-item measure of positive expectancies regarding alcohol's effects that yields 6 factors: (1) global; (2) sexual

performance; (3) physical and social pleasure; (4) increased social assertiveness; (5) relaxation and tension reduction; and (6) arousal and power (Brown et al., 1980). Despite its widespread use, the AEQ has been criticized for a lack of theoretical construct in its development, as well as psychometric shortcomings, such as questionable test-retest reliability (Corcoran & Parker, 1989). Moreover, the tension reduction scale of the AEQ does not predict tension reduction following administration of an alcoholic or placebo beverage (Corcoran, 1994). Addressing these shortcomings, Fisher and colleagues developed the College Drinking Influences Survey (CDIS) which measures drinking expectations as well as psychosocial influences and values (Fisher, Fried, & Anushko, 2007). The CDIS is tailored for a college population making it an appropriate tool for assessing alcohol expectancies in university students. This population exhibits high rates of alcohol consumption and problem drinking (Balodis, Potenza, & Olmstead, 2009b), so it is becoming increasingly important to understand the factors that contribute to this maladaptive behaviour.

Alcohol expectancies appear to be well-established even before an individual's first drinking experience (Christiansen, Goldman, & Inn, 1982). This suggests that the development of alcohol expectancies is more closely related to social learning than to experience with the intoxicating effects of the drug. Nonetheless, alcohol expectancies become increasingly developed with greater drinking experience (Christiansen et al., 1982). Once drinking has been initiated, expectancies mediate alcohol use in that, as the strength of alcohol expectancies increases, the level of alcohol consumption increases in

both pathological and non-pathological populations (Brown, Goldman, & Christiansen, 1985a). This finding has been replicated in a college student population (Southwick, Steele, Marlatt, & Lindell, 1981). In fact, among college students, alcohol expectancies are predictive of drinking behaviour beyond variance accounted for by demographic variables, such as gender, religiosity, or socioeconomic status (Brown, 1985).

Predictably, alcoholics hold stronger expectancies about the positive effects of alcohol compared to non-pathological drinkers (Brown, Goldman, & Christiansen, 1985a).

One of the most common expectancies of alcohol effects is relaxation or tension reduction (Brown et al., 1980). Tension reduction alcohol expectancies are reported by early adolescents, even those with little to no experience with alcohol (Christiansen et al., 1982). Importantly, these particular expectancies are predictive of high-risk drinking (Mann, Chassin, & Sher, 1987). College students who are moderate-to-heavy drinkers report greater tension reduction alcohol expectancies than light drinkers (Rohsenow, 1983). In fact, Brown and colleagues (1985) found that tension reduction alcohol expectancies were the single best predictor of problem drinking in a college sample (Brown, 1985). Tension reduction alcohol expectancies also appear to mediate the stress response-dampening effects of alcohol. For example, among participants with social phobia – a population that demonstrates a marked response to psychosocial stressors – those with high expectancies experience greater anxiety reduction following consumption of a placebo beverage (Abrams & Kushner, 2004).

Further evidence for a tension reduction effect of alcohol is that both laboratory and

epidemiological evidence indicates that differences in tension reduction expectancies modulate drinking behaviour. On days that are perceived to be more stressful, men who strongly endorse tension reduction expectancies, operationalized as “careless unconcern” (p. 989; S. Armeli, Carney, Tennen, Affleck, & O'Neil, 2000), demonstrated an increased subjective desire to drink as well as elevated alcohol consumption. Conversely, men who expect negative outcomes from drinking, such as greater impairment, drank less on stressful days. Alcohol expectancies have also been closely linked to drinking behaviour in college samples (Sher et al., 1996): compared with other types of expectancies, tension reduction expectancies were the strongest predictor of problem drinking in students (Brown, 1985). In contrast, expectancies related to social and physical effects of alcohol were related to frequent, but not problematic, drinking in this population (Brown, 1985). Importantly, students who specifically use alcohol for coping purposes report higher weekly drinking frequency and more heavy-drinking days compared to students who use other coping strategies (Britton, 2004). Thus, tension reduction expectancies of alcohol may mediate the alcohol intoxication-stress reactivity relationship, which may contribute to risk for drinking initiation and subsequent abuse.

In sum, researchers and theorists have long suggested that cognitive factors must be considered in any explanation or description of alcohol use (Roehling & Goldman, 1987). More specifically, many cognitive theories propose that the transference of alcohol cognitions – alcohol expectancies, in particular – contributes to familial transmission of alcohol use, misuse and abuse (Campbell & Oei, 2010). Alcohol expectancies, therefore,

may be an important determinant of drinking behaviour (Armeli et al., 2000) and a promising direction for understanding and treating alcohol abuse in particular, and addiction in general.

Current Study

Stressful events or situations produce a highly organized set of responses, involving physiological, affective and cognitive changes (Boyce & Ellis, 2005; Schlotz et al., 2008). This stress response is linked to drinking behaviour in non-pathological populations, as well as the development and maintenance and alcohol use in substance abusers (Kreek et al., 2005). The relationship between stress and drinking may be moderated by the tension-reducing effects of the drug (Polivy et al., 1976), which appear to be a motivator for alcohol use in some individuals (Boys et al., 2001; Cooper et al., 1988; Wanberg, 1969). The fact that stress reduction occurs in experiments with a placebo condition (Marlatt et al., 1973; Testa et al., 2006) suggests that cognitive factors, particularly alcohol expectancies, are a critical component in the tension-reducing effects of alcohol.

Over two decades ago, Roehling and Goldman (1987, p. 109) asked the question: “Do people actually receive the benefits from alcohol consumption that they report anticipating?” The current study endeavoured to answer this question as it relates to tension-reduction expectancies of alcohol. Specifically, this study focused on how expectancies of tension-reduction affect stress reactivity following a psychosocial

stressor in university students.

Stress reactivity was measured in two modalities: subjective and cognitive. Cognitive stress reactivity is operationalized as impairment in psychometric testing, specifically in a sustained attention (i.e., vigilance) test. Previous findings demonstrate that vigilance performance is inversely related to stress, specifically a distressing state (Shaw et al., 2010), an effect that is attributed to elevated cortisol levels (Bohnen, Houx, Nicolson, & Jolles, 1990; Bohnen & Jolles, 1992). I expect that performance on this task will be impaired following the stressor. Previous studies did not observe an effect of alcohol on sustained attention, even at high doses (0.8 g/kg; Koelega, 1995a), therefore alcohol is not expected to impair this ability. An auditory verbal learning test was used as a stress control as performance on this task is not impaired by psychosocial stressors (Hoffman & al'Absi, 2004). By contrast, alcohol impairs performance on verbal list learning tasks (Birnbaum, Parker, Hartley, & Noble, 1978); therefore I predict that an effect of alcohol will be observed on this task. Subjective stress was measured using a self-report questionnaire of state anxiety. Trait anxiety was also assessed in order to examine its contribution, relative to the experimental manipulations (i.e., stress and alcohol administration), to subjective stress. The tension-reduction effects of alcohol were assessed using a measure of college drinking expectancies, psychosocial influences and values. Sex differences have been identified with respect to tension reduction, with men reporting higher levels than women in a college sample (Rohsenow, 1983); I expect to replicate this sex difference. The stressor was a validated psychosocial stressor and the

beverage administration was a validated protocol previously used in our laboratory.

Hypotheses regarding the primary outcomes of the study are as follows:

- (1) The stressor will elicit subjective and cognitive stress responses.
- (2) Alcohol will reduce stress responses in a step-wise fashion with placebo, low, moderate and high dose alcohol groups showing reductions across both measures.
- (3) Tension reduction expectancies of alcohol will moderate the effects of alcohol, in both the alcohol and placebo groups.

The findings from the present study will contribute to our understanding of the cognitive factors that contribute to the stress-dampening effects of alcohol, which are known to contribute to the reinforcing effects of the drug.

Chapter 2

Methods

Participants

Power analysis for the moderator analysis – repeated measures analysis of variance (ANOVA) interaction - was conducted using the program G*Power 3.1.2 (Faul, Erdfelder, Lang, & Buchner, 2007). After inputting the effect size (small; Cohen's $d = 0.30$), power (0.95), number of groups (5), and number of measurements (2) as well as correlation between measures (Pearson's $r = 0.03$), calculations revealed that a sample size of 105 was required. Participants were 56 male and 53 female university undergraduate students, which exceeds the required sample size per the power analysis. Participants were recruited using the introductory psychology subject pool, as well as print advertisements on campus. To be eligible for participation, students were required to be at least 19 years of age (the current legal drinking age in Ontario) and had to report drinking alcohol at least once per month on a pre-screening questionnaire, in order to ensure that they were familiar with drinking and alcohol consumption would not be novel. Exclusion criteria included a previous medical history contraindicating the use of alcohol, allergy to alcohol and/or use of medication that may interact with the effects of alcohol. Due to the deleterious effects of alcohol use during pregnancy, women were only permitted to participate if they were menstruating at the time of testing, or had not had sexual intercourse since their last menstruation. Participants were randomly assigned to

one of five beverage consumption groups – sober ($n = 36$; 22 male, 14 female), placebo ($n = 22$; 10 male, 12 female) or alcohol which included low ($n = 17$; 6 male, 11 female), medium ($n = 16$; 6 male, 10 female) and high ($n = 18$; 12 male, 6 female) doses – using a random number generator. Due to the fact that single participants were automatically placed in the sober condition (see details regarding alcohol administration, below), this group grew at a more rapid rate than the other groups; as such, once the current number for the sober group had been reached, subsequent random assignment was between the four remaining groups. Participants were requested to fast for three hours prior to testing in order to minimize variability in the rate of alcohol absorption. Participants were compensated with course credit through the Psychology Subject Pool or cash remuneration (\$10).

Self Report Measures

College Drinking Influences Survey

The College Drinking Influences Survey (CDIS; Fisher, Fried, & Anushko, 2007) is a 53-item questionnaire that assesses factors affecting the decision to drink. It is comprised of three distinct scales that each use a five-point Likert scale (strongly disagree to strongly agree) to measure drinking expectations, psychosocial influences, and values. (1) The first scale, the Psychosocial Drinking Inventory (PDI), measures psychosocial influences on drinking behaviour by asking participants to rate 51 items corresponding to 3 types of psychosocial drinking influences: Social Influences (e.g., “If I were at a party and other people were drinking”); Stress Reduction (e.g., “If I was

worried about family problems at home”), and Sensation Seeking (e.g., “If there were a chance I would take sexual risks when I got drunk”). These provide information on how likely individuals are to drink given a particular situation (extremely unlikely to drink – extremely likely to drink), (2) The Drinking Values Scale (DVS) measures personal, social and moral value systems that impact the decision to drink. Participants rate their agreement (strongly disagree to strongly agree) with 19 statements corresponding to 3 types of drinking expectations: Social Responsibility (e.g., “Drinking to excess is wrong because it can have unfavourable effects on others”); Personal Choice (e.g., “How much a person drinks isn’t a matter of right or wrong but a matter of personal choice”); and Institutional Obligations (e.g., “The university has an obligation to parents of students to monitor and enforce drinking policies”). (3) The third scales measures drinking expectations using the College Drinking Expectancies Scale (CDES). This questionnaire asks participants to rate 16 statements corresponding to 2 types of drinking expectations: Drinking Norms (e.g., “Drinking is a normal part of college life”) and Drinking Consequences (e.g., “If I drink I may do things I would not ordinarily do”), based on how much they agree with the particular statement. All three scales of the CDIS have demonstrated strong psychometric properties. For example, the subscales maintain good test-retest ($r = 0.3 - 0.78$) and inter-item ($\alpha = .71-.94$) reliability among undergraduate students, and all demonstrate concurrent validity with the AEQ (Fisher et al., 2007). Furthermore, the CDIS is appropriate for the study as it has specific applicability to a college population (Fisher et al., 2007).

State-Trait Anxiety Inventory

The State-Trait Anxiety Inventory (STAI; Appendix A) is a questionnaire measuring feelings associated with anxiety, namely tension, apprehension, nervousness, and worry (Spielberger, 1983) using a four-point Likert scale. The STAI-Trait (STAIT) is a measure of “trait” anxiety (long-standing individual quality) that asks respondents to rate twenty items (e.g., “I am ‘calm, cool and collected’.”). The STAI-State (STAIS) is a twenty-item (e.g., “I am tense.”) measure of “state” anxiety (i.e., how the participant feels “at this moment”). The STAIT demonstrates good test-retest reliability among college students, with correlations ranging from $r = 0.73 - 0.86$. The STAIS demonstrates low test-retest reliability in the same population ($r = 0.31 - 0.33$), which is what one would expect when measuring transient state anxiety (Spielberger, 1983). This measure also demonstrates adequate internal consistency, with alpha coefficients of 0.92 and 0.90 for the STAIS and STAIT, respectively (Spielberger, 1983). Validity of this measure is supported in that STAIS scores increase significantly in stressful, compared to neutral conditions, while the STAIT remains unchanged (Spielberger, 1983).

Perceived Intoxication Questionnaire

To measure the perceived level of alcohol intoxication, participants completed a modified version of the Drug Effects Questionnaire (de Wit et al., 2003; Ortner, MacDonald, & Olmstead, 2003) after the final drink was consumed (see Appendix B). This brief self-report questionnaire asks participants to estimate how much alcohol they

have consumed (bottles of beer), how intoxicated they feel, how much they enjoy how they feel and the extent to which they want more alcohol (scales 1 – 9). This questionnaire served as a manipulation check to assess the effectiveness of the placebo condition.

Behavioural Measures

Continuous Performance Test

The Continuous Performance Test (CPT) is a measure of sustained attention (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). It is commonly used to identify deficits in attentional capacity, including those seen in pathological populations such as schizophrenia and attention deficit hyperactivity disorder (Groom et al., 2008). The basic design involves serial presentation of stimuli during which participants are instructed to respond to a single stimulus (e.g., a blue dot) or to one stimulus followed by another (e.g., a blue dot followed by a red dot), which typically remains constant across the test (Rosvold et al., 1956). The CPT variant used in this study was the Immediate Memory Task (IMT) in which five-digit numbers were serially presented in the middle of a computer screen for 0.5 seconds with a 0.5 second delay between presentations. Participants were required to click the mouse button when two consecutive numbers were identical (Dougherty, Marsh, & Mathias, 2002). The IMT has a lower cognitive load than other CPT variants, so is not typically impaired by alcohol (Dougherty et al., 1999). On each trial of the IMT, black digits (2 cm x 3.3 cm each) were presented against a white

background and there was an equal probability of target stimuli (identical to the preceding number), catch stimuli (differing from the previous number by only one, randomly-positioned digit), and filler stimuli (novel numbers) being presented. Filler stimuli always appeared following target or catch stimuli presentations.

The IMT yields various measures that tap different cognitive processes: correct detections, commission errors, filler errors, discriminability and signal detection. Correct detection occurs when a participant responds appropriately to the target stimulus; the number of correct detections is a reflection of sustained attention (Dougherty, Marsh, & Mathias, 2002). Commission errors occur when a participant responds to ‘catch’ stimuli – those stimuli which are visually similar, but not identical, to the target stimulus. A higher frequency of commission errors is associated with impulsivity. For example, populations with impulse control problems, such as men with a history of conduct disorder, demonstrate higher rates of commission errors compared to normal controls (Dougherty, Bjork, Marsh, & Moeller, 2000b). Furthermore, this behaviour is significantly correlated with self-reported impulsivity, as measured by the Barratt Impulsivity Scale (Dougherty, Bjork, Marsh, & Moeller et al., 2000b). Filler errors are responses made to novel stimuli; this measure was used to screen for incorrect completion of the task as filler error rates above 2-3% may indicate that a participant did not understand the task requirements. Discriminability (d') is a parametric measure of signal-to-noise that is operationalized as the ratio between commission errors and correct detections, where higher d' values represent better discrimination. Beta is a parametric measure of response bias (Dougherty

& Marsh, 2002). We also used two nonparametric measures: A' (discriminability) and B_d' (response bias), which were developed by Donaldson (Donaldson, 1992) based on the Signal Detection Theory. A' is represented by the equation:

$$A' = \frac{1 + (\text{hits} - \text{false alarms})(1 + \text{hits} - \text{false alarms})}{4 \times [\text{hits}(1 - \text{false alarms})]}$$

where hits are correct detections and false alarms are commission errors. Scores on this measure range from -1 to 1, with higher values representing greater discriminability.

B_d' measures response bias and is represented by the equation:

$$B_d' = \frac{(1 - \text{hits})(1 - \text{false alarms}) - (\text{hits})(\text{false alarms})}{(1 - \text{hits})(1 - \text{false alarms}) + (\text{hits})(\text{false alarms})}$$

with scores ranging from -1 (liberal strategy) to 1 (conservative strategy).

Hopkins Verbal Learning Test

The Hopkins Verbal Learning Test (HVLT) is a widely-used measure of verbal learning and memory, involving free recall of words (Brandt, 1991). The task uses a list of 12 words that, although randomly ordered, form three semantic groups (e.g., dwellings, furniture). The administrator reads the words at a constant rate with two seconds separating each word and the participant is required to repeat as many words as

they can remember, in any order. This process is repeated twice using the same list, each subsequent trial beginning immediately after the participant can no longer identify novel words. Each three-trial task takes approximately five minutes to complete. Participants receive a global score of all correct words identified across the three trials (maximum possible score of 36). Repetitions and intrusion errors are scored as incorrect responses.

Alcohol Administration

Alcohol was administered in accordance with a protocol previously established in our lab (Ortner et al., 2003). Participants in the alcohol and placebo groups were weighed at the beginning of the session. The alcohol groups were administered alcohol (0.2, 0.5 or 0.8 g/kg) across three drinks composed of calorie-free soda and 40% alcohol vodka in a 2:1 ratio. The placebo group was given flattened tonic water in a glass rimmed with vodka, which lends the odour of alcohol without increasing the individual's Breath Alcohol Level (BAL). The sober group was given calorie-free soda alone. The alcohol and placebo groups were both told that they were receiving alcohol and the sober group was told that the beverage was soda. Blood alcohol concentrations were estimated through BAL using the Intoxilyzer 400D (CMI Inc., Owensboro, KY), a handheld breath alcohol testing instrument. BAL was used as an indicator of intoxication for the different alcohol groups, not a specific measure of alcohol level; as such, BAL was not included as a factor in statistical analyses. Participants blew air through a mouthpiece into a fuel cell which measured the alcohol concentration in the expired breath.

Stressor

The Trier Social Stress Test (TSST) was used to induce a stress response in participants (Kirschbaum et al., 1993). The TSST is a psychosocial stressor that capitalizes on highly stressful factors including uncontrollability, forced failure and social-evaluative threat. The procedure reliably elicits elevations in stress measures in healthy participants, (Kudielka, Wust, Kirschbaum, & Hellhammer, 2007) including a robust HPA axis response with a long recovery time (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999).

In this task, each participant performed a 5-minute speech in front of a panel of student actors, followed by a 5-minute mental arithmetic task. The panellists were introduced to the participant as a doctoral student in linguistics, who specializes in non-verbal behaviour, and a professor. The participant was asked to give a mock job talk for a position as a research assistant in the Psychology department. The participant was given a pen and paper and allowed five minutes to prepare. When the preparation time had expired, any notes the participant had made were taken away and the individual was told that his/her performance would be compared against the written information. All participants were able to see their image on the LED screen of a camcorder; performances were recorded for the 91 individuals who specifically consented to having these data captured. The two panellists watched the participant intently and made notes during their speech. Throughout the performance, the audience maintained an affectively

neutral appearance and did not provide any positive feedback. If the participant stopped talking, the audience informed them of the amount of time remaining and instructed them to continue. The mental arithmetic task required participants to subtract a prime number serially from a four-digit number. Participants were instructed to maintain eye contact during this task, to speed up their delivery and to start over when they provided an incorrect number.

Procedure

The experimental protocol was approved by the General Research Ethics Board (GREB) of Queen's University. Individuals were pre-screened via email to ensure they were eligible for the study and eligible participants were asked to come in to the lab for testing. Testing was conducted in singles or pairs between the hours of 1630 h and 2030 h. The timeline of the experimental procedure for each session is presented in Table 1. Within five minutes of arrival, eligibility and maintenance of fasting prior to the study were confirmed. Participants then completed a consent form and were asked to provide a saliva sample (S1). Saliva samples were collected for future analyses of changes in the hormone, cortisol, and the enzyme, alpha-amylase, two biochemical markers of stress. All participants then completed the STAIS, were weighed and provided a baseline BAL. Participants then gave the next saliva sample (S2). Before receiving their first beverage, participants were informed that they would be receiving alcohol (alcohol and placebo groups) or soda (sober group). Single participants were never administered alcohol to

maintain ecological validity, because drinking alone occurs rarely (~15%) among university students (O'Hare, 1990). Most importantly, individuals who drink alone, compared those who drink in a social context, show an exacerbated negative mood response to daily stressors (Armeli et al., 2000; Armeli et al., 2003). Therefore, drinking alone may create an aversive state, especially when participants are subsequently exposed to a stressor. Whereas all single participants were in the sober group, not all participants in the sober group were alone.

All participants consumed three beverages across 45 minutes. They were told to consume each beverage steadily over 10 minutes, and provided a saliva sample following each beverage (S3 – S5). Following beverage consumption, BALs of alcohol and placebo participants were measured (without showing the results to the participants) and participants completed the manipulation check. Participants then completed the CPT and HVLT, in different rooms so that the participant completing the CPT could not hear the words being read for the HVLT and would not be distracted. Following the cognitive tasks, they completed the STAIS again and provided another saliva sample (S6).

Administration of the cognitive tasks was counterbalanced. Then participants were taken to another room and told that they would be completing a task that tests their alertness before repeating the cognitive tasks. In this room, they were introduced to the stressor panellists and exposed to the TSST, which took approximately 20 minutes to complete. Immediately following the stressor, participants returned to the room to give their final saliva sample (S7) and complete another STAIS. Another version of the CPT and HVLT

were then repeated. If two participants were participating, cognitive tests were completed in a staggered fashion; depending on the order of stressor administration, participants completed an online questionnaire composed of the CDIS and STAIT, either while the other participant was undergoing the stressor or at the end of the experiment. Participants in the alcohol and placebo groups then gave another BAL reading. All participants were paid and thoroughly debriefed, including providing an explanation of the deception involved with the stressor and the placebo conditions. Participants in the alcohol groups were not permitted to leave until their BAL reached 0.06%. These individuals were provided with a taxi ride home.

Table 1. Timeline of the experimental procedure. *Note.* STAIS = State-Trait Anxiety Inventory - State; BAL = blood alcohol level; S1 – S7 = saliva sample 1 – saliva sample 7; TSST = Trier Social Stress Test; CPT = Continuous Performance Task; HVLT = Hopkins Verbal Learning Test

Time	Task
0 min	Arrival at lab. Consent given.
+2 min	S1
+6 min	STAIS 1. BAL. Body weight obtained.
+11 min	S2
+15 min	Beverage administration. S3 – S5.
+1 h	BAL 2. Manipulation check.
+1 h 15 min	Cognitive Tasks I (CPT, HVLT)
+1 h 30 min	STAIS 2. S6.
+2 h 10 min	Stressor (TSST)
+2 h 25 min	STAIS 3. S7.
+2 h 35 min	Cognitive tasks II (CPT, HVLT)
+ 2 h 45 min	Compensation. Debriefing.
+2 h 55 min	Questionnaires (Demographics, CDIS, STAIT)
	Sobering (alcohol condition)

Statistical Analyses

Data were analysed using SPSS version 18.0. Statistical significance was set at a probability of $p < 0.05$. The dependent measures were analysed as described below. When assumptions of homogeneity of variance for analyses were not met, the Huynh-Feldt correction was used.

Self Report Measures

Self-report measures (CDIS subscales, STAIS) were analyzed using separate one-way ANOVAs with group and sex as the between-subjects factors. Tukey's post-hoc analyses were used to examine differences among pairs of groups.

Alcohol Effects

Manipulation Check

Subjective ratings of perceived intoxication were analyzed using one-way analysis of variance (ANOVA) with scores on each question of the modified DEQ as the dependent variable and beverage group as the between-subjects factor. Tukey's post-hoc analyses were used to examine differences among pairs of groups.

Subjective Effects

STAIS scores were analyzed using a 2 x 5 repeated measures ANOVA with time (arrival in lab, following beverage consumption) as the within-subjects factor and beverage group as the between-subjects factor. When significant main effects were

observed in between-subjects analyses, Tukey post-hoc tests were performed. Regression analyses were used to examine the relationship between trait anxiety (STAIT) and state anxiety (STAIS) upon arrival in the lab and following beverage consumption, including examination of Pearson's correlations (r).

Cognitive Effects

Performance on the first set of cognitive tasks (i.e., following beverage consumption) was analyzed using one-way ANOVAs with beverage group as the between-subjects factor. Tukey's post-hoc analyses were used to examine differences among pairs of groups. The outcome measures for the CPT were correct detections, commission errors, discriminability/signal detection (parametric, d' ; and non-parametric, A') and response bias (parametric, β ; and non-parametric, Bd''). None of the participants exceeded the 3% cut off for filler errors so this variable was not analysed further. The dependent variable for the HVLT was total number of correct words across three trials (maximum score = 36).

Stress Effects

Subjective Effects

STAIS scores were analyzed using a 2 x 5 repeated-measures ANOVA with time (post-beverage/ pre-stressor, post-stressor) as the within-subjects factor and beverage group as the between-subjects factor. Tukey's post-hoc analyses were used to examine differences among pairs of groups. Regression analyses were used to examine the

relationship between trait anxiety (STAIT) and state anxiety (STAIS) pre- and post-stressor, including examination of Pearson's correlations (r).

Cognitive Performance

Performance on the cognitive tests was analyzed using 2 x 5 repeated-measures ANOVAs with time (pre-stressor, post-stressor) as the within-subjects factor and beverage group as the between-subjects factor. Tukey's post-hoc analyses were used to examine differences among pairs of groups. The outcome measures for the CPT were correct detections, commission errors, discriminability/signal detection (parametric, d' , and non-parametric, A') and response bias (parametric, β , and non-parametric, Bd''). The dependent variable for the HVLIT was total number of correct words across three trials (highest possible total = 36).

Cognitive Moderator of Stress Response

For data in which a stress-dampening effect of alcohol was seen, a 5 (beverage group) x 3 (median-split Stress Reduction scale) x 2 (time) ANOVA was conducted, with the latter factor as a repeated measure.

Chapter 3

Results

Participants

The participant sample was composed of 51% men ($n = 56$) and 49% women ($n = 53$) between the ages of 19 and 41 ($M = 20.7$, $SD = 2.98$). Participants reported drinking an average of 2.93 times per week ($SD = 1.23$). Men and women did not differ in the number of times they drank per week, $t(102) = 0.05$, $p = .96$; however, men reported consuming more drinks per session ($M = 4.37$, $SD = 1.68$) than women ($M = 3.62$, $SD = 1.39$), $t(103) = 2.44$, $p < .05$.

Self Report Measures

The experimental groups (sober, placebo, low dose, medium dose and high dose) did not differ in their ratings on the CDIS subscales (all p -values $> .05$; see Appendix C). As shown in Table 1, there was a significant sex difference on the Social Influences subscale within the PDI, with men reporting greater social influences on their decision to drink than women. Similarly, on the Drinking Consequences subscale, men had greater expectations of negative outcomes related to drinking compared to women. Men and women did not differ on any of the other subscales of the CDIS (all p -values $> .05$).

Table 2. Mean (standard error of the mean (SEM)) scores on the College Drinking Influence Scale subscales for male and female participants. F and p vales from the one-way ANOVAs are presented in the final two columns. *Note.* PDI = Psychosocial

Drinking Inventory. DVS = Drinking Values Scale. CDES = College Drinking Expectations Scale.

Subscale	Men (<i>n</i> = 56)	Women (<i>n</i> = 53)	<i>F</i>	<i>p</i>
PDI				
Social Influences	3.01 (0.06)	2.67 (0.08)	12.29	.001*
Stress Reduction	1.87 (0.06)	1.86 (0.07)	0.01	.94
Sensation Seeking	3.03 (0.07)	2.92 (0.08)	1.06	.31
DVS				
Social Responsibility	3.02 (0.13)	3.20 (0.11)	1.08	.30
Personal Choice	4.12 (0.09)	4.04 (0.08)	0.43	.51
Institutional Obligation	3.39 (0.12)	3.54 (0.09)	0.99	.32
CDES				
Drinking Norms	3.31 (0.07)	3.98 (0.08)	0.01	.90
Drinking Consequences	4.26 (0.07)	3.98 (0.09)	5.62	.02*

There were no group, $F(4, 99) = 0.15, p = .96$, or sex, $F(1, 102) = 3.30, p = .07$, differences on the STAIT. The average STAIT score was 39.16 ($SD = 9.29$) with scores ranging from 24 to 64.

Alcohol Effects

Intoxication

BALs and subjective ratings of intoxication are shown in Table 2. A one-way ANOVA showed a main effect of group on BAL, $F(4, 103) = 83.60, p < .001$. The low, medium and high dose groups were significantly different from one another, and all three were

significantly different from the sober and placebo groups (all p -values $< .05$). There was no significant difference in BALs of sober and placebo groups ($p = 1.00$).

Table 3 also shows self-reports of perceived intoxication levels in the placebo and alcohol groups. The sober group is not included in this table because they did not provide reports of perceived intoxication and BAL levels in this group were 0%. There was a main effect of group on subjective feelings of intoxication, $F(3, 69) = 5.83, p = .001$, with the placebo group ratings being significantly lower than all three alcohol groups (post-hoc p -values $< .05$). Subjective ratings of intoxication did not differ significantly between the low, medium and high dose groups (p -values $> .05$).

There was a main effect of group on the estimation of alcohol consumed, $F(3, 69) = 10.15, p < .001$. The placebo group did not differ significantly from the low dose group ($p = 1.00$), but was significantly lower than the medium ($p < .05$) and high ($p < .001$) dose groups. The low dose group had significantly lower estimations than the medium ($p < .05$) and high ($p = .001$) dose groups, but the medium and high dose groups did not differ significantly on this measure ($p = .78$).

By contrast, there was no effect of group on participants' estimation of their BAL, $F(3, 69) = 0.77, p = .51$. Similarly, the five groups did not differ significantly on how much they liked the effects they felt, $F(3, 69) = 1.80, p = .16$, or how much they wanted more alcohol, $F(3, 69) = 0.65, p = .58$.

Table 3. Mean (SEM) blood alcohol levels (BAL) and self-reports of perceived intoxication in placebo and alcohol groups. Participants estimated the alcohol they had consumed in terms of the number of beer bottles. In addition, they rated their level of

intoxication, how much they liked the effects they felt and how much they desired more alcohol (all scales 1-9).

Beverage Group	BAL (%)	Perceived Intoxication			
		Alcohol Consumed (Bottles of Beer)	Intoxication Level	Liking the Effects	Desiring More Alcohol
Placebo ($n = 22$)	0.00 (0.00)	3.82 (0.31)	2.59 (0.25)	6.32 (0.37)	6.04 (0.49)
Alcohol Low dose ($n = 17$)	0.02 (0.00)	3.88 (0.36)	3.94 (0.51)	7.12 (0.38)	5.35 (0.70)
Alcohol Medium dose ($n = 16$)	0.06 (0.01)	5.25 (0.25)	4.19 (0.40)	6.38 (0.36)	5.50 (0.71)
Alcohol High dose ($n = 18$)	0.09 (0.01)	5.67 (0.23)	4.56 (0.35)	7.22 (0.30)	6.44 (0.59)

Anxiety

There was no main effect of group (i.e., no effect of alcohol), $F(4, 98) = 0.58, p = .68$, on STAIS ratings combined across the arrival and post-beverage consumption sampling points. In contrast, the main effect of time reached statistical significance, $F(4, 98) = 3.96, p = .05$, in that STAIS scores decreased significantly from arrival to post-beverage consumption (see Figure 1). The time x condition interaction was not statistically significant, $F(4, 98) = 2.26, p = .07$.

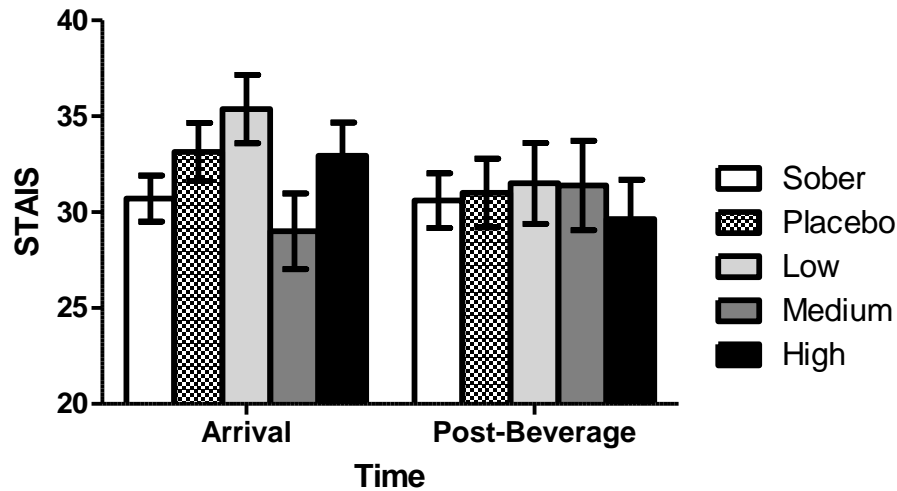


Figure 1. Effect of alcohol on state anxiety. Bars represent mean (+SEM) total STAIS scores of participants upon arrival in the lab and following beverage consumption for sober, placebo and alcohol (low medium and high dose) groups. *Note.* STAIS = State-Trait Anxiety Inventory – State version.

Figures 2 and 3 show that trait anxiety (i.e., STAIT scores) was significantly correlated with state anxiety (STAIS) upon arrival in the lab, $r = 0.58, p < .001$, and following beverage consumption, $r = 0.50, p < .001$. In addition, state anxiety upon arrival in the lab was a significant predictor of state anxiety following beverage consumption, even controlling for trait anxiety (STAIT), $F(2, 95) = 27.91, p < .001$. Trait anxiety accounted for 25% of the variance in post-beverage anxiety and state anxiety upon arrival in the lab accounted for 12% of the variance above the effect of trait anxiety.

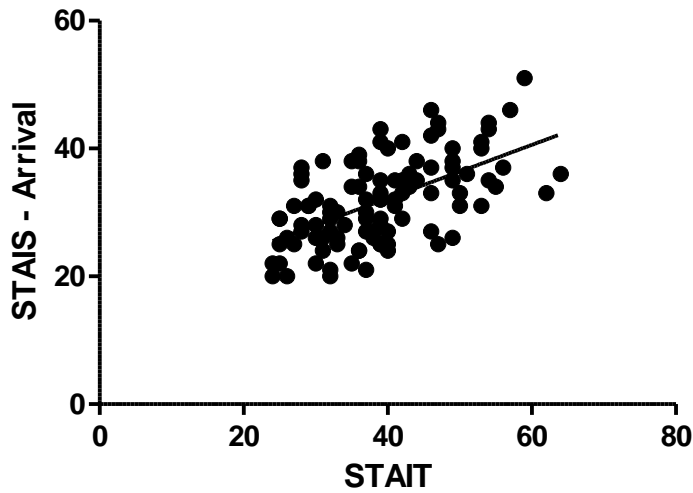


Figure 2. Relationship between trait anxiety and state anxiety upon arrival in the lab. Points represent the correlation between STAIS and STAIT scores for each participant. *Note.* STAIS = State-Trait Anxiety Inventory – State version . STAIT = State-Trait Anxiety Inventory – Trait version.

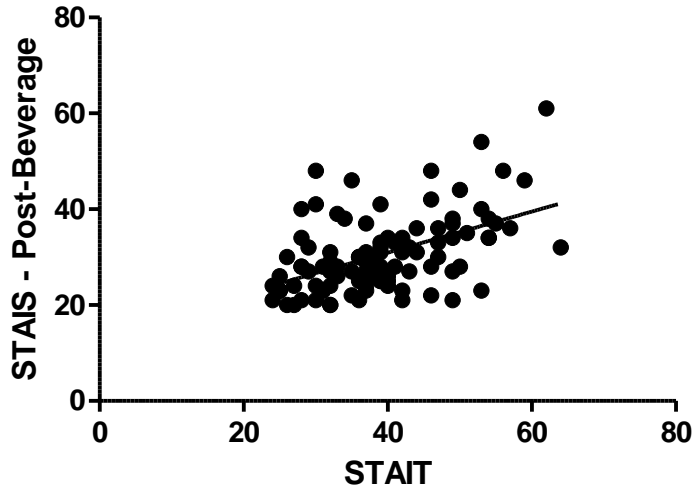


Figure 3. Relationship between trait anxiety and state anxiety following beverage consumption. Points represent the correlation between STAIS and STAIT scores for each participant. *Note.* STAIS = State-Trait Anxiety Inventory – State version. STAIT = State-Trait Anxiety Inventory – Trait version.

Behavioural Measures

In the CPT, there was a main effect of alcohol on correct detections, $F(4, 102) = 4.90, p = .001$, with the low dose group exhibiting significantly better performance than the medium ($p = .02$) and high ($p = 0.01$) dose groups. Similarly, the sober group had significantly better performance than the high dose group ($p = .02$) and there was a trend to significance with the medium dose group ($p = .055$). These results are summarized in Figure 4.

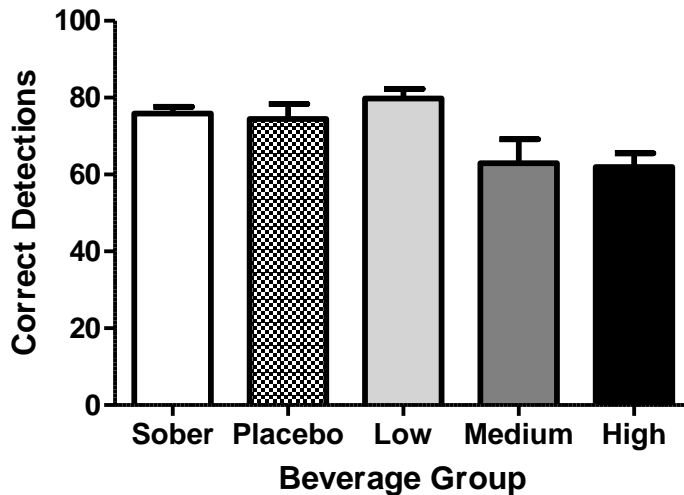


Figure 4. Effect of alcohol on correct detection on the Continuous Performance Test. Bars represent mean (+SEM) percent correct detections for sober, placebo and alcohol (low, medium and high dose) groups following beverage consumption.

There was also a main effect of alcohol on parametric discriminability (d'), $F(4, 102) = 2.85, p < .05$, in that the high dose group had significantly poorer discriminability than the low dose group (see Figure 5).

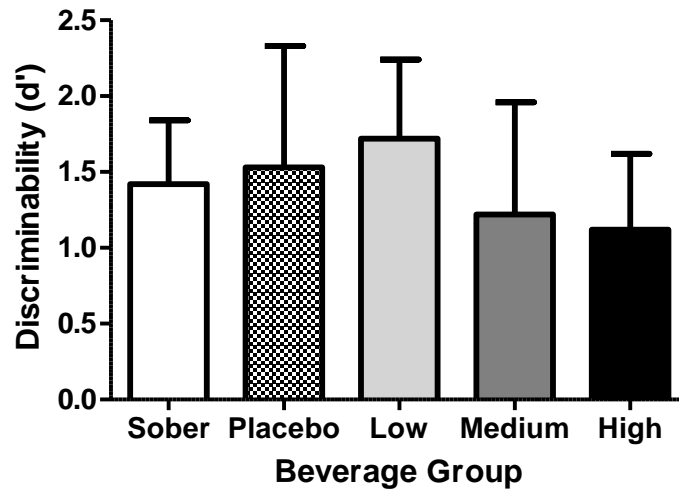


Figure 5. Effect of alcohol on discriminability (d') on the Continuous Performance Test. Bars represent mean (+SEM) discriminability (ratio between commission errors and correct detections) for sober, placebo and alcohol (low, medium and high dose) groups following beverage consumption.

Figure 6 shows a main effect of alcohol on A' (non-parametric discriminability/signal detection), $F(4, 103) = 3.93, p < 0.05$, with the medium dose group exhibiting significantly poorer A' performance compared to the low dose ($p < 0.05$) and placebo ($p < 0.05$) groups.

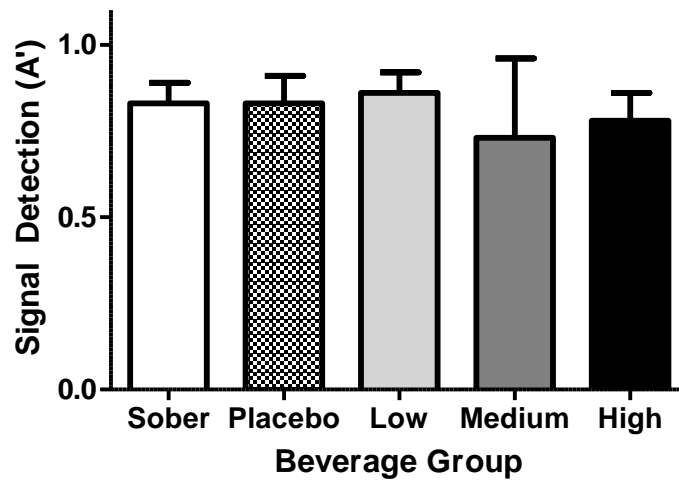


Figure 6. Effect of alcohol on nonparametric signal detection on the Continuous Performance Test. Bars represent mean (+SEM) A' values for sober, placebo and alcohol (low, medium and high dose) groups following beverage consumption.

As shown in Figure 7, there was a significant main effect of alcohol on Bd'' (nonparametric response bias), $F(4, 103) = 2.98, p < 0.05$, however none of the post-hoc analyses were statistically significant (all p -values > 0.05).

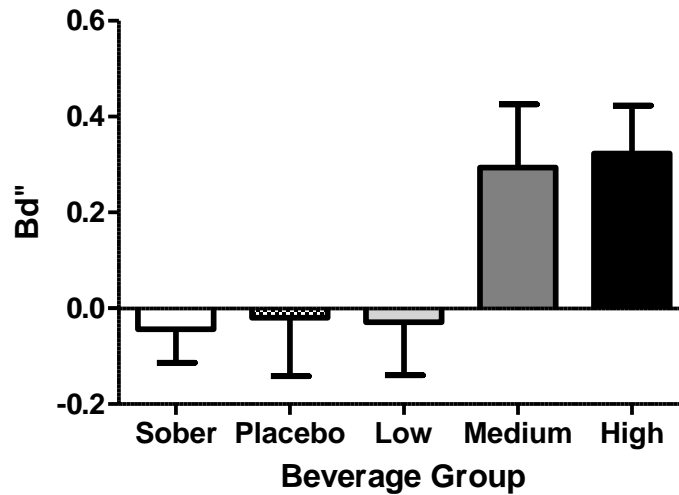


Figure 7. Effect of alcohol on parametric response bias on the Continuous Performance Test. Bars represent mean (+SEM) Bd'' scores for sober, placebo and alcohol (low, medium and high dose) groups following beverage consumption.

Alcohol had no significant effect on the remaining CPT measures: commission errors, $F(4, 102) = 0.54, p = 0.70$, and beta (response bias), $F(4, 103) = 1.17, p = 0.33$.

Figure 8 shows that alcohol significantly altered HVLIT performance, $F(4, 99) = 5.98, p < .001$. Specifically, the medium dose group exhibited significantly lower scores than the placebo ($p < 0.05$), sober ($p < 0.05$) and low dose ($p < 0.05$) groups.

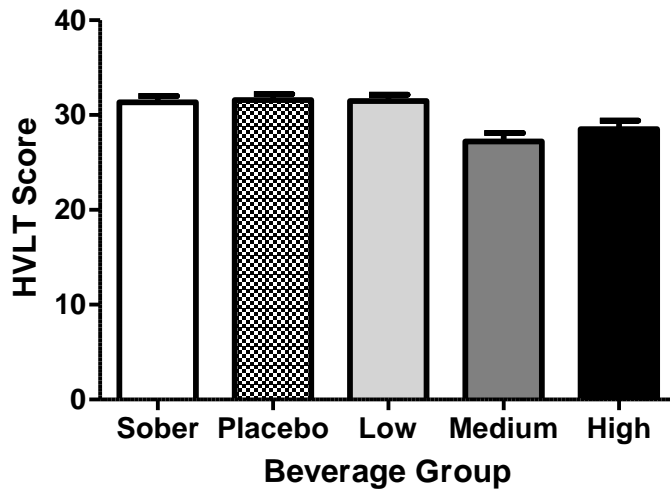


Figure 8. Effect of alcohol on HVL T performance. Bars represent mean (+SEM) total HVL T scores for sober, placebo and alcohol (low, medium and high dose) groups following beverage consumption. *Note.* HVL T = Hopkins Verbal Learning Task.

Stress Effects

Anxiety

Figure 9 shows that stress significantly affected STAIS scores, $F(1, 97) = 78.18, p < .001$, with post-stressor STAIS scores being significantly higher than pre-stressor scores. There was no effect of alcohol on STAIS scores, $F(4, 96) = 0.78, p = 0.54$, when the measures were taken following the stressor; however, the stress x group interaction was statistically significant, $F(4, 97) = 2.66, p < 0.05$. As seen in Figure 9, the five groups had comparable pre-stressor STAIS scores; post-stressor, however, the sober group reported higher STAIS scores than the placebo and alcohol groups.

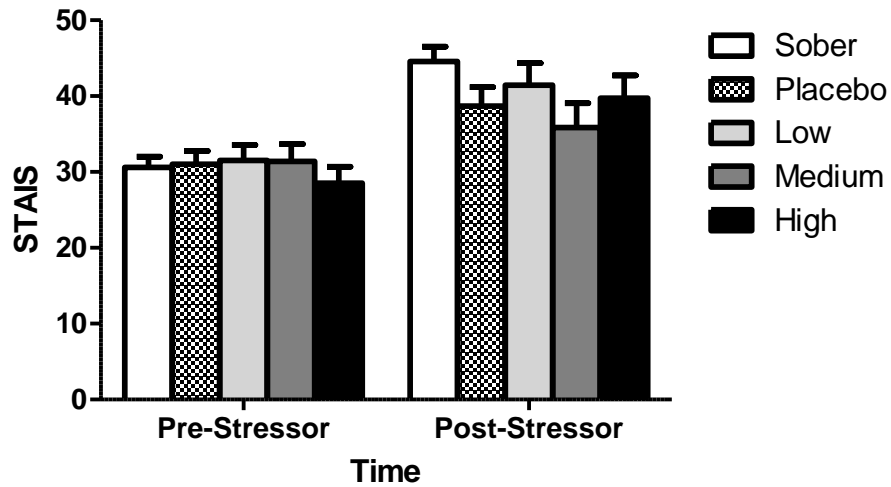


Figure 9. Effect of psychosocial stress on state anxiety for each beverage group. Bars represent mean (+SEM) total STAIS score for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* STAIS = State-Trait Anxiety Inventory – State version. TSST = Trier Social Stress Test.

In order to investigate the pharmacological versus the expectancy effects of alcohol on stress reduction, the three alcohol groups were combined (see Figure 10). As with the original analysis, the main effect of stress, $F(1, 99) = 92.48, p < .001$, and the stress x beverage group interaction were significant, $F(2, 99) = 3.61, p < .05$.

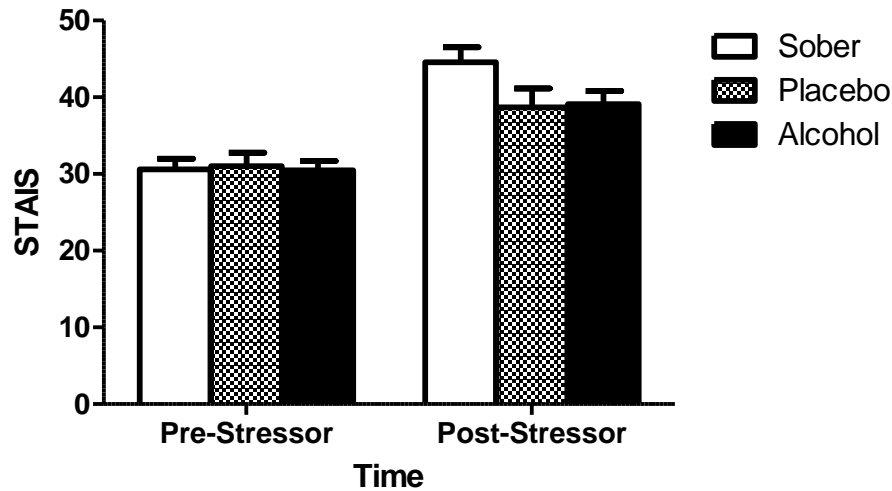


Figure 10. Effect of psychosocial stress on state anxiety for sober, placebo and alcohol groups. Bars represent mean (+SEM) total STAIS score for each group with the low, medium and high doses of alcohol combined into one group. STAIS scores are presented prior to, and following, the TSST. *Note.* STAIS = State-Trait Anxiety Inventory – State version. TSST = Trier Social Stress Test.

Trait anxiety (i.e., STAIT scores) was significantly correlated with state anxiety (STAIS) post-stressor, $r = 0.38, p < .001$. In addition, state anxiety pre-stressor was a significant predictor of post-stressor state anxiety, even controlling for trait anxiety (STAIT), $F(2, 95) = 16.52, p < .001$. Trait anxiety accounted for 14% of the variance in post-stressor anxiety and pre-stressor state anxiety accounted for 11% of the variance above the effect of trait anxiety (see Figure 11).

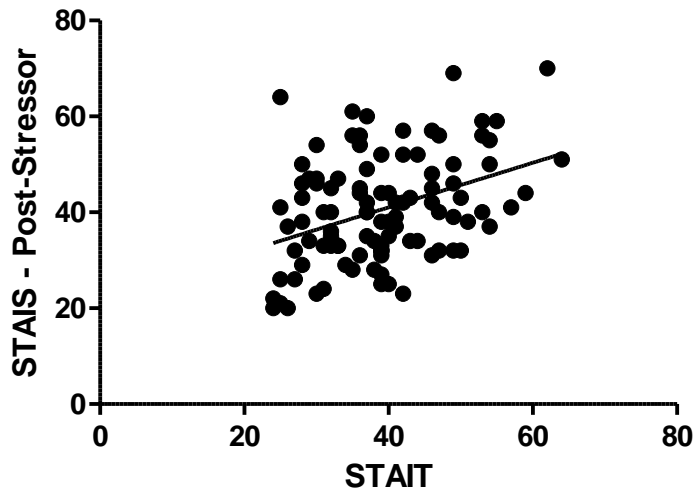


Figure 11. Relationship between trait anxiety and state anxiety following the psychosocial stressor. Points represent the correlation between STAIS and STAIT scores for each participant. *Note.* STAIS = State-Trait Anxiety Inventory – State version . STAIT = State-Trait Anxiety Inventory – Trait version.

Behavioural Measures

There was no main effect of stress on CPT correct detections, $F(1, 98) = 1.79, p = .18$, but the main effect of alcohol was statistically significant, $F(4, 98) = 5.00, p = .001$. Following the psychosocial stressor, the high dose group was significantly lower than the low dose ($p < .05$), placebo ($p < .05$) and sober ($p < .05$) groups, and the medium dose group was significantly lower than the low dose group ($p < .05$). The stress x beverage group interaction was not statistically significant, $F(4, 98) = 0.44, p = .78$.

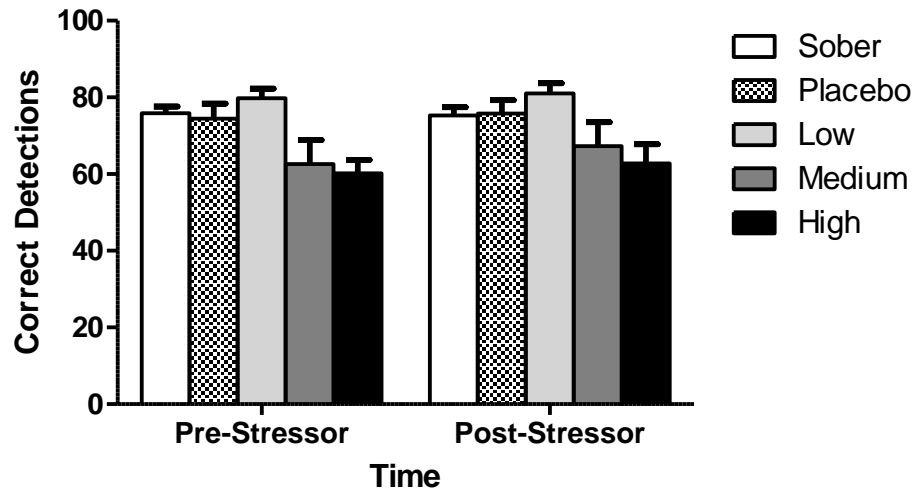


Figure 12. Effect of psychosocial stress on correction detection on the Continuous Performance Test. Bars represent mean (+SEM) percent correct detections for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

As shown in Figure 13, there was a significant main effect of stress on commission errors, $F(1, 98) = 39.17, p < .001$, in that errors were lower post-stressor compared to pre-stressor. There was no main effect of alcohol on commission errors, $F(4, 98) = 0.92, p = .46$, and the stress x beverage group interaction was also non-significant, $F(4, 98) = 1.93, p = .11$.

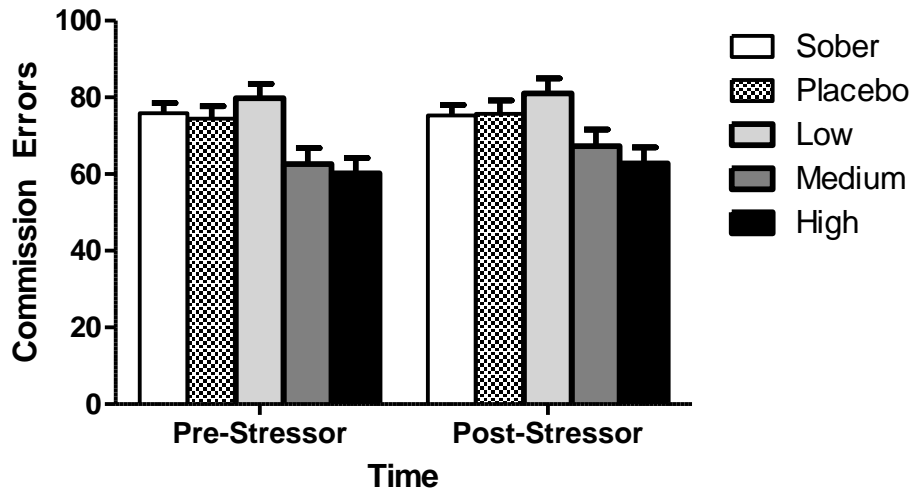


Figure 13. Effect of psychosocial stress on commission errors on the Continuous Performance Test. Bars represent mean (+SEM) percent commission errors for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

There was a main effect of stress on d' (parametric discriminability), $F(1, 98) = 46.95, p < .001$, in that this measure was higher post-stressor compared to pre-stressor (see Figure 14). There was no effect of alcohol on d' (discriminability), $F(4, 98) = 1.85, p = .13$, and the stress \times beverage group interaction was not significant, $F(4, 98) = 1.21, p = .31$.

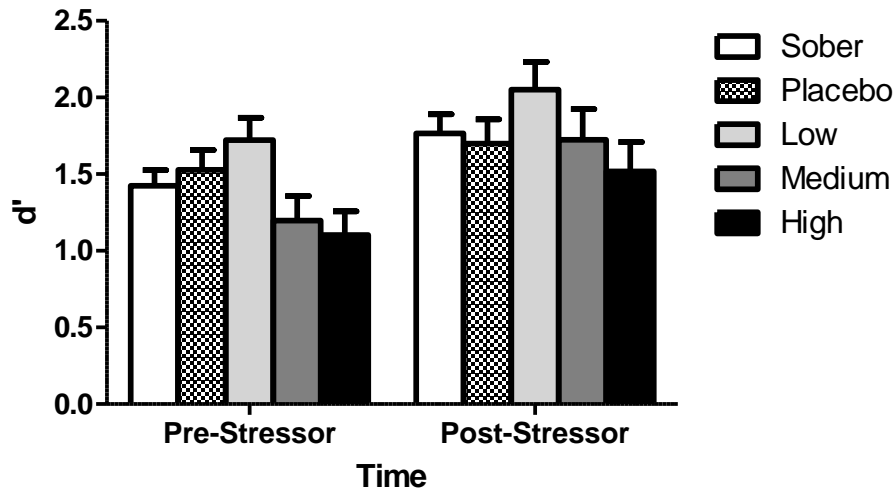


Figure 14. Effect of psychosocial stress on d' (discriminability) on the Continuous Performance Test. Bars represent mean (+SEM) percent commission errors for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

There was also a significant effect of stress, $F(1, 99) = 34.05, p < .001$, and alcohol, $F(4, 99) = 3.44, p < .05$, on A' scores (non-parametric discriminability/ signal detection) on the CPT (see Figure 14). A' scores increased from pre- to post-stress measures and the medium dose group had significantly lower scores than both the low dose and sober groups (all p -values $< .05$). The stress x beverage group interaction was non-significant, $F(4, 99) = 1.36, p = .25$.

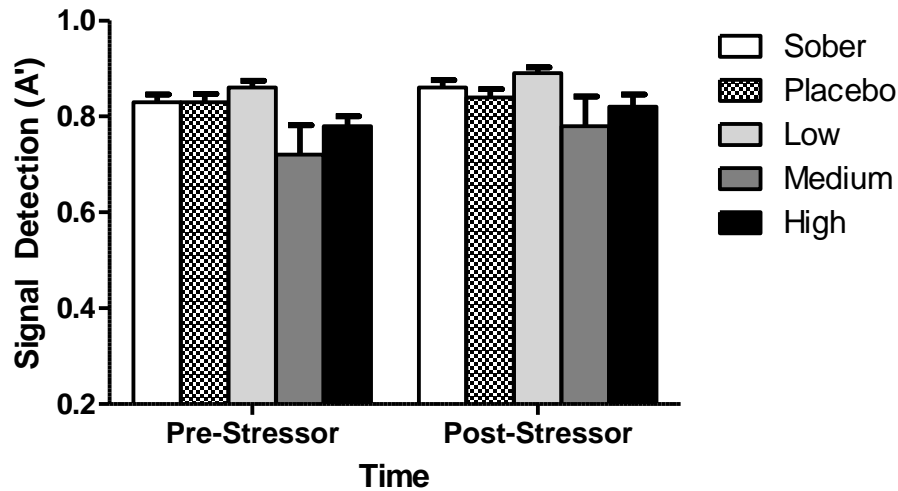


Figure 15. Effect of stress on nonparametric signal detection on the Continuous Performance Test. Bars represent mean (+SEM) A' scores (signal detection) for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

Similarly, there was a main effect of stress on beta (parametric response bias) on the CPT, in that scores were significantly higher post-stressor compared to pre-stressor, $F(1, 99) = 19.54, p < .001$ (see Figure 16). The main effect of alcohol, $F(4, 99) = 0.73, p = .57$, and the stress x beverage group interaction, $F(4, 99) = 0.53, p = .71$, were both non-significant.

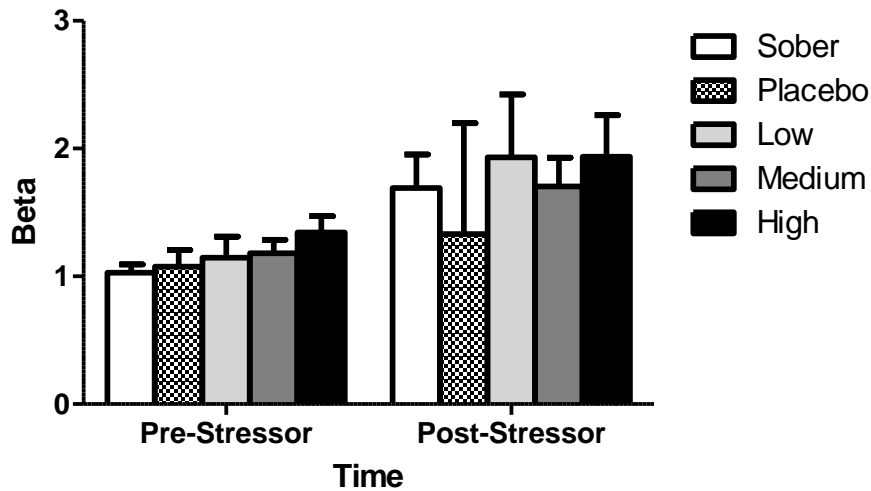


Figure 16. Effect of stress on parametric response bias on the Continuous Performance Test. Bars represent mean (+SEM) beta scores (response bias) for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

As shown in Figure 17, psychosocial stress had a significant effect on B'd (non-parametric response bias), $F(1, 99) = 8.98, p < .05$, in that scores increased significantly post-stressor compared to pre-stressor. The effect of alcohol was also significant, $F(4, 99) = 3.27, p < .05$, in that the placebo group had significantly higher scores compared to the high dose alcohol group, $p = .05$. The stress x beverage condition, however, was non-significant, $F(4, 99) = 0.72, p = .58$.

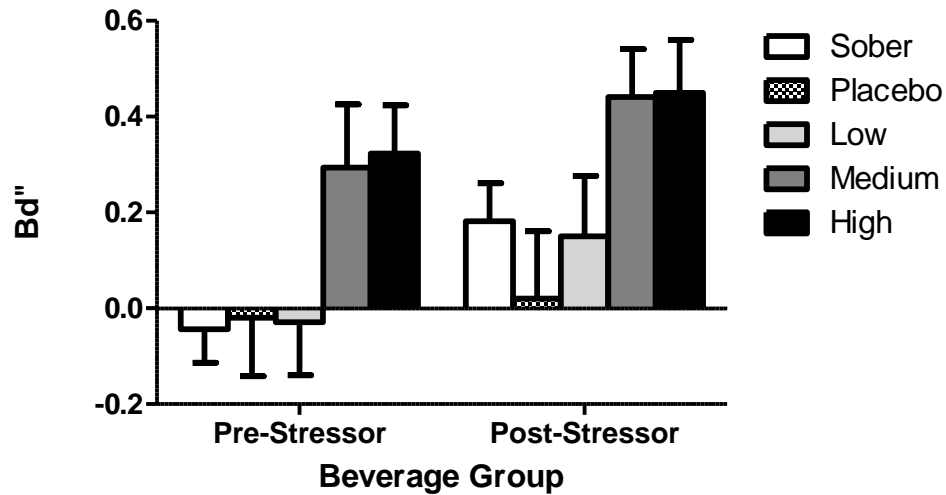


Figure 17. Effect of stress on parametric response bias on the Continuous Performance Test. Bars represent mean (+SEM) Bd'' scores for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* TSST = Trier Social Stress Test.

Figure 18 shows that there was a main effect of stress on HVLIT performance, $F(1, 95) = 3.99, p = .05$, with scores being significantly higher post-stressor than pre-stressor. Similarly, there was a main effect of alcohol, $F(4, 95) = 6.52, p < .001$, in that the medium dose group had significantly lower scores than the sober ($p < .001$), placebo ($p = .001$) and low dose ($p = .001$) groups. The stress x beverage group interaction was not significant, $F(4, 95) = 0.42, p = .79$.

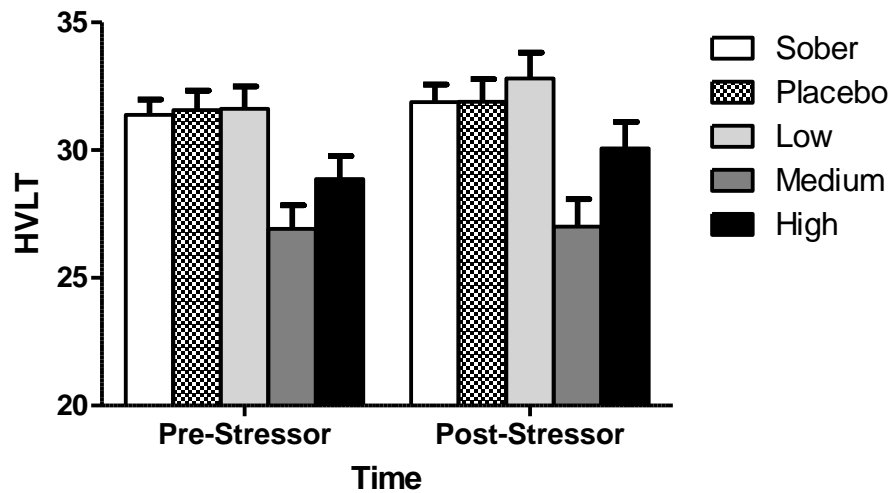


Figure 18. Effect of stress on HVL T performance. Bars represent mean (+SEM) total HVL T scores for sober, placebo and alcohol (low, medium and high dose) groups prior to, and following, the TSST. *Note.* HVL T = Hopkins Verbal Learning Task. TSST = Trier Social Stress Test.

Cognitive Moderator of Stress Response

The hypothesized stress-dampening effect of alcohol was supported with STAIS scores being lower in the placebo and alcohol groups than in the sober group post-stressors. Thus, the moderating effect of alcohol expectancies on this relationship were explored, revealing a main effect of time, $F(1, 84) = 72.73, p < .001$; but not beverage group, $F(4, 84) = 0.80, p = .52$, or Stress Reduction group, $F(2, 84) = 1.10, p = .39$. None of the interaction terms – stress x beverage group, stress x Stress Reduction scale or stress x beverage group x Stress Reduction scale – was statistically significant (all p -values > .05).

Although the moderating effect of the Stress Reduction scale was not supported, further exploratory analyses were conducted to examine the relationship between the Stress Reduction scale of the PDI in the CDIS and the stress response. The Stress Reduction scale was not significantly correlated with trait anxiety (STAIT), $r = 0.006$, $p = .95$. Similarly, this scale was not significantly correlated with state anxiety (STAIS) upon arrival in the lab, $r = 0.11$, $p = .26$, or post-stressor, $r = 0.10$, $p = .30$. Interestingly, Stress Reduction showed a significant positive correlation with state anxiety (STAIS) following beverage consumption, $r = 0.26$, $p < .05$. Moreover, no other CDIS subscale was significantly related to post-beverage consumption STAIS (all p -values $> .05$).

Chapter 4

Discussion

The aim of this study was to investigate whether a cognitive factor, namely tension reduction alcohol expectancies, moderates the stress-dampening effect of alcohol. Several hypotheses were made regarding the outcomes of this study. Specifically, it was predicted that the stressor would be effective in eliciting a stress response, operationalized as increased subjective anxiety and impairment on a sustained attention task. Secondly, it was hypothesized that this stress response would be dampened by alcohol, in a dose-dependent manner. Most importantly, the stress-dampening effect of alcohol was expected to depend inversely on participants' ratings of tension reduction alcohol expectancies.

The first critical hypothesis, that the stressor would reliably elicit a stress response, was supported in that STAIS scores increased significantly in all groups following the stressor. This elevation in subjective anxiety following the TSST is consistent with other findings (Lackschewitz, Hüther, & Kröner-Herwig, 2008; Schlotz et al., 2008; Uhart, Chong, Oswald, Lin, & Wand, 2006; Uhart, Oswald, McCaul, Chong, & Wand, 2006). In contrast, the TSST did not disrupt cognitive processes, a finding that contradicts previous studies in which sustained attention on the CPT is impaired following a stressor (Bohnen et al., 1990; Bohnen & Jolles, 1992). As predicted (Hoffman & al'Absi, 2004), there was no stress-induced impairment on the HVLT, which served as a cognitive control. Interestingly, performance on both the CPT (all measures except correct detections) and

the HVLТ improved following the stressor. This could reflect practice effects as verbal list-learning (Theisen, 1997) and some CPT measures (correct detections and discriminability) improve over trials (Chen, Hsiao, Hsiao, & Hwu, 1998). On the other hand, some studies report CPT practice effects on commission errors only (Barkley, Murphy, O'Connell, & Connor, 2005), the one measure in our study that did not improve post-stressor. Moreover, the impact of practice effects was probably minimized in our study as different versions of the HVLТ and CPT tests were given (i.e. different lists and random number sequences, respectively) before and after the stressor. Given that the research in this area remains equivocal, it is not clear that practice effects can explain the improved cognitive performance following a stressor in our task.

Another possibility is that the TSST improved cognitive performance by increasing arousal. Arousal can be viewed as a general physical and emotional activation that sits on a continuum from sleep to excitement (Gould & Krane, 1992). It has both a somatic (physiological) and cognitive component (Gould & Udry, 1994) that are altered with increasing arousal. Cognitive arousal can range from stupor to alertness (Blum, Geiwitz, & Stewart, 1967; Blum, Graef, & Hauenstein, 1968); increased cognitive arousal is required to meet the demands of cognitive tasks (Lambourne & Tomporowski, 2010). In that respect, arousal can have a beneficial effect on cognitive functions, such as memory, particularly when the arousal has an emotional component (McGaugh, 2006). Similarly, decreased arousal is associated with poorer performance on sustained attention, or vigilance, tasks (Helton & Warm, 2008). This relationship between arousal and cognitive

performance is inverted at higher levels (the classic inverted-U) in that stress-induced arousal improves performance up to a critical threshold (the point differs depending on the task), after which performance is hindered (Yerkes & Dodson, 1908). We predicted that the TSST would impair cognitive performance based on the potent nature of this stressor and its ability to evoke significant subjective and physiological stress responses (Kirschbaum et al., 1993). Although the manipulation dependably elevates arousal (Childs, Van Dam, & Wit, 2010), this may have reached a sub-threshold level in our sample producing beneficial, as opposed to deleterious, effects on cognitive performance.

Our second main hypothesis, that alcohol would reduce stress responses, was supported. Prior to the stressor, the groups reported similar STAIS scores; post-stressor, however, the sober group reported higher STAIS scores than both the placebo and alcohol (low, medium, and high dose) groups. In other words, the subjective anxiety of individuals who believed they had consumed alcohol increased to a lesser extent than in sober individuals. This highlights two distinct points; the first is that alcohol effectively dampens a stress response. Given that two of the most predictive conditions for observing the stress-dampening effect of alcohol are a potent stressor and a high dose of alcohol (Levenson, Sher, Grossman, Newman, & Newlin, 1980), our findings fit with previous research. The second point is that the expectancy of receiving alcohol was as effective as the drug itself in dampening a stress response. The follow-up comparison, in which the alcohol groups were collapsed, confirmed that the placebo and alcohol groups had similar subjective anxiety levels post-stressor: both increased post-stressor but not to the extent

of the sober group. This lends further support to the notion that the mere expectancy of alcohol – not just the pharmacological action of the drug – has a significant impact on subjective anxiety responses to stress.

As predicted based on a meta-analytic review of vigilance tasks (Koelega, 1995), high-dose alcohol did not impair performance on the CPT, as measured by beta (response bias) and commission errors. That is, alcohol did not affect conservative versus liberal responding, as measured specifically by beta. Also, alcohol did not affect commission errors, which are an indicator of impulsivity (Dougherty, Bjork, Marsh, & Moeller, 2000a). By contrast, alcohol had significant effects on the remaining CPT measures. In particular, alcohol, in the absence of stress, impaired performance on CPT correct detections, discriminability (d' , A'), and lowered response bias (Bd'') scores (i.e., produced more liberal responding). Furthermore, the effect of alcohol persisted, following the introduction of a stressor, on CPT correct detections and A' (discriminability).

In contrast to our results, the majority of studies report that vigilance tasks are insensitive to the effects of alcohol, even at high doses (Koelega, 1995b). Our findings, however, replicate a study by the developers of the particular CPT task used in this study (Dougherty, Marsh, Moeller, Chokshi, & Rosen, 2000). Specifically, Dougherty et al (2000a, 2000b) reported the same alcohol-induced impairment of correct detections (2000a) and discriminability (A' ; 2000b) that we observed. These authors attribute the failure of most studies to show an alcohol-induced impairment on CPT to insensitive

measures and poorly operationalized outcome variables (Dougherty, Marsh et al., 2000). There was one contradictory finding between our study and that of Dougherty et al. (2000b): they reported that alcohol lowered Bd' scores, or produced more conservative responding, whereas we found that alcohol increased Bd' scores, indicating more liberal responding. It is not clear what explains these differences, although our results are consistent with the authors original hypotheses of alcohol-induced increases in Bd' scores (Dougherty, Marsh et al., 2000).

In addition to showing that alcohol impairs performance on specific CPT measures, we also observed differences in alcohol effects across doses. On pre-stressor correct detections, the medium and high dose groups had significantly poorer performance than the low dose group; on post-stressor correct detections, the score of the high dose group was significantly lower than that of the low, placebo and sober groups. Regarding pre-stressor discriminability, the high dose alcohol group performed significantly worse than the low dose group on d' , and the medium dose group performed significantly worse than the low dose alcohol and placebo groups on A' . This U-shaped relationship between alcohol and cognitive effects could reflect compensatory responding to intoxication in the high dose group. In other words, it is likely that the low dose group was not impaired on these cognitive measures because their level of intoxication was so low (BAL = 0.02). The high dose group was clearly intoxicated (BAL = 0.09) and may have attempted to compensate for this in the cognitive tasks. Compared to the high dose, the medium dose group probably experienced reduced perceptual effects of alcohol (BAL = 0.06), and may

not have felt the need to compensate in the cognitive tasks. The idea that high dose participants may have attempted to overcome the detrimental effects of intoxication fits with evidence that many individuals can improve performance on cognitive tasks through a concerted effort. Several studies have shown that individuals who believe they have consumed alcohol are hypervigilant and use compensatory strategies in order to make up for the impaired cognitive abilities they anticipate (Testa et al., 2006). For example, sustained attention is improved when biofeedback on automatic arousal is provided (O'Connell et al., 2008), which directly demonstrates that cognitive performance can be improved with effort. Furthermore, informal observations of participants in this study indicated that they were motivated to perform optimally in the cognitive tasks and highly intoxicated individuals were frequently more reserved and subdued than were less intoxicated individuals.

Our third, and most important, hypothesis conjectured that ratings on the Stress Reduction scale of the CDIS would moderate the stress-response dampening effect of alcohol; there was no evidence to suggest that this prediction is supported in our data. This may not be surprising as there is little consensus in the literature on the factors that explain individual differences in stress and alcohol responses (Sher, Bartholow, Peuser, Erickson, & Wood, 2007). Moreover, the scale used to assess stress reduction alcohol expectancies is relatively new (Fisher et al., 2007), so the psychometric properties may not be fully established. Furthermore, the stress dampening effect of alcohol may depend on the time course of intoxication (Sher et al., 2007), a factor which was not included in

our analysis. This possibility will be examined in future studies by analyzing the time course of biochemical changes (cortisol and alpha-amylase) through analysis of saliva samples. Nonetheless, since our final analysis included less participants than the recommended sample size of 105, it is possible that our null findings are due to that fact that our study lacked sufficient statistical power to detect an effect.

In sum, the primary findings of our study were that the TSST reliably elicits an increase in subjective anxiety, and alcohol is effective in dampening this response. By contrast, our cognitive variable of interest – tension reduction alcohol expectancies – did not moderate the stress-response dampening effect of alcohol. There are many implications to the current research. Firstly, these data replicate studies supporting the efficacy of the TSST in reliably eliciting a stress response. Furthermore, the finding that alcohol dampens this response in subjective anxiety lends credence to the view that alcohol is reinforcing in its ability to help individuals cope with stress. Although our findings regarding stress reduction as a cognitive moderator were not supported, they contribute to a better understanding of which factors reliably predict the stress-response dampening effects of alcohol; in this case, we have shown that this particular scale does not account for significant variance in the relationship between stress and alcohol intoxication. More research is needed to elucidate which factors are, in fact, effective in predicting this relationship. Specifically, follow up studies should focus on expectancies because manipulation of expectancies could modify drinking behaviour. Importantly, reinforcing expectancies can be incorporated into relapse-prevention, and the

identification of particularly “high-risk” expectancies early in childhood or adolescence may guide early interventions that help circumvent maladaptive drinking patterns and abuse (Brown, Goldman, & Christiansen, 1985). The latter point is particularly important given the chronic and remitting nature of the illness, once addiction has been established.

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Appendix A

State-Trait Anxiety Inventory (sample)

SELF-EVALUATION QUESTIONNAIRE STAI Form Y-1

Please provide the following information:

Name _____ Date _____ S _____

Age _____ Gender (Circle) M F T _____

DIRECTIONS:

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right now*, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

VERY MUCH SO
 MODERATELY SO
 SOMEWHAT
 NOT AT ALL

- | | | | | |
|--------------------------|---|---|---|---|
| 1. I feel calm | 1 | 2 | 3 | 4 |
| 2. I feel secure | 1 | 2 | 3 | 4 |
| 3. I am tense | 1 | 2 | 3 | 4 |
| 4. I feel strained | 1 | 2 | 3 | 4 |
| 5. I feel at ease | 1 | 2 | 3 | 4 |

Appendix B

Perceived Intoxication Questionnaire

Drug Effects Questionnaire

Please rate your feelings on the following scales by circling the number that approximates your current state.

1. I feel...
1 2 3 4 5 6 7 8 9
Sober - Drunk

2. I like/dislike the effects I feel:
1 2 3 4 5 6 7 8 9
Dislike - Like

3. I do/do not want more to drink:
1 2 3 4 5 6 7 8 9
Do not want more - Want more

Manipulation Check

Now, we are interested in knowing how accurately you can estimate your present level of intoxication. Please complete the questions below.

1. How intoxicated do you feel right now?
1 - not at all
2 - mildly
3 - moderately
4 - very
5 - extremely

2. Estimate, in bottles of beer, how much alcohol you have consumed.
1 - no beer
2 - less than 1 bottle
3 - 1 bottle
4 - 2 bottles
5 - 3 bottles
6 - 4 bottles
7 - 5 bottles
8 - 6 bottles
9 - more than

3. Recall that pure alcohol is 40% alcohol. Think of the mixed drinks that you received, what do you think the alcohol content was?
0% 5% 10% 15% 20% 25% 30% 35% 40% (pure alcohol)

4. The legal blood alcohol limit in Ontario is 0.08%.
If you were to estimate your blood alcohol level *right now*, what do you think it would be?
_____ %

Appendix C

College Drinking Influences Survey Data by Group

Appendix C. Mean (SD) scores on the College Drinking Influences Survey (CDIS) subscales for each beverage group. *F* and *p* values from the one-way ANOVA are presented in the two final columns. *Note.* PDI = Psychosocial Drinking Inventory. DVS = Drinking Values Scale. CDES = College Drinking Expectations Scale.

Subscale	Sober	Placebo	Low	Medium	High	<i>F</i>	<i>p</i>
PDI							
Social Influences	2.82 (0.53)	2.85 (0.54)	2.88 (0.63)	2.75 (0.47)	2.99 (0.42)	0.45	0.77
Stress Reduction	1.88 (0.51)	1.87 (0.52)	1.82 (0.43)	1.81 (0.33)	1.92 (0.42)	0.15	0.96
Sensation Seeking	2.95 (0.52)	2.98 (0.61)	2.88 (0.61)	2.98 (0.52)	3.14 (0.47)	0.55	0.70
DVS							
Social Responsibility	3.13 (0.91)	3.20 (0.94)	3.23 (0.95)	3.29 (0.53)	2.62 (0.77)	1.57	0.19
Personal Choice	4.08 (0.70)	4.18 (0.51)	3.97 (0.79)	3.90 (0.61)	4.25 (0.61)	0.82	0.52
Institutional Obligation	3.36 (0.82)	3.42 (0.63)	3.62 (0.63)	3.67 (0.65)	3.35 (1.05)	0.67	0.61
CDES							
Drinking Norms	3.22 (0.51)	3.38 (0.60)	3.43 (0.58)	3.21 (0.41)	3.43 (0.44)	0.94	0.44
Drinking Consequences	4.08 (0.53)	4.04 (0.73)	4.18 (0.71)	4.21 (0.56)	4.24 (0.53)	0.41	0.80