# THE EFFECTS OF ALCOHOL AND ALCOHOL EXPECTANCY ON THE RESPONSE TO HYPERVENTILATION AMONG HIGH AND LOW ANXIETY SENSITIVE YOUNG ADULTS

by

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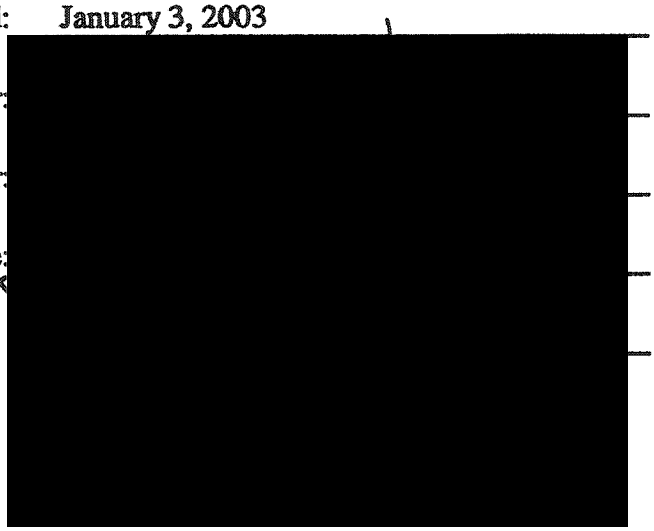

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Dedication

For my parents, John and Carolyn MacDonald, whose belief in me and unwavering support continue to light the path of my life.

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Abstract

Study One evaluated the hypothesis that high anxiety sensitivity (AS) levels may negatively reinforce alcohol use/abuse by promoting a heightened sober reactivity to theoretically-relevant stressors and heightened sensitivity to alcohol's emotional reactivity dampening effects. One-hundred-and-two undergraduate participants (51 high AS, 51 low AS) were assigned to either a placebo, low dose alcohol, or high dose alcohol beverage condition. Following beverage consumption and absorption, participants underwent a three-minute voluntary hyperventilation challenge. High AS - placebo participants displayed increased affective and cognitive reactivity to the challenge compared to low AS - placebo participants. Dose-dependent alcohol dampening of affective and cognitive reactivity to hyperventilation was observed only among high AS participants, suggesting that they may be particularly sensitive to alcohol-induced reductions in their degree of fear and negative thinking in response to the experience of physical arousal sensations. Study Two tested the hypothesis that alcohol outcome expectancies might contribute to alcohol's reactivity-dampening effects in high AS individuals, over-and-above the pharmacological effects of alcohol. Forty-eight high AS undergraduates were randomly assigned to one of three beverage conditions: alcohol, placebo, and control. The same procedure as Study One was followed. As in Study One, participants in the alcohol condition showed dampened affective and somatic responses to the challenge, and marginally dampened cognitive responses to the challenge, compared to both placebo and control participants. However, placebo participants did not display dampened responses to the challenge relative to control beverage condition participants, suggesting that alcohol expectancies may not mediate alcohol's stress response dampening effects in high AS individuals. Additional analyses suggested that tension-reduction expectancies might have contributed to an "inverse placebo" effect among high AS participants administered placebo. Implications of these results for the AS risk model of alcohol abuse are discussed, along with ideas for future research and prevention strategies for alcohol problems among high AS individuals.

List of Abbreviations

| | |
|---|---|
| ANCOVA | analysis of covariance |
| ANOVA | analysis of variance |
| AS | anxiety sensitivity |
| DSM-IV | Diagnostic and Statistical Manual of Mental Disorders – 4th edition |
| HVQ | Hyperventilation Questionnaire |
| GABA | Gamma-aminobutyric acid |
| MAST | Michigan Alcoholism Screening Test |
| STAI-T | State Trait Anxiety Inventory – Trait Subscale |

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# CHAPTER ONE

## Defining the Construct of Anxiety Sensitivity

Anxiety sensitivity is an individual difference variable that refers to a fear of anxiety-related bodily symptoms. The construct of anxiety sensitivity arose from a broader theoretical framework known as expectancy theory (Reiss, 1980). Reiss applied a re-conceptualization of Pavlovian conditioning by animal researchers to account for the acquisition of common fears in humans, noting that a contiguous relationship between a conditioned stimulus and an unconditioned stimulus was not necessary for the acquisition of a fear response. Rather, Reiss (1991) argued that humans could learn to *expect* an unconditioned stimulus (e.g., "I believe I might have an anxiety attack on the flight") without direct conditioning. Reiss and McNally (1985) coined the term "anxiety sensitivity" to capture an individual, fundamental fear of the consequences of anxiety symptoms themselves (e.g., "An anxiety attack would be unbearable"). These fears are based on a set of specific expectations that anxious somatic sensations will lead to negative consequences such as illness, embarrassment, and loss of control (Reiss, Peterson, Gursky, & McNally, 1986; Reiss, 1991; Reiss & McNally, 1985; Stewart, Taylor, & Baker, 1997). Thus, anxiety sensitivity can be viewed as an "anxiety amplifier", increasing the probability that an individual will experience future anxiety, fear and/or panic.

To illustrate, picture two individuals entering a classroom to write a university exam. Both are experiencing several symptoms consistent with anxiety, such as an increased heart rate, stomach upset, and dizziness. For one of these individuals, these

symptoms are unpleasant but harmless, an annoyance to be worked through during the exam. For the other individual, these symptoms are linked to fears which may include having a heart attack, embarrassing him or herself in front of the entire classroom, or even losing complete control of him or herself. The second individual would be characterized by high levels of anxiety sensitivity. While anxiety sensitivity is an interesting concept in itself, it is also an individual difference variable which is of great relevance for public health and welfare. Evidence is building that individuals characterized by high levels of anxiety sensitivity may one day be at increased risk of alcoholism.

The construct of anxiety sensitivity is measured on a scale known as the Anxiety Sensitivity Index (Reiss et al., 1986; see Appendix A), a well-validated self-report inventory about beliefs concerning the negative consequences associated with the experience of anxiety symptoms. Just as everyone experiences anxiety, so too does everyone harbour some level of fear of those anxiety sensations. Thus, anxiety sensitivity can be viewed as lying on a continuum, with individuals exhibiting different levels of fear of anxiety-related bodily arousal symptoms.

Anxiety Sensitivity vs. Trait Anxiety

In contrast to anxiety sensitivity, the more traditional construct of trait anxiety refers to an individual who responds fearfully to stressors across many potentially stressful situations (McNally, 1996), but who does not specifically fear bodily arousal symptoms (Stewart, Samoluk, & MacDonald, 1999). Moreover, the trait anxiety construct uses frequency of past anxiety experiences as an indicator to predict frequency of future anxiety reactions, while anxiety sensitivity relies on a belief system about the

hazardous nature of bodily arousal cues to predict the frequency of future anxiety and fear (Reiss, 1997). Although the constructs of anxiety sensitivity and trait anxiety are related, they are not redundant. Estimates of the shared variance ($r^2$) between measures of the constructs have ranged from 0 - 36% (McNally, 1996). In fact, a substantial body of research has supported the idea that the constructs of trait anxiety and anxiety sensitivity are conceptually and empirically distinct from one another (see reviews in McNally, 1996; Reiss; 1997; Taylor, 1996).

Anxiety Sensitivity and Anxiety Disorders

High levels of anxiety sensitivity are associated with many of the anxiety disorders, including social phobia, post traumatic stress disorder (PTSD), and panic disorder (Cox, Borger & Enns, 1999). Panic disorder patients are characterized by particularly high anxiety sensitivity levels (Stewart, Knize & Pihl, 1992; Taylor, Koch & McNally, 1992). In fact, highly elevated anxiety sensitivity levels distinguish panic disorder patients from generalized anxiety disorder patients (Taylor et al., 1992). In contrast, these two anxiety disorders are associated with similarly elevated trait anxiety (Taylor et al., 1992). Anxiety sensitivity has been referred to as an "anxiety amplifying" factor. That is, high anxiety sensitive individuals who "catastrophically" misinterpret the meaning of anxiety-related somatic sensations consequently experience increases in state anxiety levels which may even spiral into panic attacks (Reiss, 1991). For example, the student who experiences an increased heart rate prior to writing an exam may interpret this sensation as a sign of serious or impending heart problems, raising his or her anxiety level and associated somatic symptoms and providing positive feedback for the catastrophic cognition. As this feedback loop strengthens, fearful cognitions leading to

increased subjective anxiety and somatic symptoms, leading to more fearful cognitions, the chances of experiencing a panic attack increase. Longitudinal research has provided strong support for the idea that high anxiety sensitivity levels may lead to the later development of panic attacks (Schmidt, Lerew & Jackson, 1997; Schmidt, Lerew, & Jackson, 1999). In addition, important empirical evidence has recently been gathered to suggest that anxiety sensitivity may mediate a causal relationship between childhood learning experiences (involving parental modeling and reward of a child's anxiety sensations) and panic attacks in young adults (Stewart, Taylor, Jang, Cox, Watt, Fedoroff & Borger, 2001).

Although anxiety sensitivity appears to be a risk factor for developing panic attacks, it is distinguishable from anxiety disorders such as panic disorder in that it does not require the experience of clinically significant anxiety or panic in its own development (Reiss, 1991). For example, even among non-clinical samples with no history of panic attacks, anxiety sensitivity predicts fearful responding to a number of arousal induction procedures including carbon dioxide ($CO_2$) inhalation and voluntary hyperventilation challenges (e.g., Asmundson, Norton, Wilson & Sandler, 1994; Donnell & McNally, 1989; Rapee & Medoro, 1994). According to Peterson and Plehn (1999), non-clinical individuals with high levels of anxiety sensitivity may share a belief system about the dangerousness of anxiety sensations that is consistent with panic disorder although they do not experience the disorder.

## Anxiety Sensitivity and Alcohol Use Disorders

### Co-morbidity of Alcohol Abuse and Anxiety Disorders

We have suggested that high anxiety sensitive individuals may be at increased

risk for the development of alcohol use disorders (Stewart et al., 1999; also see McNally, 1996). In part, this idea has drawn support from the high co-morbidity observed between alcohol abuse and anxiety disorders, particularly when the anxiety disorder in question is characterized by high anxiety sensitivity levels (Stewart et al., 1999). For example, researchers have highlighted an increased incidence of alcohol abuse in panic disorder patients (Schuckit, Tipp, Bucholz, Nurnberger, Hesselbrock, Crowe, & Kramer, 1997). Co-morbidity rates for alcohol abuse in panic disorder patients range as high as 42% in some studies compared to 6% in populations who do not have an anxiety disorder (see review by Kushner, Sher, & Beitman, 1990). Lehman, Brown and Barlow (1998) have observed that panic disorder patients report using alcohol to cope with feared situations. Similarly, post traumatic stress disorder symptoms in substance abusers have been associated with heavy alcohol consumption in negative situations including unpleasant emotions, physical discomfort and conflict with others (Stewart, Conrod, Samoluk, Pihl & Dongier, 2000) and with increased incidence of alcohol abuse problems (see reviews by Stewart, 1996, 1997). Stewart et al. (2000) have provided evidence that anxiety sensitivity mediates the relationship between heavy drinking in negative contexts and post traumatic stress disorder symptoms in substance abusers. In other words, substance abusers with significant post traumatic stress disorder symptoms tend to drink in negative contexts, such as when experiencing symptoms of physical discomfort, at least partially because they are fearful of anxiety-related bodily symptoms. Life-time co-morbidity rates for alcoholism have also been shown to be significantly higher in social phobics (Schuckit et al., 1997), another anxiety disorder characterized by high anxiety sensitivity levels (Cox et al., 1999). In addition, researchers have noted a strong link between social

anxiety and heavy drinking among university students (Burke & Stevens, 1999). Not surprisingly, social anxiety is also characterized by high levels of anxiety sensitivity (Ball, Otto, Pollack, Uccello, & Rosenbaum., 1995). Conversely, anxiety disorders such as obsessive compulsive disorder and specific phobia, which are not characterized by elevated anxiety sensitivity levels (Cox et al., 1999; Taylor et al., 1992), do not appear to share this markedly increased risk for alcohol-related problems (Kushner et al., 1990; Schuckit et al., 1997). We (Stewart et al., 1999) have suggested that anxiety sensitivity may play the role of a mediator between anxiety disorders and the development of alcohol abuse.

Studies of Alcohol and Drug Abusers

While the preceding co-morbidity findings offer indirect support for a relationship between anxiety sensitivity and alcohol abuse, studies of clinically diagnosed substance abusers have provided more direct evidence for an anxiety sensitivity-alcohol abuse link. A recent study tested personality risk factors for substance abuse in a community sample of substance-abusing women (Conrod, Pihl, Stewart & Dongier, 2000). Using cluster analysis, Conrod, Pihl et al. (2000) found that female substance abusers characterized by an anxiety-sensitive personality subtype were at greater lifetime risk for anxiolytic dependence. Considering the evidence for alcohol as an anxiolytic agent (see reviews in Greely & Oei, 1999; and Sher, 1987), these findings suggest that individuals high in anxiety sensitivity could abuse and become dependent on alcohol for its anxiety-dampening qualities. Similarly, another study examined substance-abusing men seeking treatment for addictions (Norton, Rockman, Ediger, Pepe, Goldberg, Cox & Asmundson, 1997). Men who scored highly on the Anxiety Sensitivity Index were more likely to

prefer alcohol as their drug of choice compared to men who had low Anxiety Sensitivity Index scores, irrespective of their level of anxiety (measured on the Beck Anxiety Inventory: Beck & Steer, 1990).

Karp (1993) reported that individuals who carried a DSM-III-R (Diagnostic and Statistical Manual of Mental Disorders, 3rd edition - Revised: American Psychiatric Association, 1987) diagnosis of either alcohol abuse or alcohol dependence reported scores on the Anxiety Sensitivity Index that were significantly higher than non-clinical norms. This finding remained significant regardless of whether a co-morbid anxiety disorder was present (Karp, 1993). We (Stewart et al., 1999) presented two possibilities to explain these results. First, we suggested that anxiety sensitivity may be a pre-morbid vulnerability factor in the development of alcohol problems. That is, anxiety sensitivity may be a preliminary link in a causal chain that leads to alcohol abuse and dependence. Alternatively, we raised the possibility that high levels of anxiety sensitivity could result from heavy, chronic alcohol consumption. This interpretation is consistent with a theory that high levels of autonomic reactivity may contribute to higher levels of anxiety sensitivity (Reiss & McNally, 1985). We (Stewart et al., 1999) suggested that withdrawal symptoms following heavy drinking could lead to increased autonomic reactivity and subsequently to increased anxiety sensitivity. But even in the case where alcohol abuse contributes to the initial development of anxiety sensitivity, it should be recognized that coping-oriented usage of anxiolytic agents (such as alcohol: Greely & Oei, 1999; Sher, 1987) may maintain high anxiety sensitivity levels by preventing exposure to interoceptive anxiety cues (Stewart & Westra, 1996; Westra & Stewart, 1998). In fact, preventing exposure to anxiety symptoms may reinforce the belief structure that such

sensations are dangerous and even increase anxiety sensitivity levels (Fava et al., 1994; Fava, 1996).

## Studies with Non-Alcoholic Young Adults

The two possibilities noted above highlight a general interpretative problem in using alcoholic participants in this area of research - namely, the confounding of cause and consequence (cf., Pihl & Stewart, 1991) in examining the anxiety sensitivity-alcohol abuse relationship. One solution to this dilemma involves studying young adults with high anxiety sensitivity who do not yet show signs of alcohol abuse or dependence. If young, non-alcoholic, high anxiety sensitive adults fit a variety of risk profiles for the development of alcohol problems, this would provide more compelling evidence for the idea that high anxiety sensitivity is a risk factor for later alcohol abuse (McNally, 1996; Stewart et al., 1999) than the evidence gleaned from studies of alcoholics.

### Self-Report Studies

A series of self-report studies have been conducted to examine the association, if any, of anxiety sensitivity and maladaptive drinking patterns. These studies involve examinations of excessive alcohol consumption, risky situational drinking, risky motivations for alcohol use, and pre-morbid alcohol abuse symptoms in samples of young, non-alcoholic, high anxiety sensitive individuals.

Excessive Alcohol Consumption in High Anxiety Sensitive Individuals. Several studies have shown a link between high anxiety sensitivity levels and increased levels of alcohol consumption. Stewart, Peterson and Pihl (1995) found more frequent drinking on a per week basis in a non-clinical sample of young women with high anxiety sensitivity compared to a similar sample of low anxiety sensitive women. Similarly, they found that

a non-clinical sample of young women with high anxiety sensitivity levels reported drinking to the point of intoxication almost five times more often than women with low levels of anxiety sensitivity (77 occasions per year compared to 16.2 occasions per year). A more recent study examined whether these effects were maintained for men as well as women. Stewart, Zvolensky, and Eifert (2001) looked at a university population of young women and young men and found that high levels of anxiety sensitivity were associated with increased drinking behaviours across genders. High anxiety sensitive participants reported drinking more frequently in a typical week than moderate or low anxiety sensitive participants. In addition, relative to low anxiety sensitive participants, high anxiety sensitive participants reported more frequent excessive drinking. Comparably elevated drinking patterns have been observed in individuals suffering from panic disorder with agoraphobia (Cox, Swinson, Shulman, Kuch, & Reichman, 1993), a clinical group that is also known to be high in anxiety sensitivity. Moreover, anxiety sensitivity levels were significantly and positively related to alcohol consumption among male patients with panic disorder and agoraphobia. Several researchers have produced evidence that excessive alcohol consumption substantially increases the risk of developing alcohol problems (McNally, 1996; O'Neill, Parra & Sher, 2001; Sher, 1991; Stewart et al., 1999). For example, in a cross-sectional study of university students and university graduates, O'Neill et al. (2001) found that heavy drinking during the college years was significantly related to the development of alcohol abuse and/or dependence up to 10 years later. Thus, given evidence for a link between excessive alcohol use and later alcohol-use disorders, it is reasonable to predict that higher frequency and volume of drinking in high anxiety sensitive young adults may increase their chances of

experiencing future alcohol abuse or dependence.

High Anxiety Sensitive Individuals and Risky Drinking Situations. Using factors derived from the Inventory of Drinking Situations (IDS-42; Annis, Graham & Davis, 1987), Samoluk and Stewart (1998) observed that Anxiety Sensitivity Index scores in a large sample of university drinkers were significantly related to self-reported frequency of alcohol consumption on a factor measuring negatively reinforcing drinking situations. That is, there was a positive correlation between Anxiety Sensitivity Index scores and drinking frequency in situations where alcohol use might ease negative affect, unpleasant somatic symptoms, and the discomfort associated with unpleasant interactions with others. In contrast, no significant correlation emerged between anxiety sensitivity levels and degree of drinking on a factor measuring drinking in positively reinforcing situations such as those involving pleasant times with others or pleasant emotions. It has been demonstrated that the use of alcohol in "negative" contexts is related to increased incidence of alcohol problems (Cunningham, Sobell, Sobell, Gavin, & Annis, 1995). Moreover, given that these situations do not necessitate the presence of others (e.g., drinking following interpersonal conflict), Samoluk and Stewart (1998) suggested that high anxiety sensitive adults may be more motivated than others to drink in solitary vs. social contexts. For example, a high anxiety sensitive individual who felt discouraged and inadequate after a stressful day at work may be more likely to drink at home alone rather than out with friends. Cooper, Russell, Skinner and Windle (1992) have highlighted solitary drinking as a risky pattern for alcohol abuse considering that the drinker cannot compare with others to regulate the amount he/she is consuming. Thus, Samoluk and Stewart's (1998) suggestion that high anxiety sensitive individuals may be at increased

risk for future alcohol abuse and/or dependence has some converging support.

High Anxiety Sensitivity and Risky Motives for Drinking. Individuals report drinking alcohol for a variety of reasons. Some of these reasons appear to be associated with increased drinking levels and alcohol related problems. For example, it has been suggested that individuals who drink for "coping" related reasons (e.g., drinking to deal with emotional upset) are at greater risk of developing alcohol problems than individuals who drink for "social" reasons (e.g., drinking to have fun with friends) (Cooper, 1994; Cooper, Russell, Skinner, & Windle, 1992; Cooper, Russell, Skinner, Frone, & Mudar, 1992; Stewart et al., 1999). Individuals who drink to cope tend to use excessive levels of alcohol and also tend to drink alone, situations that have been discussed previously as risk factors for future alcohol abuse/dependence (Carrigan, Samoluk & Stewart, 1998; Cooper, Russell, Skinner, & Windle, 1992; Sher, 1991). In contrast, individuals who are motivated to drink for social reasons tend to consume less and are at lower risk for alcohol problems (Cooper, Russell, Skinner, & Windle, 1992). Not surprisingly, high anxiety sensitive individuals have been shown to endorse coping motivations for alcohol use (i.e., drinking to deal with negative affect) at a significantly higher rate than low anxiety sensitive individuals (Stewart & Zeitlin, 1995). Conversely, low anxiety sensitive individuals have reported significantly more social motivations for alcohol use than high anxiety sensitive individuals (Stewart & Zeitlin, 1995). These results were supported by later work that indicated a positive correlation between Anxiety Sensitivity Index levels and drinking primarily for coping motives, and negative correlations between Anxiety Sensitivity Index levels and drinking primarily for social affiliation reasons (Stewart, Karp, Pihl & Peterson, 1997). Building upon this work, Stewart, Zvolensky, et al. (2001)

provided the strongest evidence to date for the idea that coping motives may lead to excessive drinking behaviour in high anxiety sensitive individuals. Using a sample of male and female university students, Stewart, Zvolensky, et al. (2001) looked at the relationship between Anxiety Sensitivity Index scores, drinking motives and a self-report quantity-frequency measure of average drinking levels. They found that negatively reinforcing, coping motives for drinking mediated the link between increased levels of anxiety sensitivity and increased drinking behavior across the entire sample of men and women. An association between high anxiety sensitivity and "risky" coping motives has even been seen in teenage drinkers, suggesting that these relationships may be formed very early in an individual's drinking career (Comeau, Stewart, & Loba, 2001).

Drinking-Related Problems in Non-Alcoholic High Anxiety Sensitive Individuals. Perhaps the most direct support for a high anxiety sensitivity-alcohol abuse link comes from research conducted with non-alcoholic young males with no family history of alcohol problems. Conrod, Pihl and Vassileva (1998) found that a significantly greater proportion of high anxiety sensitive males, compared to low anxiety sensitive males, reported at least one drinking related problem on a self-report measure of alcohol problems known as the Brief Michigan Alcoholism Screening Test (Pokorny, Miller & Kaplan, 1972; see Appendix C). Conrod et al. (1998) suggested that these results might indicate that high anxiety sensitive males are at increased risk for developing drinking problems compared to low anxiety sensitive men.

Summary of Self-Report Findings

The findings from self-report studies have shown that high anxiety sensitive young adults are more prone to excessive drinking and alcohol abuse symptoms, report

more unhealthy drinking motivations (i.e., drinking to cope), and more drinking in risky situations (i.e., negatively reinforcing contexts), compared to low (and sometimes compared to moderate) anxiety sensitive students (Conrod et al., 1998; Samoluk & Stewart, 1998; Stewart, Karp et al., 1997; Stewart, Peterson, et al., 1995; Stewart & Zeitlin, 1995; Stewart, Zvolensky, et al., 2001). These studies suggest that anxiety sensitivity may represent a personality risk factor for substance abuse (Stewart et al., 1999) and support an anxiety sensitivity risk model of alcohol abuse.

## Risk Models for Alcohol Abuse

### The Tension Reduction Hypothesis

The "anxiety sensitivity risk model" has its roots in the tension reduction hypothesis for substance abuse (Conger, 1956). Conger used results from an approach-avoidance conflict paradigm to argue that alcohol consumption in rats and cats was reinforced owing to the drive-reducing properties of alcohol. That is, he observed that alcohol reduced the behavioural avoidance component of the fear response in animals and inferred that they would learn to consume alcohol to achieve this effect. This hypothesis was then generalized by subsequent researchers to make predictions about alcohol use in both humans and animals, in studies using a wide range of behavioural, self-report, and autonomic measures of tension (see review by Greely & Oei, 1999). The general tension reduction hypothesis for alcohol is composed of two basic tenets. The first tenet is that alcohol reduces an aversive state known as "tension." The second tenet holds that animals and humans will be negatively reinforced to consume alcohol for its tension-reducing properties (Greely & Oei, 1999). In other words, they will learn to consume alcohol

because of its tension-reducing properties and due to these learning experiences, will be more likely to drink in future when experiencing tension.

Problems with the Traditional Tension Reduction Hypothesis. Although the tension reduction hypothesis benefits from being parsimonious and intuitively appealing, several criticisms have been raised about its use as a general model for the development of alcohol problems. First, Sher (1987) has noted that tension is a "vague and difficult to define construct" (Sher, 1987, p. 228). In particular, Greely and Oei (1999) discussed the difficulty in understanding how various experimental measures, including self-report, galvanic skin response, cardiovascular, and noradrenaline levels, map onto the construct of "tension." Second, the tension reduction hypothesis does not consider the situational context under which tension reduction effects may take place (Levenson, Sher, Grosman, Newman & Newlin, 1980; Sher, 1987). For example, Levenson et al. (1980) explained that that it was crucial to examine the timing of alcohol effects (either during anticipation of a stressor or immediately following the onset of a stressor) in order to understand the specific tension-reducing properties of alcohol. Third, there is no consideration in the tension reduction hypothesis of the potential effects of cognitive or individual difference variables. These include individual expectancies about the outcomes of consuming alcohol (see review in Goldman, Del Boca & Darkes, 1999), pre-alcoholic personality characteristics (Cox, 1987; Stewart et al., 1999; Welte, 1985) and familial genetic risk for alcoholism (Conrod, Pihl & Ditto, 1995; Conrod et al., 1998). Fourth, the hypothesis does not accommodate alternative motivations for drinking other than tension reduction, such as drinking to cope with depressive affective states (Stewart, Karp, et al., 1997), social affiliative motives (Samoluk, Stewart, Sweet & MacDonald, 1999), or drinking to

enhance a positive emotional state (Stewart & Zeitlin, 1995). Finally, alternative alcohol effects to tension reduction are not considered in the tension reduction hypothesis. In fact, several studies have indicated that alcohol may actually increase physiological and self-report measures of tension in certain contexts (Dengerink & Fagan, 1978; Levenson et al., 1980; MacDonald & Stewart, 1996). In a review of the tension reduction literature, Greely and Oei (1999) summed up these disclaimers by noting that

> "...some individuals...who hold certain beliefs about alcohol, will, under certain circumstances...consume alcohol for its stress-response dampening effects. However, these same individuals may also consume alcohol at other times for other reasons..." (Greely & Oei, 1999, p. 41)

We (Stewart et al., 1999) noted that the tension reduction hypothesis of alcohol consumption could still be applicable to three specific populations – those with certain anxiety-related personality traits, those with certain anxiety disorders, and those with a particular sensitivity to alcohol-induced dampening of the stress response.

An Alternative to the Tension Reduction Hypothesis – Stress Response Dampening

In light of conflicting results as to the required circumstances and generalizability of the tension reduction hypothesis, Greely and Oei (1999) advised that the tension-reducing properties of alcohol must be studied under conditions in which tension reduction effects are strong and uncontaminated. They proposed the use of a stress response dampening model (Sher, 1987) to replace the more traditional Tension Reduction Hypothesis. Sher (1987) defined stress response dampening as a sub-theory of the tension reduction hypothesis. Indeed, as a model, stress response dampening shares important features with its predecessor. Both models propose that alcohol consumption can lead to a dampening of stress (or "tension") responses. Moreover, both models argue

that alcohol consumption is consequently reinforced via operant conditioning processes. Where the models differ is that stress response dampening does not view stress as necessary or sufficient for the initiation of alcohol consumption. Indeed, the stress response dampening model suggests that drinking in response to a stressor is most likely when alternative coping responses are not available and when the probability of drinking leading to increased stress is low (e.g., drinking at home alone vs. drinking during a stressful meeting at work). Furthermore, stress response dampening effects are thought to be influenced by individual differences in terms of sensitivity to stress response dampening rather than a general tendency to experience stress (Sher, 1987). Finally, the stress response dampening model is clearer about the involvement of stressful circumstances. For example, while the tension reduction hypothesis would predict alcohol dampening of baseline levels of tension, the stress response dampening model would more specifically predict alcohol dampening of the natural increase in tension that occurs in response to stress.

Greely and Oei (1999) listed four benefits of using the stress response dampening model. First, they noted that such a model was more specific than the traditional tension reduction hypothesis in its predictions (e.g., the timing of alcohol effects). Second, they explained that the model could account for alcohol's effects in decreasing and increasing anxious responses to stressors. Third, they noted that the model acknowledges that not all stressful situations elicit drinking. Finally, they noted that individual differences (such as familial genetic history, pre-alcoholic personality traits, or co-morbid anxiety disorders) could be considered as predisposing factors in the relationship between alcohol and stress response dampening. Sher (1987) also noted the benefits of testing a stress response

dampening model experimentally, in that alcohol can be consumed and absorbed before the onset of a stressor. This is an important methodological consideration, given that stress response *recovery* is difficult to achieve in a lab-based setting, especially in humans. That is, it is difficult to maintain the effects of a stressor for a prolonged period until alcohol is consumed, absorbed and can begin to exert its pharmacological effects. Moreover, unlike the tension reduction hypothesis, the stress response dampening model predicts alcohol consumption in anticipation of a stressor and consequent dampening of the stress response. In contrast, the tension reduction hypothesis predicts alcohol consumption in response to stress (Sher, 1987).

The Anxiety Sensitivity Risk Model for Alcohol Abuse

Several theoretical models have been put forward to explain the association between anxiety sensitivity and alcohol abuse (see review by Stewart & Kushner, 2001). Moderator models (Baron & Kenny, 1986) contend that anxiety s ensitivity influences the conditions under which alcohol consumption is related to either anxiety level or anxiety dampening. One of these models states that an individual's level of anxiety sensitivity determines the strength of association between high anxiety levels and alcohol consumption (Stewart & Kushner, 2001). That is, individuals with high levels of anxiety are predicted to exhibit increased alcohol consumption, particularly if those individuals also have high levels of anxiety sensitivity. McNally (1996) has suggested that the increased frequency of anxiety experiences characteristic of a highly trait anxious individual, combined with a fear of those experiences as a result of high anxiety sensitivity, would provide more opportunities for an individual to learn about the arousal-dampening properties of alcohol and thus increase potential for alcohol abuse. Stewart

and Kushner (2001) also noted that Reiss' (1991) expectancy theory fits this moderator model, in that anxiety sensitivity would heighten anxiety expectancies. These heightened expectancies, in turn, could provide motivation to avoid anxiety experiences through the use of alcohol. This coping-motivated usage of alcohol could ultimately lead to alcohol problems (Cooper, Russell, Skinner, & Windle, 1992).

A second moderator model posits that anxiety sensitivity levels moderate the strength of the relationship between alcohol consumption and dampened anxious reactivity to stressors (Stewart & Kushner, 2001). That is, alcohol is proposed to have a strong dampening effect on stress and anxiety responses in highly anxiety sensitive people but a relatively weak dampening effect on anxious reactivity in low anxiety sensitive individuals. Thus, increased sensitivity to the reactivity-dampening effects of alcohol among high anxiety sensitive individuals may negatively reinforce alcohol consumption in this population, as high anxiety sensitive individuals learn to self-medicate their anxiety with alcohol. This model has received some empirical support (Conrod et al., 1998; Stewart & Pihl, 1994).

A mediator (Baron & Kenny, 1986) model has also been proposed in which anxiety sensitivity plays a more direct, causal role in alcohol use and abuse by increasing anxiety symptoms which, in turn, mediate the anxiety sensitivity – alcohol consumption relationship (Stewart & Kushner, 2001). Thus, anxiety sensitivity is the more distal of the two causal factors, while anxiety levels are the more proximal causal factors of alcohol intake. In this model, when anxiety sensitive individuals experience anticipatory anxiety, their fear of anxiety sensations heightens their anticipatory anxiety state. These higher levels of anticipatory anxiety lead to increased avoidance or escape behaviours,

such as alcohol consumption. That is, it is theorized that highly anxiety sensitive people will consume more alcohol to avoid/escape experiencing these heightened unpleasant anticipatory anxiety symptoms. In contrast, individuals with lower levels of anxiety sensitivity do not experience similarly elevated anticipatory anxiety and thus are predicted to exhibit reduced avoidance/escape behaviours such as alcohol consumption.

The anxiety sensitivity mediator model shares some similarities to traditional "high risk" models involving trait anxiety (e.g., Welte, 1985). Both models contend that certain personality variables (anxiety sensitivity or trait anxiety) are associated with increased risky alcohol use. Moreover, both models incorporate increased state (anticipatory) anxiety as the mediator between the personality factor (anxiety sensitivity or trait anxiety) and risk for alcohol abuse. Specifically, both highly anxiety sensitive individuals and highly trait anxious individuals are thought to be at heightened risk for experiencing elevated state anxiety in response to stress. This heightened state anxiety, in turn, is thought to increase the probability that an individual might experience potentially negatively-reinforcing consequences of drinking (i.e., stress response dampening effects). The anxiety sensitivity and trait anxiety risk models differ, however, in two important respects. First, under stressful conditions involving heightened bodily sensations of arousal, anxiety sensitivity is a better predictor than trait anxiety of state increases in anxious responding (Rapee & Medoro, 1994). Thus, high anxiety sensitive individuals, compared to highly trait anxious individuals, would have increased opportunities for learning about the negatively-reinforcing effects of alcohol in circumstances that involve bodily arousal. Second, trait anxiety models assume that anxiety is a naturally aversive state (Welte, 1985) and that trait anxious people would be

motivated to learn to drink to alleviate this aversive experience. In contrast, the anxiety sensitivity risk model does not assume that anxiety experiences are inherently aversive (Reiss, 1991; Reiss, 1997). As opposed to trait anxiety, anxiety sensitivity specifically involves fear of the occurrence of anxiety symptoms, and a heightened emotional response to such symptoms (Reiss, 1991). Thus, it is high anxiety sensitive individuals who should be most motivated to learn to use a drug (i.e., alcohol) that is capable of reducing bodily arousal symptoms, subjective anxiety states, and/or the tendency to interpret sensations as dangerous.

Experimental evidence has supported an anxiety sensitivity risk model as a superior predictor of alcohol abuse compared to more traditional trait anxiety models. First, Karp (1993) found a positive relation between anxiety sensitivity and positive alcohol expectancies (i.e., beliefs about the consequences of drinking alcohol) in an alcoholic sample. In particular, anxiety sensitivity levels were significantly and positively related to expectancies that alcohol would reduce tension and promote relaxation. In contrast, trait anxiety levels were unrelated to tension reduction and relaxation expectancies in alcoholics. Thus, in an alcoholic sample, it was shown that the fear of anxiety sensations, as opposed to the frequency of anxiety experiences, is a better predictor of tension reduction expectancies. Meanwhile, positive alcohol expectancies, particularly expectancies for tension reduction, have been demonstrated to predict heavy drinking (e.g., Rutledge & Sher, 2001) and alcohol abuse problems (see reviews by Goldman, Darkes & Del Boca, 1999; Goldman, Del Boca, & Darkes, 1999). Additionally, Sher (1987; 1991) reviewed a number of studies indicating that trait anxiety was not a good predictor of alcohol use or developing problems. Thus, compelling

evidence supports the further investigation of a specific anxiety sensitivity risk model for alcohol abuse (Stewart et al., 1999) compared to a general, relatively unsupported, trait anxiety model (Sher, 1987; Sher, 1991).

## Evaluating the Anxiety Sensitivity Risk Model

The anxiety sensitivity risk model contends that alcohol consumption should be negatively reinforced in high anxiety sensitive individuals who are particularly receptive to alcohol's ability to reduce frightening bodily arousal sensations, thereby increasing their risk for developing alcohol abuse, alcohol dependence, or both (Stewart et al., 1999). Stewart and Kushner (2001) listed three criteria for supporting a mediator model for the relationship between anxiety sensitivity and alcohol abuse. First, they noted that the relationship between anxiety sensitivity and state anxiety levels must be both positive and significant. That is, individuals with high levels of anxiety sensitivity should demonstrate significant anxious reactivity in response to stressors that induce bodily arousal. This would support the idea that high anxiety sensitive people are at risk of using alcohol to cope with stress because their reactivity may provide them with opportunities for learning about the tension-reducing properties of alcohol. Further, Sher (1987) has suggested that it is not anxiety per se, but anxiety that is experienced as aversive and/or debilitating (conditions seen in high anxiety sensitive individuals' responses to arousal-inducing stressors: e.g., Asmundson et al., 1994; Donnell & McNally, 1989; Rapee & Medoro, 1994) which may lead to alcohol consumption as a response.

Second, alcohol consumption must be demonstrated to offer an effective avoidance or escape response from anxiety, with heightened state anxiety motivating increased alcohol consumption in highly anxiety sensitive individuals (Stewart &

Kushner, 2001). Thus, in meeting the first part of this criterion, evidence of alcohol-induced dampening of anxious reactivity in high anxiety sensitive individuals is required. This would lend credence to the idea that alcohol consumption is a potentially rewarding activity for high anxiety sensitive individuals owing to alcohol's ability to reduce fearful anxious sensations. In addition, a demonstration that alcohol dampens anxious reactivity to stressors in high anxiety sensitive individuals to a larger degree than in low anxiety sensitive individuals would support the idea that alcohol consumption may be differentially reinforced in the high anxiety sensitive population. It would also support a slight modification to the first tenet of the tension reduction hypothesis – alcohol reduces tension – by highlighting a unique receptivity to alcohol's reactivity dampening effects among high anxiety sensitive individuals.

The remaining component of Stewart and Kushner's (2001) second criterion, namely, alcohol consumption as an instrumentally conditioned response to state anxiety, could be tested in a lab-based setting. If high anxiety sensitive individuals consume more alcohol in response to an anxiety-provoking stressor than their low anxiety sensitive counterparts, this would be consistent with the presence of an instrumentally conditioned response specific to high anxiety sensitive people. Moreover, it would support the second tenet of the tension reduction hypothesis, but specifically for high anxiety sensitive individuals – i.e., consumption of alcohol for its tension-reducing properties. Stewart and Kushner's (2001) third criterion states that the strength of the relationship between high anxiety sensitivity levels and increased alcohol use should be attenuated by controlling the previous two relationships (high anxiety sensitivity – increased state anxiety, increased state anxiety – alcohol consumption as an avoidance/escape response), in line

with suggestions by Baron and Kenny (1986) for establishing mediation in psychological research.

Experimental Tests of the Anxiety Sensitivity Risk Model

While self-report measures of alcohol use and effects provide a glimpse into people's naturalistic drinking behaviour, there are a number of limitations to the use of self-reports. First, the accuracy of such reports can be an issue, considering that the correlations between reported alcohol use by experimental subjects and reported alcohol use by collateral informants are moderate at best (Sobell & Sobell, 1990). Subjectivity in judgment, retrospective memory biases, sensitivity to demand characteristics, ability to introspect, response biases, and ability to scale experiences on Likert-type scales are all known to influence information gathered from self-reports (Baker & Brandon, 1990; Sher, 1987; Sobell & Sobell, 1990; Stewart et al., 1999).

Lab-based, analogue studies of alcohol use and effects offer a number of advantages over self-report methodologies. First, lab-based studies allow for a far greater degree of experimental control in examining alcohol effects (Sobell & Sobell, 1990). Moreover, theoretical models can be examined more objectively through prospective testing vs. retrospection (Stewart et al., 1999). Finally, the central tenets of the anxiety sensitivity risk model can be evaluated directly in a lab-based setting. That is, differential sober reactivity, alcohol-induced dampening of reactivity, differential dampening effects, and increased alcohol consumption in response to stress can be examined prospectively in high anxiety sensitive individuals as compared to controls.

Sober Reactivity Studies in High Anxiety Sensitive Individuals

A number of laboratory studies have demonstrated increased sober reactivity to relevant stressors in high anxiety sensitive participants. Holloway and McNally (1987) reviewed literature that showed that patients with panic disorder experienced panic symptoms when exposed to the so-called "biological" stress challenges such as sodium lactate infusion, CO2 inhalation, and voluntary hyperventilation, whereas non-panic disorder patients did not (or were significantly less likely to do so). These challenges share a common denominator – they all elicit arousal-related bodily sensations. Holloway and McNally (1987) suggested that, given the high degree of concordance in the belief systems of high anxiety sensitive non-clinical individuals and panic disorder patients, people with high levels of anxiety sensitivity should respond in a similar fashion to biological challenges. Using a non-clinical, young adult sample, Holloway and McNally (1989) had participants hyperventilate for a short period of time and looked at the level of their anxious response. They found that high anxiety sensitive participants reported significantly more hyperventilation sensations and anxiety than low anxiety sensitive participants. They concluded from these results that a fear of anxiety, as seen in high anxiety sensitive people, amplifies anxious responding to a biological challenge irrespective of a formal anxiety disorder diagnosis.

Although Holloway and McNally (1987) excluded the presence of anxiety disorders as an explanatory variable for high anxiety sensitive individuals' responses, they did not account for potential differences in trait anxiety or the possible influence of panic attack history. Donnell and McNally (1989) tested the idea that heightened reactivity to challenge stressors among high anxiety sensitive individuals may actually be

a result of a history of panic attacks, given the strong relation of elevated anxiety sensitivity to panic history (see review by Cox et al., 1999). Using hyperventilation as the arousal-induction "stressor", Donnell and McNally (1989) tested non-clinical, young adult volunteers who were high and low in anxiety sensitivity. Both groups contained persons with and without a history of panic attacks. The results indicated that high anxiety sensitive participants reported more anxiety and somatic sensations in response to hyperventilation than low anxiety sensitive participants regardless of panic attack history. Furthermore, in the absence of high anxiety sensitivity, panic attack history did not contribute to increased reactivity to the hyperventilation challenge.

Similarly, Rapee and Medoro (1994) raised the possibility that, given moderately strong correlations between anxiety sensitivity and measures of trait anxiety (e.g., Lilienfeld, 1996; McNally, 1996), high anxiety sensitive people might show enhanced reactivity to challenges because of higher levels of trait anxiety rather than because of a specific fear of anxiety-related bodily sensations. They tested this idea using a sample of high and low anxiety sensitive university students in a voluntary hyperventilation procedure. For their dependant measure, they developed a self-report inventory known as the Hyperventilation Questionnaire (Rapee & Medoro, 1994; see Appendix G), a measure that has the benefit of simultaneously tapping into several different domains of anxious responding to hyperventilation. These include somatic (bodily sensations), cognitive (negative thoughts), and affective (feelings of fear) symptoms. Rapee and Medoro (1994) replicated previous findings of increased sober reactivity in high vs. low anxiety sensitive participants even when trait anxiety levels were held constant. These results were supported by Sturges, Goetsch, Ridley, and Whittal (1998), who noted that anxiety

sensitivity accounted for additional variance beyond trait anxiety in accounting for high anxiety sensitive individuals' reports of subjective-emotional distress in response to voluntary hyperventilation. Rapee and Medoro (1994) also found that anxiety sensitivity accounted for unique variance in response to hyperventilation on affective and cognitive measures over and above the variance accounted for by trait anxiety. Interestingly, anxiety sensitivity did not account for additional variance on measures of somatic symptoms. They concluded that high and low anxiety sensitive individuals appear to experience the same level of somatic sensations but interpret them differently. That is, high anxiety sensitive people interpret these sensations as dangerous, amplifying their affective and cognitive responses, while low anxiety sensitive people view the same sensations as unpleasant but harmless. This interpretation is supported by recent findings that, under stressful conditions, high anxiety sensitive individuals estimate their heart rate to be significantly higher than low anxiety sensitive individuals, even when actual heart rate reactivity is the same in both groups (Stewart, Buffet-Jerrott, & Kokaram, 2001).

Although voluntary hyperventilation has emerged as a popular direct arousal provocation procedure which elicits consistent emotional hyper-reactivity effects among high anxiety sensitive participants (Asmundson et al., 1994; Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee & Medoro, 1994; Sturges et al., 1998), other arousal-induction challenge procedures have also shown that levels of anxiety sensitivity predict emotional reactivity. These include $CO_2$ inhalation challenges (Beck, Shipherd, & Zebb, 1996; Beck & Wolf, 2001; Eke & McNally, 1996; McNally & Eke, 1996) and oral administration of caffeine (Sturges & Goetsch, 1996; Telch, Silverman, & Schmidt, 1996). Traditional lab-based stressors such as exposure to unavoidable shock (Conrod et

al., 1998) and exposure to signaled loud noise bursts (Stewart & Pihl, 1994) have also been used to indirectly bring on the arousal-related bodily sensations that are so feared by high anxiety sensitive individuals; these procedures have also been demonstrated to elicit emotional hyper-reactivity in high anxiety sensitive individuals. Donnell and McNally's (1989) original findings have been replicated in high anxiety sensitive individuals regardless of history of panic attacks, trait anxiety levels, or the particular method of arousal-induction utilized.

Alcohol Administration Studies

To date, a relatively small number of studies involving alcohol administration have been conducted to directly test the idea that high anxiety sensitive individuals may receive greater negative reinforcement for alcohol consumption than low anxiety sensitive individuals. Such experimental support is necessary to build a case for the anxiety sensitivity risk model for alcohol problems. We (Stewart et al., 1999) have suggested that differences in self-reported drinking behaviours may be a result of a unique response to alcohol in high anxiety sensitive individuals. Thus, if unique alcohol responding can be demonstrated in this population, then a case can be made that high anxiety sensitive people are especially sensitive to alcohol consequences that may be negatively reinforcing.

Alcohol-induced Stress Response Dampening in Panic Disorder Patients. Considering that panic disorder is characterized by high levels of anxiety sensitivity (Stewart, Knize, et al., 1992; Taylor et al., 1992), individuals with panic disorder represent a clinical group that can provide an informative, indirect look at the influence of anxiety sensitivity on responses to alcohol. One study examined the effects of alcohol

on panic and anxious responses in a group of non-alcoholic panic disorder patients (Kushner et al., 1996). Half of the group received a placebo beverage while the other half was administered a moderately intoxicating dose of alcohol. Both groups were then exposed to a $CO_2$ inhalation stressor. Alcohol was shown to reduce self-report measures of anxiety and panic symptoms both before and after the onset of the stressor. Alcohol-induced dampening of physiological measures, while in the same direction, failed to achieve statistical significance. Based on these results, Kushner et al. (1996) suggested that panic disorder patients might learn to use alcohol as a means of gaining anxiety relief. Considering that high anxiety sensitive individuals share a belief system with panic disorder patients about the dangerousness of bodily arousal cues (Peterson & Plehn, 1999), these findings strongly suggest that alcohol could also provide a relief mechanism for high anxiety sensitive people without panic disorder, and subsequently become negatively reinforcing for this non-clinical group as well.

Alcohol-induced Stress Response Dampening in High Anxiety Sensitive Individuals. Stewart and Pihl (1994) directly tested the idea that alcohol use might be negatively reinforced in a non-clinical population characterized by high anxiety sensitivity levels. They tested female university students who had high, moderate, or low levels of anxiety sensitivity. In a within-subjects design, each participant was exposed to a signaled noise burst stressor, first while sober and then following absorption of a 1.0 ml/kg dose of alcohol. Autonomic responses to the stressor were measured via skin conductance levels and heart rate changes. Subjective emotional responses to both anticipation of, and receipt of, noise bursts were measured through the use of two distinct self-report questionnaires. Replicating previous research, Stewart and Pihl (1994)

observed that sober high anxiety sensitive individuals reported more subjective-emotional arousal (e.g., tension, anxiety) in anticipation of the stressor compared to their low anxiety sensitive counterparts. However, unlike some previous findings (Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee & Medoro, 1994; Sturges et al., 1998), there were no differences between anxiety sensitive groups in sober subjective distress responses (e.g. , discomfort, pain) immediately following the stressor. Stewart and Pihl (1994) also found that scores on the Anxiety Sensitivity Index were most highly correlated with sober subjective-emotional reactivity, marginally correlated with sober skin conductance levels and unrelated to sober cardiovascular reactivity – a pattern that is consistent with contentions that anxiety sensitivity effects on anxiety production are primarily cognitively mediated (Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee & Medoro, 1994; Stewart et al., 1999). In addition, alcohol consumption dampened subjective-emotional responses to the noise bursts in all groups but produced the greatest reduction in these responses in the high anxiety sensitive group. Selective dampening of skin conductance levels in the high anxiety sensitive group was only marginally significant. Stewart and Pihl (1994) used these results to suggest that anxiety sensitivity moderates (Baron & Kenny, 1986) the emotional-reactivity dampening effects of alcohol.

Conrod et al. (1998) partially replicated Stewart and Pihl's (1994) results in high anxiety sensitive men through use of an unavoidable shock paradigm. The subjective-emotional measures used in the study were similar to those used by Stewart and Kushner (1994), except that participants were asked to rate anticipation of, and response to, shock rather than noise bursts. Sober high anxiety sensitive men were found to show higher

subjective-emotional, but not physiological, responsiveness to the stressor compared to low anxiety sensitive men. Conrod et al. (1998) also showed that alcohol dampened subjective emotional responses for both groups. However, unlike Stewart and Pihl's (1994) findings, the high anxiety sensitive men reported elevated sober subjective-emotional reactivity compared to low anxiety sensitive students both while anticipating, and following receipt of, the shock stressor. Stewart and Pihl (1994) only looked at subjective emotional response (e.g., tension, anxiety) in anticipation of the stressor in their study but not following the stressor (instead, a measure of pain/discomfort was used to measure responses to the noise burst stressor). In addition, alcohol-induced subjective-emotional dampening was not shown to be differentially stronger in the high anxiety sensitive group compared to the low anxiety sensitive group in the Conrod et al. (1998) study. In contrast, skin conductance responses were selectively dampened in the high anxiety sensitive group, similar to the findings of Stewart and Pihl (1994). According to Fowles (1980), skin conductance levels are thought to be the best physiological indicator of the anxiety response.

Despite some minor inconsistencies in results, the experimental studies conducted by Stewart and Pihl (1994) and Conrod et al. (1998) support the idea that high anxiety sensitive individuals may be motivated to consume alcohol for its emotional reactivity dampening effects. This experimental evidence is consistent with the self-report research which shows excessive drinking, drinking in risky situations, risky drinking motivations and pre-morbid alcohol abuse symptoms in high anxiety sensitive individuals (Conrod et al., 1998; Samoluk & Stewart, 1998; Stewart, Karp, et al., 1997; Stewart, Peterson, et al., 1995; Stewart & Zeitlin, 1995; Stewart, Zvolensky, et al., 2001). Interestingly, studies

using participants differing in trait anxiety levels have failed to demonstrate differential sensitivities to the reactivity dampening effects of alcohol (Keane & Lisman, 1980; Sher, 1987; Sher & Levenson, 1982). These findings support the distinction between anxiety sensitivity and trait anxiety both as constructs (McNally, 1996; Reiss, 1997) and as moderators of the reactivity-dampening effects of alcohol (Stewart et al., 1999).

Inconsistencies and Limitations in Studies of Alcohol-Induced Stress Response Dampening Among High Anxiety Sensitive Individuals. Despite strong support from Stewart and Pihl (1994) and Conrod et al. (1998) that high anxiety sensitive individuals may be negatively reinforced to use alcohol, several issues remain unresolved. First, both teams of researchers used a within-subjects design. All participants were initially tested in a non-alcohol condition followed sequentially by an alcohol condition. Thus, the apparent subjective-emotional dampening seen in the high anxiety sensitive groups following alcohol consumption may have actually represented a habituation effect. Indeed, Beck and Wolf (2001) have shown that high anxiety sensitive individuals display habituation of their subjective reports of anxiety after repeated exposure to a $CO_2$ inhalation challenge. Similar findings of habituation of subjective anxious responses have been reported in panic disorder patients (Beck & Shipherd, 1997).

Second, alcohol expectancies may have played a role in the Stewart and Pihl (1994) and Conrod et al. (1998) alcohol dampening findings, as no placebo control condition was included in either study. It has been demonstrated that beliefs about the possible outcomes of drinking alcohol, particularly positive alcohol expectancies (e.g., "alcohol reduces tension") may lead to tension reduction effects after consumption of placebo beverages (Wilson & Abrams, 1977). Without a placebo control, it is difficult to

ascertain whether experimental results are attributable to the pharmacological effects of alcohol (Sher, 1987), cognitively mediated expectancies about the potential positive outcomes of drinking alcohol (Goldman, Del Boca, & Darkes, 1999), or both.

Third, both the Stewart and Pihl (1994) and Conrod et al. (1998) studies used only one, moderately high, dose of alcohol and were unable to test possible dose-dependent alcohol effects. Thus, it remains uncertain whether alcohol-induced dampening effects in high anxiety sensitive individuals might be seen at relatively lower alcohol doses. What is also unknown is the shape of a possible alcohol dose response curve. If stress response dampening effects can be shown to increase with increasing doses of alcohol, stronger support would be gained for the idea that excessive alcohol consumption is negatively reinforced in high anxiety sensitive individuals.

Fourth, another limitation of both the Stewart and Pihl (1994) and Conrod et al. (1998) studies involved the use of indirect bodily arousal provocations: loud bursts of noise and unavoidable electric shock, respectively. Failure to observe sober hyper-reactivity among high anxiety sensitive participants immediately following the stressor in the Stewart and Pihl (1994) study may have been attributable to the fact that exposure to signaled noise is an indirect method of producing the bodily arousal sensations so feared by high anxiety sensitive individuals. A review of the literature reveals that neither noise bursts nor unavoidable shock have been commonly used in research measuring high anxiety sensitive emotional responsiveness to aversive stimulation (Asmundson et al., 1994; Beck et al., 1996; Beck & Wolf, 2001; Donnell & McNally, 1989; Holloway & McNally, 1987; McNally & Eke, 1996, Rapee & Medoro, 1994; Sturges & Goetsch, 1996; Sturges et al., 1998; Telch et al., 1996). Other stressors such as voluntary

hyperventilation, sodium lactate infusion, isoproterenol infusion, yohimbine infusion, $CO_2$ inhalation, and caffeine provocations are direct methods of producing such symptoms (see reviews by Stein & Rapee, 1999; and McNally, 1996) and thus may represent more theoretically-relevant stressors in the study of the anxiety sensitivity construct (Stewart et al., 1999).

Fifth, the Conrod et al. (1998) and Stewart and Pihl (1994) findings diverged in terms of alcohol-induced subjective-emotional reactivity dampening in high anxiety sensitive vs. low anxiety sensitive participants. Stewart and Pihl (1994) showed a heightened sensitivity to these effects among high anxiety sensitive women, while Conrod et al. (1998) noted that subjective-emotional dampening was uniform for both high and low anxiety sensitive men. It is possible that the measures used in the Stewart and Pihl (1994) and Conrod et al. (1998) studies, which assessed physical discomfort and circumscribed aspects of negative affect (i.e., tension, anxiety, worry, fear and anger), may have been inadequate to distinguish between high and low anxiety sensitive individuals. Stewart et al. (1999) have noted that it is catastrophic interpretation of anxiety symptoms which differentiates high and low anxiety sensitive individuals' response to bodily arousal. Thus, a measure which assesses subjective anxiety experience and catastrophic cognitions may better illuminate differences between these groups.

Finally, the results in the Conrod et al. (1998) and Stewart and Pihl (1994) studies may have been influenced by undetermined gender variables. While Stewart and Pihl (1994) used all women in their study and Conrod et al. (1998) used all men, previous research in sober reactivity among high anxiety sensitive individuals have used samples with a mix of genders (Asmundson et al., 1994; Beck et al., 1996; Beck & Wolf, 2001;

Donnell & McNally, 1989; Holloway & McNally, 1987; McNally & Eke, 1996, Rapee & Medoro, 1994; Telch et al., 1996).

## Testing Potential Alcohol-Induced Stress Response Dampening in High Anxiety Sensitive Individuals

Clearly, replication and extension of Stewart and Pihl's (1994) and Conrod et al.'s (1998) work is required to confirm or disconfirm a special receptivity to alcohol-induced subjective-emotional dampening in the high anxiety sensitive population. Use of a measure which taps into some of the unique fears and negative thoughts about bodily arousal among high anxiety sensitive individuals (Stewart et al., 1999) would help to highlight potential differences between high and low anxiety sensitive groups. In addition, a more theoretically relevant stressor which has been experimentally validated for use in the high anxiety sensitive population (e.g., hyperventilation) would provide a direct and ecologically sound means of eliciting the fears of anxiety sensitive participants (e.g., Rapee & Medoro, 1994). A between-subjects design would support the idea that stress response dampening effects are real and not a result of habituation (e.g., Beck & Wolf, 2001). Moreover, controlling for expectancy effects (see reviews by Goldman, Del Boca, & Darkes, 1999) by inclusion of a placebo beverage control would allow for a more definitive test of whether the pharmacological properties of alcohol contribute to stress response dampening effects in high anxiety sensitive individuals. Finally, testing different doses of alcohol would help to ascertain whether stress response dampening effects in high anxiety sensitive individuals are seen at lower doses of alcohol than those used in Stewart and Pihl (1994) and Conrod et al. (1998) and would provide information on the shape of a potential alcohol dose response curve. If a special receptivity to alcohol-

induced stress response dampening could be demonstrated in high anxiety sensitive individuals compared to their low anxiety sensitive counterparts, the anxiety sensitivity risk model of alcohol abuse (Stewart et al., 1999) would gain strong empirical support. These considerations led to the development of Study One, presented in the next chapter.

# CHAPTER TWO: Study One

## The Effects of Alcohol on the Response to Hyperventilation of Participants High and Low in Anxiety Sensitivity

The present study examined the effects of alcohol on the responses to hyperventilation challenge in individuals high and low in anxiety sensitivity. This study served as a replication and extension of previous work on sober reactivity to hyperventilation challenge in high versus low anxiety sensitive participants (Rapee & Medoro, 1994; Asmundson et al., 1994; Donnell & McNally, 1989; Holloway & McNally, 1987) as well as a further examination of the effects of alcohol on high versus low anxiety sensitive participants' responses to stress (Conrod et al., 1998; Stewart & Pihl, 1994). The design limitations noted for the studies by Stewart and Pihl (1994) and Conrod et al. (1998) study were improved upon by employing a between subjects design, a placebo control, two doses of alcohol, and the more theoretically-relevant voluntary hyperventilation challenge as a method of direct arousal induction. As individuals may respond differently to hyperventilation in various response domains, the Hyperventilation Questionnaire (Rapee & Medoro, 1994; see Appendix G) which assesses affective, cognitive, and somatic responses to hyperventilation, was chosen as the dependent measure.

This study tested three major hypotheses. First, I hypothesized that sober high anxiety sensitive participants (i.e., high anxiety sensitive – placebo) would show greater reactivity to hyperventilation than sober low anxiety sensitive controls, particularly on measures tapping affective and cognitive symptoms in response to the challenge

(Asmundson et al., 1994; Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee & Medoro, 1994). Second, I hypothesized that alcohol would produce stronger reductions relative to placebo in affective and cognitive responses to the challenge among high anxiety sensitive than among low anxiety sensitive individuals (Conrod et al., 1998; Stewart & Pihl, 1994). Third, I hypothesized that alcohol's dampening effects in high anxiety sensitive individuals would be dose dependent. Since high anxiety sensitive individuals frequently drink to the point of intoxication (Stewart, Peterson, et al., 1995; Stewart, Zvolensky, et al., 2001), I expected that alcohol-dampening effects on high anxiety sensitive individuals' reactivity to hyperventilation would follow a linear trend, with increased dampening from increased alcohol dose. These hypotheses would be supported by significant Beverage Condition (linear) x Anxiety Sensitivity Group x Time interactions for the affective and cognitive reactivity measures (Baron & Kenny, 1986; Rogosch, Chassin, & Sher, 1990).

## Method

### Participants

Participants were recruited from the Dalhousie University Department of Psychology subject pool. Willing psychology students completed the Anxiety Sensitivity Index (Reiss et al., 1986; see Appendix A) as a screening measure during class time. Participants were selected according to their Anxiety Sensitivity Index screening scores. Individuals scoring one standard deviation above the Anxiety Sensitivity Index sample mean for their gender served as potential high anxiety sensitive participants and individuals scoring one standard deviation below the Anxiety Sensitivity Index sample mean for their gender served as potential low anxiety sensitive participants ($\underline{M} = 17 \pm 9$

for females, and $\underline{M}$ = 15 ± 8 for males). These overall sample means on the Anxiety Sensitivity Index compare well with published Anxiety Sensitivity Index norms for university students (i.e., $\underline{M}$'s = 20.5 for women and 15.4 for men; Peterson & Reiss, 1992). Additionally, only students of the legal drinking age in the province of Nova Scotia (i.e., 19 years of age or older) were considered for inclusion. Students who met these selection criteria and indicated a willingness to participate were contacted by the experimenter to arrange for testing.

At the time of telephone contact with each potential participant, the experimenter ensured that there were no medical reasons the individual should not consume alcohol, such as concurrent medication use or a medical condition contraindicating alcohol consumption (see Appendix B). Females who indicated that there was a possibility that they were currently pregnant (i.e., that they had engaged in recent sexual activity without the use of contraception), or who were planning to conceive in the near future, were excluded. Students who indicated current use of any prescribed or over-the-counter medications which could adversely interact with alcohol (e.g., amphetamines, antibiotics) were also excluded. As well, individuals who responded affirmatively that they had a medical condition which contraindicated alcohol consumption and/or participation in the voluntary hyperventilation task (e.g., pulmonary disease, cardiac disease) were excluded from the study. The above set of exclusionary drugs and medical conditions were derived from those listed in the Encyclopedia of Alcoholism (O'Brien & Chafetz, 1982) and those used in a breathing challenge experiment by Kushner et al. (1996), and were verified with a medical expert. Only students who were "regular social drinkers" (i.e., reports of having consumed at least one alcoholic beverage within the last month on a

self-report measure) were included to reduce likelihood of severe nausea to alcohol administration. Students were also administered the Brief Michigan Alcoholism Screening Test (Pokorny et al., 1972; see Appendix C). Individuals scoring greater than 5 (indicating a possible problem drinker) were excluded. On the day of the study, any participants who met the minimum panic frequency and/or minimum symptom criteria for a DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th edition: American Psychiatric Association, 1994) diagnosis of panic disorder, as assessed by the Panic Attack Questionnaire - Revised (Cox, Norton & Swinson, 1992; see Appendix D), were excluded from the eventual analysis.

Four high anxiety sensitive participants originally run in the study were excluded from the final analysis based on DSM-IV (American Psychiatric Association, 1994) criteria for panic disorder as measured by the Panic Attack Questionnaire - Revised (Cox et al., 1992). These criteria included history of spontaneous panic attacks, recurrent attacks, at least one month of significant concerns about the implications of having future panic attacks, and panic attacks characterized by at least four of the symptoms as listed in the DSM-IV (American Psychiatric Association, 1994). They were replaced with four additional high anxiety sensitive participants with no history of panic disorder to create equal N across cells.

The final sample consisted of one hundred and two students who met the selection criteria (51 high anxiety sensitive, 51 low anxiety sensitive). There were seventeen participants in each cell: high anxiety sensitive-high alcohol dose, high anxiety sensitive-low alcohol dose, high anxiety sensitive-placebo, low anxiety sensitive-high alcohol dose, low anxiety sensitive-low alcohol dose, and low anxiety sensitive-placebo.

Although a greater proportion of women than men volunteered for this study (resultant sample = 65.7% women), this gender composition is consistent, and therefore comparable, with previous studies of young adults' responses to hyperventilation challenge (63.8% women (Donnell & McNally, 1989) to 81.0% women (Rapee & Medoro, 1994)). Moreover, the present gender imbalance reflects the gender composition of the population from which participants were drawn, since females are typically over-represented among the enrollment of undergraduate psychology courses (Stewart, Taylor, et al., 1997). Participants were compensated either monetarily, at a rate of five dollars per hour and/or with course credit points, at a rate of one point per hour, at study completion (to a maximum of $20 or 4 credit points). Participants were instructed to abstain from alcohol use for 24 hours, and to refrain from eating for four hours, prior to testing.

Materials

Anxiety Sensitivity Index. The Anxiety Sensitivity Index (Reiss et al., 1986; see Appendix A) is a 16-item questionnaire that measures level of fear of anxiety symptoms. Items express concerns about anticipated negative consequences associated with anxiety-related physical sensations (e.g., "When I notice that my heart is beating rapidly, I worry that I may have a heart attack"). Items are self-rated on a five point Likert scale, with anchors of 0 (very little) and 4 (very much), expressing the degree to which the respondent agrees with each item. Total possible scores can range from 0 to 64. The Anxiety Sensitivity Index possesses good internal consistency, good test-retest reliability, and good construct and criterion-related validity (Peterson & Reiss, 1992).

Demographics Questionnaire. An author-compiled demographics questionnaire was used to obtain information on age, gender, typical alcohol consumption levels, and for the women, starting date of last menses and current use of oral contraceptives (see Appendix E). In the assessment of alcohol consumption levels, participants estimated the number of occasions per week on which they normally consumed alcohol (frequency), and the average number of alcoholic beverages they normally consumed per drinking occasion (quantity). Weekly frequency and quantity were multiplied to yield a composite total weekly alcohol consumption measure (Conrod et al., 1998).

State Trait Anxiety Inventory - Trait Subscale. The State Trait Anxiety Inventory - Trait Subscale (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983; see Appendix F) is a measure of trait anxiety (i.e., frequency of anxiety experiences across a variety of stressful situations), consisting of 20 items rated on a four-point Likert scale with anchors of 1 (almost never) and 4 (almost always). The respondent indicates how often he or she generally feels each experience (e.g., "I feel inadequate"). Total possible scores can range from 20 to 80 and are obtained by adding the weighted scores for the 20 items, taking into account that scoring is reversed for nine of the 20 items (e.g., "I feel rested"). The State Trait Anxiety Inventory - Trait Subscale has good internal consistency, adequate test-retest reliability, and good validity as a measure of the trait anxiety construct (Speilberger et al., 1983).

Panic Attack Questionnaire - Revised. The Panic Attack Questionnaire - Revised (Cox et al., 1992; see Appendix D) was used as a measure of panic symptoms. It assesses both spontaneous (i.e., "out of the blue") and cued (i.e., "in specific situations") panic attacks. The Panic Attack Questionnaire - Revised describes a panic attack according to

DSM-IV (American Psychiatric Association, 1994) criteria and asks if the participant has ever experienced such an attack. Information on frequency of panic attacks, number of panic symptoms and their severity, and distress caused by fear of having another attack, are also assessed. The Panic Attack Questionnaire - Revised was used to exclude any subject with a likely history of panic disorder, and to describe the final sample in terms of relevant panic history variables (i.e., percentages with a history of uncued panic attacks, cued panic attacks, or both uncued and cued attacks).

Hyperventilation Questionnaire. The Hyperventilation Questionnaire (Rapee & Medoro, 1994; see Appendix G) was used as the measure of degree of hyperventilation response. The version of the Hyperventilation Questionnaire used in this study consisted of 31 items conceptually divided into three subscales: cognitive (6 items, e.g., feeling of losing control); affective (7 items, e.g., nervousness); and somatic (18 items, e.g., breathlessness). Each symptom item is rated on a four point Likert scale with anchors of 0 (not at all) and 3 (markedly). Subscale scores were obtained by adding the relevant weighted scores, taking into account that scoring for one of the affective items is reversed (i.e., relaxation). Thus, the range of possible values was 0-21 for the affective subscale, 0-18 for the cognitive subscale and 0-54 for the somatic subscale. The three subscales have all been shown to possess good to excellent internal consistency, with alpha coefficients of 0.92 for somatic, 0.88 for affective and 0.80 for cognitive (Rapee & Medoro, 1994). The Hyperventilation Questionnaire has been found to discriminate between high and low anxiety sensitive participants when used as a measure of response to hyperventilation challenge (Rapee & Medoro, 1994). Each participant in the present study completed the

Hyperventilation Questionnaire at three time points: pre-drinking baseline, post-drinking baseline, and immediately following hyperventilation.

Subjective Intoxication. Subjective intoxication was measured using a visual analogue scale (Kushner et al., 1996; see Appendix H). The respondents rated how intoxicated (i.e., "drunk") they felt by placing a mark on a 100 mm line with anchors of 0 (not at all) and 100 (extremely so). Scores were determined by measuring (in mm) the distance from the left anchor of the scale to the participant's mark.

Blood Alcohol Concentration. Blood alcohol concentrations were measured using an Alcosensor III, (Intoximeters Inc.) in mg alcohol per 100 ml blood (O'Brien & Chafetz, 1982).

Procedure

After participants provided their informed consent (see Appendix J), fasting requirements were verified verbally and a baseline blood alcohol concentration was taken to verify abstinence requirements. The participant's body weight was then taken on a digital electronic scale for use in determining alcohol dose. Next, participants were administered the control measures (i.e., demographics questionnaire, State Trait Anxiety Inventory - Trait Subscale, and The Panic Attack Questionnaire - Revised). Participants then completed the pre-drinking baseline hyperventilation response measure (i.e., the Hyperventilation Questionnaire) with instructions to rate how they were presently feeling. Participants in the placebo and low alcohol dose conditions completed the experiment first and were randomly assigned to either of those conditions at that time. Participants in the high alcohol dose group completed the experiment one year later and therefore were not randomly assigned to a Beverage Condition. However, the

experimenter interacting with the high alcohol dose participants (Michelle Skinner) was kept blind to their Beverage Condition assignment until the completion of the study.

Participants in each anxiety sensitive group were thus assigned to one of the three Beverage Conditions to complete six cells of equal N (17 per cell) and were provided with their assigned beverage (i.e., placebo, low dose, or high dose alcohol). Four to five drinks (depending on total volume) were placed in front of the participants, along with a bottle of tonic water and a bottle of 100-proof vodka. Both the participant and the experimenter interacting with the participants were blind to the participants' anxiety sensitivity group status. The low alcohol dose used was 1.90 ml 50% USP units of alcohol per kilogram of body weight for females and 2.38 ml 50% USP units of alcohol per kilogram of body weight for males, mixed 1:4 parts alcohol to tonic water. The high alcohol dose used was 2.28 ml 50% USP units of alcohol per kilogram of body weight for females and 2.73 ml 50% USP units of alcohol per kilogram of body weight for males, mixed 1:4 parts alcohol to tonic water. Pilot testing was conducted with a group of university students to arrive at an age-adjusted version of an original formula by Fisher, Simpson, and Kapur (1987) to target blood alcohol concentrations of 0.05% and 0.08% for the low and high dose groups, respectively. The modified formula included information on gender, body weight, and age (see Appendix I).

All participants were told that they would be receiving an amount of alcohol equivalent to four or five mixed bar drinks, in order to control for alcohol expectancies (Kushner et al., 1996). In order to further enhance the beverage deception in the placebo condition, the placebo drinks, which were matched for volume with the alcohol drinks, contained a small amount of vodka on top to provide smell and taste cues of alcohol

(Kushner et al., 1996). The open bottle of vodka on the same table as the drinks served as a visual cue.The beverages were consumed steadily over a 25-30 minute period (25 minutes for four drinks, 30 minutes for five drinks), after which the participant rested for an additional 25-30 minutes to allow for absorption (25 minutes for four drinks to 30 minutes for five drinks). Since a small amount of alcohol was present in the placebo drinks, participants in this group were expected to show slightly elevated blood alcohol concentrations. However, resultant blood alcohol concentrations were not expected to be significantly greater than .000% in the placebo group by the time the hyperventilation stressor was applied (cf., Stewart, Finn, & Pihl, 1992, 1995). Following absorption, the post-drinking baseline measure of hyperventilation symptoms was completed (i.e., Hyperventilation Questionnaire), again with participants receiving instructions to rate how they were presently feeling. Level of intoxication was then measured using a blood alcohol concentration test and the visual analogue scale. Next, participants were provided with the following instructions regarding the hyperventilation procedure:

> "You are about to take part in an experiment in which responses to a breathing task are being studied. For the next three minutes, you will listen to a set of instructions. Please follow the instructions as closely as you can. Though you may feel like stopping at some point in the procedure, please try not to." (Donnell & McNally, 1989, p. 332)

Participants then listened to a set of taped instructions which paced their breathing at a rate of 30 breaths per minute for three minutes, with further instructions to breathe as deeply as they could through their mouths in the time provided, as if they were trying to blow up a balloon (Rapee & Medoro, 1994). The experimenter remained in the room with the participant, but out of the participant's range of vision. Immediately following

the hyperventilation challenge, participants completed the Hyperventilation Questionnaire, rating how they felt during the "paced breathing task". Participants were required to remain in the lab until their blood alcohol concentrations reached half the legal limit (i.e., blood alcohol concentration < 0.04%). Alcohol condition participants were reminded that they should not drive for two hours following the experiment. All subjects were then debriefed, including an explanation to placebo participants as to the nature and necessity of the placebo deception.

## Results

### Anxiety Relevant Measures

Means (and SDs) for the anxiety-related measures (Anxiety Sensitivity Index, State Trait Anxiety Inventory - Trait Subscale, and percentage of different types of panic on the Panic Attack Questionnaire - Revised) are shown in Table 1 as a function of Anxiety Sensitivity Group and Beverage Condition. State Trait Anxiety Inventory - Trait Subscale scores were subjected to a 2 x 3 (Anxiety Sensitivity Group x Beverage Condition) Analysis of Variance (ANOVA). There was a significant main effect of Anxiety Sensitivity Group, with the high anxiety sensitive participants scoring higher in trait anxiety than the low anxiety sensitive participants, $F(1, 96) = 21.34$, $p < .001$ (see Table 1). Panic Attack Questionnaire - Revised scores were subjected to a 2 x 3 (Anxiety Sensitivity Group x Beverage Condition) chi-square analyses. No significant Anxiety Sensitivity Group, Beverage Condition, or interaction effects were observed for the proportion of participants who had experienced only cued panic attacks (i.e., situationally bound or situationally predisposed attacks). Similarly, no significant Anxiety Sensitivity Group, Beverage Condition, or interaction of Anxiety Sensitivity Group and Beverage

Table 1

Means (and SDs) on Control Measures as Functions of Anxiety Sensitivity (AS) Group and Beverage Condition.

| AS Group | Beverage Condition | | | | | |
|---|---|---|---|---|---|---|
| High AS | Placebo (N = 17) | | Low dose (N = 17) | | High dose (N = 17) | |
| Measure | Mean | SD | Mean | SD | Mean | SD |
| ASI | 33.1 | 8.9 | 30.5 | 5.9 | 30.3 | 6.2 |
| STAI-T | 43.2 | 11.2 | 38.4 | 9.1 | 41.5 | 7.6 |
| Panic Attacks (% uncued) | 18.0 | ---- | 0.0 | ---- | 6.0 | ---- |
| Panic Attacks (% cued) | 6.0 | ---- | 6.0 | ---- | 6.0 | ---- |
| Panic Attacks (% both) | 6.0 | ---- | 0.0 | ---- | 6.0 | ---- |
| Age (years) | 20.5 | 2.1 | 21.4 | 2.4 | 21.4 | 4.3 |
| Gender (% female) | 65.0 | ---- | 71.0 | ---- | 53.0 | ---- |
| Drinks per week | 6.2 | 6.9 | 6.3 | 4.0 | 8.2 | 6.4 |
| Days Since Last Menses | 12.8 | 7.3 | 16.7 | 12.6 | 14.9 | 10.9 |
| Oral Contraception (% users) | 45.0 | ---- | 75.0 | ---- | 67.0 | ---- |
| Low AS | Placebo (N = 17) | | Low dose (N = 17) | | High dose (N = 17) | |
| Measure | Mean | SD | Mean | SD | Mean | SD |
| ASI | 5.7 | 2.8 | 6.3 | 2.9 | 5.8 | 2.1 |
| STAI-T | 35.8 | 9.3 | 30.9 | 6.5 | 31.9 | 8.8 |
| Panic Attacks (% uncued) | 0.0 | ---- | 0.0 | ---- | 6.0 | ---- |
| Panic Attacks (% cued) | 0.0 | ---- | 12.0 | ---- | 6.0 | ---- |
| Panic Attacks (% both) | 0.0 | ---- | 0.0 | ---- | 0.0 | ---- |
| Age (years) | 23.7 | 6.7 | 20.2 | 1.3 | 24.0 | 6.7 |
| Gender (% female) | 76.0 | ---- | 76.0 | ---- | 53.0 | ---- |
| Drinks per week | 5.7 | 4.3 | 5.5 | 5.9 | 7.9 | 10.2 |
| Days Since Last Menses | 12.5 | 7.9 | 15.6 | 8.1 | 11.7 | 7.8 |
| Oral Contraception (% users) | 27.0 | ---- | 54.0 | ---- | 56.0 | ---- |

Notes: ASI = Anxiety Sensitivity Index (Reiss et al., 1986); STAI-T = State Trait Anxiety Inventory–Trait subscale (Spielberger et al., 1983); days since last menses and oral contraceptives use are for the women only.

Condition effects were observed for the proportion of participants who had experienced only uncued (i.e., unexpected) panic attacks, or both cued and uncued panic attacks.

Demographic Measures

A series of 2 x 3 (Anxiety Sensitivity Group x Beverage Condition) ANOVAs and chi square analyses (for dichotomous variables) were performed on several demographic variables (see Table 1) to ensure equivalence between groups on control measures. No significant effects were obtained.

Manipulation Check- Intoxication Levels

A 2 x 3 (Anxiety Sensitivity Group x Beverage Condition) ANOVA was performed on the visual analogue scale scores of subjective intoxication. There were no significant effects involving the Anxiety Sensitivity Group factor, but a significant Beverage Condition main effect was revealed, $F$ (2, 96) = 44.07, $p < .0001$. Newman-Keuls post-hoc comparisons showed that both the low dose alcohol ($M$ = 54.50, $SD$ = 20.73), and high dose alcohol ($M$ = 56.77, $SD$ = 20.27), groups rated themselves as being significantly more intoxicated than the placebo group ($M$ = 19.15, $SD$ = 14.57) (both $p <$ .01). However, the high dose alcohol participants did not rate themselves as being significantly more intoxicated than the low dose alcohol group. A $t$-test revealed that the placebo group reported subjective intoxication ratings that were significantly greater than zero, $t$ (33) = 7.66, $p < .0001$ (two-tailed test).

Another 2 x 3 (Anxiety Sensitivity Group x Beverage Condition) ANOVA was conducted on the blood alcohol concentrations collected just prior to hyperventilation. There was a significant main effect of Beverage Condition, $F$ (2, 96) = 258.00, $p < .0001$. Newman- Keuls comparisons revealed that the high dose alcohol group reached a higher

mean blood alcohol concentration (M = .086%, SD = .019%) than the low dose alcohol (M = .062%, SD = .022%) and placebo (M = .001%, SD = .003%) groups (both $p < .01$). The low dose alcohol group also reached a significantly higher mean blood alcohol concentration than the placebo group, $p < .01$. Unexpectedly, there was also a significant main effect of Anxiety Sensitivity Group, $F(1, 96) = 10.26$, $p < .005$, with high anxiety sensitive subjects reaching a higher overall mean blood alcohol concentration (M = .054%, SD = .042%) than low anxiety sensitive subjects (M = .044%, SD = .038%).

Analysis of Hyperventilation Questionnaire Baseline Scores

Hyperventilation Questionnaire pre- and post-drinking baseline scores are illustrated in Table 2, as a function of Anxiety Sensitivity Group and Beverage Condition. These baseline subscale scores were subjected to a set of 2 x 3 x 2 (Anxiety Sensitivity Group x Beverage Condition x Time) repeated measures ANOVAs with Time (pre- and post-drinking baseline) as the repeated measure.

For the affective subscale, there was a significant main effect of Anxiety Sensitivity Group, $F(1, 96) = 16.56$, $p < .001$ and a significant main effect of Time, $F(1, 96) = 24.62$, $p < .001$, qualified by a significant Anxiety Sensitivity Group x Time interaction, $F(1, 96) = 5.15$, $p < .05$. I examined the simple effects of Time within each Anxiety Sensitivity Group. The tendency for affective scores to decrease from pre- to post-drinking baseline was somewhat stronger among the high anxiety sensitive participants, $F(1, 50) = 15.18$, $p < .001$ ($\eta^2 = 0.23$), compared to the low anxiety sensitive participants, $F(1, 50) = 10.45$, $p < .005$ ($\eta^2 = 0.17$) (see Table 2).

For the cognitive subscale, there was also a significant main effect of Anxiety

Table 2

Means (and SDs) for the Hyperventilation Questionnaire (HVQ; (Rapee & Medoro, 1994)) Sub-Scale Scores as Functions of Anxiety Sensitivity (AS) Group, Beverage Condition, and Time (Pre-Drinking Baseline and Post-Drinking Baseline).

| HVQ Subscale | Time | | | |
|---|---|---|---|---|
| a) HVQ-Affective | Pre-Drinking Baseline | | Post-Drinking Baseline | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 4.71 (4.92) | 1.76 (1.03) | 1.65 (1.66) | 1.29 (0.69) |
| Low alcohol | 2.65 (1.41) | 2.24 (1.92) | 1.35 (1.69) | 0.88 (0.93) |
| High alcohol | 3.00 (1.94) | 1.00 (0.94) | 1.82 (1.88) | 0.76 (0.83) |
| b) HVQ-Cognitive | Pre-Drinking Baseline | | Post-Drinking Baseline | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 1.00 (2.26) | 0.29 (0.77) | 0.59 (1.06) | 0.06 (0.24) |
| Low alcohol | 0.35 (0.86) | 0.00 (0.00) | 1.29 (1.36) | 0.88 (0.78) |
| High alcohol | 0.18 (0.53) | 0.24 (0.56) | 1.06 (1.14) | 0.53 (1.01) |
| c) HVQ-Somatic | Pre-Drinking Baseline | | Post-Drinking Baseline | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 7.65 (9.04) | 2.24 (3.35) | 7.65 (6.08) | 2.82 (4.14) |
| Low alcohol | 2.65 (2.55) | 2.41 (1.94) | 12.47 (7.32) | 10.29 (5.34) |
| High alcohol | 4.94 (3.56) | 1.53 (2.29) | 12.76 (6.93) | 8.47 (7.42) |

Note: Values represent raw score subscale means (possible range (affective) = 0-21; possible range (cognitive) = 0-18; possible range (somatic) = 0-54 )

Sensitivity Group, $F$ (1, 96) = 5.98, $p < .05$, owing to higher scores in the high anxiety sensitive group compared to the low anxiety sensitive group (see Table 2). In addition, there was a significant main effect of Time, $F$ (1, 96) = 10.82, $p < .005$ qualified by a significant Beverage Condition x Time interaction, $F$ (2, 96) = 9.62, $p < .001$. Although there was no simple main effect of Beverage Condition for the cognitive subscale at the pre-drinking baseline, a significant simple effect of Beverage Condition was revealed at the post-drinking baseline, $F$ (2, 99) = 4.92, $p < .01$. Newman-Keuls comparisons revealed that post-drinking baseline ratings were higher amongst both those receiving the low dose of alcohol ($p < .05$) and those receiving the high dose ($p < .06$) relative to those receiving placebo.

For the somatic subscale, there was also a significant main effect of Anxiety Sensitivity Group, $F$ (1, 96) = 13.80, $p < .001$, owing to higher scores in the high anxiety sensitive group compared to the low anxiety sensitive group (see Table 2). In addition, there was a significant main effect of Time, $F$ (1, 96) = 88.32, $p < .001$, qualified by a significant Beverage Condition x Time interaction, $F$ (2, 96) = 20.31, $p < .001$. Although there was no simple main effect of Beverage Condition for the somatic subscale at the pre-drinking baseline, a significant simple effect of Beverage Condition was revealed at the post-drinking baseline, $F$ (2, 99) = 8.95, $p < .001$. Newman-Keuls comparisons revealed that post-drinking baseline ratings were higher amongst both those receiving the low dose ($p < .05$), and those receiving the high dose ($p < .05$), relative to those receiving placebo.

Reactivity to Hyperventilation Measured on Hyperventilation Questionnaire

Post-drinking Hyperventilation Questionnaire scores were selected as the most

appropriate baseline for examining responses to hyperventilation since there were significant Anxiety Sensitivity Group and Beverage Condition effects at the post-drinking Hyperventilation Questionnaire baseline, but only Anxiety Sensitivity Group effects at the pre-drinking baseline. Therefore, post-drinking Hyperventilation Questionnaire scores were selected as the most appropriate baseline for examining responses to hyperventilation because they were the only baseline that could account for pre-existing Anxiety Sensitivity Group *and* Beverage Condition effects evident prior to hyperventilation.

Reactivity to hyperventilation on the Hyperventilation Questionnaire subscales was examined with relation to the initial hypotheses through a set of 2 x 3 x 2 (Anxiety Sensitivity Group x Beverage Condition x Time) repeated measures ANOVAs with Time (post-drinking baseline and hyperventilation) serving as the repeated measure. Due to hypothesized linear effects of dose, all dose effects were decomposed into their linear and quadratic components for analysis. Linear and quadratic coefficients were calculated for 3-way polynomial interactions with unequally spaced levels of Beverage Condition using techniques outlined by Keppel (1982). Weights for the three levels of the Beverage Condition factor were determined by using the mean blood alcohol concentration in each condition (Placebo = 0.001; Low Dose = 0.062; High Dose = 0.086). The resulting coefficients were as follows: Placebo (linear) = -.0489; Low Dose (linear) = 0.0128; High Dose (linear) = 0.0361; Placebo (quadratic) = 0.0002; Low Dose (quadratic) = -0.0009; High Dose (quadratic) = 0.0007. Three-way polynomial interactions for a repeated measures design were calculated and evaluated according to Winer's (1971) procedures.

Hyperventilation Questionnaire scores are displayed in Table 3 as a functions of Anxiety Sensitivity Group, Beverage Condition, and Time.

For the affective subscale, there were significant main effects of Anxiety Sensitivity Group, $F$ (1,96) = 11.89, $p < .001$, Time, $F$ (1,96) = 60.43, $p < .001$, and Beverage Condition (linear), $F$ (1,96) = 7.68, $p < .01$. A significant Anxiety Sensitivity Group x Time interaction was also revealed, $F$ (1,96) = 5.19, $p < .05$, as well as a significant Beverage Condition (linear) x Time interaction, $F$ (1,96) = 7.80, $p < .01$. Consistent with my primary hypotheses, these lower-order effects were qualified by a significant Anxiety Sensitivity Group x Beverage Condition (linear) x Time interaction, $F$ (1,96) = 4.93, $p < .05$. Further testing of this three-way interaction revealed a simple Anxiety Sensitivity Group x Time interaction effect in the placebo Beverage Condition, $F$ (1,32) = 6.60, $p < .05$, but not in the low or high dose alcohol conditions. Further testing of this significant simple interaction among those administered placebo revealed a significant simple effect of Anxiety Sensitivity Group at hyperventilation, $F(1,32) = 9.37$, $p<.005$, but not at post-drinking baseline (see Table 3). Thus, consistent with Hypothesis 1, high anxiety sensitive participants were more affectively reactive to challenge than low anxiety sensitive participants, but only when they were sober (i.e., administered placebo). Consistent with the alcohol dampening and dose effect hypotheses (Hypotheses 2 and 3), further analysis revealed a significant simple linear effect of Beverage Condition among the high anxiety sensitive participants at hyperventilation, $F$ (1,32) = 24.31, $p < .001$, with affective responses becoming increasingly dampened as alcohol dose increased (see Table 3). No significant linear effect of Beverage Condition was noted for high anxiety sensitive participants at post-drinking baseline, and no significant linear effect of

Table 3

Means (and SDs) for the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) Sub-Scale Scores as Functions of Anxiety Sensitivity (AS) Group, Beverage Condition, and Time (Post-Drinking Baseline and Hyperventilation).

| HVQ Subscale | Time | | | |
|---|---|---|---|---|
| a) HVQ-Affective | Post-Drinking Baseline | | Hyperventilation | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 1.65 (1.66) | 1.29 (0.69) | 6.82 (4.61) | 3.06 (2.11) |
| Low alcohol | 1.35 (1.69) | 0.88 (0.93) | 4.41 (4.27) | 3.29 (3.12) |
| High alcohol | 1.82 (1.88) | 0.76 (0.83) | 3.06 (3.17) | 1.76 (1.30) |
| b) HVQ-Cognitive | Post-Drinking Baseline | | Hyperventilation | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 0.59 (1.06) | 0.06 (0.24) | 4.18 (4.14) | 1.00 (1.37) |
| Low alcohol | 1.29 (1.36) | 0.88 (0.78) | 2.76 (3.25) | 1.71 (1.90) |
| High alcohol | 1.06 (1.14) | 0.53 (1.01) | 2.00 (2.69) | 0.53 (0.72) |
| c) HVQ-Somatic | Post-Drinking Baseline | | Hyperventilation | |
| Beverage Condition | High AS | Low AS | High AS | Low AS |
| Placebo | 7.65 (6.08) | 2.82 (4.14) | 20.35 (13.87) | 10.41 (8.19) |
| Low alcohol | 12.47 (7.32) | 10.29 (5.34) | 18.82 (8.51) | 17.24 (8.68) |
| High alcohol | 12.76 (6.93) | 8.47 (7.42) | 16.65 (9.92) | 11.53 (8.85) |

Note: Values represent raw score subscale means (possible range (affective) = 0-21; possible range (cognitive) = 0-18; possible range (somatic) = 0-54 )

Beverage Condition was noted for low anxiety sensitive participants at either hyperventilation or post drinking baseline.

For the cognitive subscale, there were significant main effects of Anxiety Sensitivity Group, $F$ (1,96) = 16.08, $p < .001$, and Time, $F$ (1,96) = 26.00, $p < .001$. A significant Anxiety Sensitivity Group x Time interaction, $F(1,96) = 7.74$, $p<.01$, and a significant Beverage Condition (linear) x Time interaction, $F(1,96) = 8.34$, $p<.01$, were also revealed. Consistent with my hypotheses, these lower-order effects were qualified by a marginally significant Anxiety Sensitivity Group x Beverage Condition (linear) x Time interaction, $F$ (1, 96) = 2.61, $p < .10$. Given that this interaction was predicted a priori, and that the pattern of means closely resembled the findings with the affective subscale, I proceeded with further breakdown of this three-way interaction for the cognitive subscale (see recommendations by Winer, 1971, regarding the relaxation of alpha for interactions that are predicted a priori). A simple interaction effect of Anxiety Sensitivity Group x Time was found in the placebo Beverage Condition, $F$ (1, 32) = 7.77, $p < .01$, but not in the low or high dose alcohol conditions. Further testing of this significant simple interaction among those administered placebo revealed a significant simple effect of Anxiety Sensitivity Group at hyperventilation, $F$ (1, 32) = 9.01, $p < .01$, but not at post-drinking baseline (see Table 3). Consistent with Hypothesis 1, this pattern suggests greater cognitive reactivity to hyperventilation in high anxiety sensitive participants compared to low anxiety sensitive participants, but only when they were sober (i.e., administered placebo). Consistent with the alcohol dampening and dose effect hypotheses (Hypotheses 2 and 3), further analysis revealed a significant simple linear effect of Beverage Condition among the high anxiety sensitive participants at

hyperventilation, $\underline{F}$ (1, 32) = 4.07, $\underline{p} < .05$, with cognitive responses becoming increasingly dampened as alcohol dose increased (see Table 3). No significant linear effect of Beverage Condition was noted for high anxiety sensitive participants at post-drinking baseline, and no significant linear effect of Beverage Condition was noted for low anxiety sensitive participants at either hyperventilation or post-drinking baseline.

For the somatic subscale, there was a significant main effect of Anxiety Sensitivity Group, $\underline{F}(1,96) = 9.91$, $\underline{p}<.01$, with high anxiety sensitive participants showing overall elevations on this subscale compared to low anxiety sensitive participants (see Table 3). There was also a significant main effect of Time, $\underline{F}(1,96) = 90.90$, $\underline{p}<.001$, qualified by a significant Beverage Condition (linear) x Time interaction, $\underline{F}(1,96) = 14.06$, $\underline{p}<.001$. Simple effects of Time were examined at each level of the Beverage Condition factor. Although higher scores were seen at hyperventilation than at the post-drinking baseline in all three Beverage Conditions (see Table 3), the magnitude of these effects appeared smaller for the high dose of alcohol, $\underline{F}(1,33) = 9.43$, $\underline{p}<.005$ ($\eta^2 = .22$), than for either the placebo, $\underline{F}(1,33) = 47.07$, $\underline{p}<.001$ ($\eta^2 = .59$) or low dose of alcohol, $\underline{F}(1,33) = 39.35$, $\underline{p}<.001$ ($\eta^2 = .54$). Thus, the administration of alcohol (particularly the high dose of alcohol) appears to have dampened participants' overall somatic reactivity to the hyperventilation challenge. No significant Anxiety Sensitivity Group x Beverage Condition (linear) x Time interaction was revealed for the somatic subscale.

## Discussion

Consistent with my first hypothesis, high anxiety sensitive - placebo participants reported greater affective and cognitive sensations than the low anxiety sensitive -

placebo participants during hyperventilation. This finding supports previous research demonstrating increased emotional and psychological reactions to hyperventilation among sober high versus low anxiety sensitive participants (Asmundson et al., 1994; Donnell & McNally, 1989; Rapee & Medoro, 1994). The absence of sober Anxiety Sensitivity Group differences on the somatic Hyperventilation Questionnaire subscale suggests that the high and low anxiety sensitive placebo individuals experienced the same degree of physical symptoms in response to hyperventilation, but interpreted the arousal differently (i.e., high anxiety sensitive - placebo individuals may have interpreted the arousal as more dangerous or "catastrophic" and thus experienced more anxiety; Stewart & Pihl, 1994; Rapee & Medoro, 1994; Shostak & Peterson, 1990). This finding replicates the work of Rapee and Medoro (1994) and provides evidence for the notion of anxiety sensitivity as an "anxiety amplifying" factor (Reiss, 1991), at least in the sober state.

The finding that both high and low anxiety sensitive subjects experienced increased cognitive (e.g., "feel like passing out") and somatic (e.g. "dizziness") symptoms at the post-drinking baseline compared to the pre-drinking baseline is consistent with previous research that alcohol can increase these kinds of symptoms in the resting state (Maisto, Connors, Tucker & McCollam, 1980). Interestingly, affective reactivity scores decreased more strongly from pre- to post-drinking baseline among high anxiety sensitive participants compared to the low anxiety sensitive participants across Beverage Conditions. This finding may suggest that the actual or perceived consumption of alcohol exerts a dampening effect on anticipatory anxiety which is greater in magnitude for high anxiety sensitive individuals than for low anxiety sensitive

individuals. However, this explanation must be qualified by the finding of overall higher affective reactivity among high anxiety sensitive compared to low anxiety sensitive participants across Time periods.

Regardless of Anxiety Sensitivity Group status, participants administered alcohol reported less somatic reactivity to the hyperventilation challenge than placebo participants, suggesting that alcohol exerted some overall physiological stress-response dampening effects (Sher, 1987). However, alcohol dampening effects specific to the high anxiety sensitive group were seen on the affective and cognitive Hyperventilation Questionnaire subscales (cf. Stewart & Pihl, 1994), consistent with my second hypothesis that high anxiety sensitive individuals would be particularly susceptible to alcohol's dampening of affective and cognitive components of the fear response.

The present findings support the notion that high anxiety sensitive individuals display greater sensitivity to alcohol-induced reductions in affective reactivity to arousal-induction than low anxiety sensitive individuals. The present findings are also consistent with the idea that high anxiety sensitive individuals are more sensitive to alcohol-induced reductions in cognitive reactivity compared to low anxiety sensitive individuals. However, support for the sensitivity to cognitive reactivity reduction hypothesis was less definitive in that the predicted Anxiety Sensitivity Group x Beverage Condition (linear) x Time interaction was only marginally significant. Nonetheless, given Winer's (1971) recommendations about relaxing alpha in cases where interactions are predicted a priori, and given that all simple effects were significant, cognitive reactivity dampening does appear to be present to some degree for high anxiety sensitive participants in the current study.

Linear trend analyses supported my third hypothesis, that there would be dose-dependent alcohol effects on all reactivity measures among high anxiety sensitive participants. The magnitude of alcohol dampening effects increased as a function of increasing alcohol dose among high anxiety sensitive participants. These observed dose effects may be important in explaining high anxiety sensitive individuals' tendency to frequently drink to the point of legal intoxication (Stewart, Peterson, et al., 1995; Stewart, Zvolensky, et al., 2001). High anxiety sensitive people appear to require relatively high doses of alcohol (i.e., doses in the legally intoxicating range) to experience large magnitude dampening effects.

Subjective intoxication ratings suggested that placebo participants did believe they were intoxicated, indicating a successful expectancy manipulation (cf. Kushner et al., 1996). Also, despite clear dose effects on the dependent Hyperventilation Questionnaire reactivity variables and on blood alcohol concentrations, subjective intoxication ratings did not distinguish high and low dose alcohol groups. Therefore, the alcohol effects noted in the current study appear more likely to be accounted for by pharmacological alcohol effects than by expectancy factors. However, participants in both alcohol groups rated themselves as feeling more intoxicated than placebo participants, suggesting that either pharmacological factors, or expectancy factors, or both could have been operative. Replication of the current study, including groups of high and low anxiety sensitive participants that expect to receive no alcohol and actually receive no alcohol (e.g., a tonic only control group; Newlin, 1989), is required to more definitively evaluate the contributions of expectancy effects, per se.

The current study found no Anxiety Sensitivity Group difference on a self-report measure of number of alcoholic beverages normally consumed per week, thus failing to replicate previous results (Stewart, Peterson, et al., 1995). However, participants in the present alcohol challenge study were selected for at least a moderate drinking history in order to ensure at least some minimal tolerance to alcohol. The average drinks per week reported by the high anxiety sensitive participants in the present study (M (SD) = 6.9 (5.9)) appears comparable to that reported by the high anxiety sensitive participants in the earlier study (M (SD) = 7.4 (6.4)). However, the mean drinks per week of the present low anxiety sensitive participants (M (SD) = 6.4 (7.2)) appears higher than that of the low anxiety sensitive participants in the earlier study (M (SD) = 2.2 (1.7)). This suggests that I may have selected a low anxiety sensitive group that drank more than would be typical for a group of low anxiety sensitive individuals. However, the fact that the two Anxiety Sensitivity Groups in the present study did not differ significantly in self-reported weekly alcohol consumption levels suggests likely equivalence between groups in alcohol tolerance.

One potential limitation to the current study is that the high and low anxiety sensitive groups differed not only in Anxiety Sensitivity Index (Reiss et al., 1986) scores but also in trait anxiety, either of which could theoretically be responsible for the high anxiety sensitive - placebo group's increased responses to hyperventilation challenge and receptivity to stress response dampening effects of alcohol (McNally, 1996; Taylor, 1996; Borden & Lister, 1994; Lilienfeld, Jacob & Turner, 1989). However, previous studies have demonstrated that it is Anxiety Sensitivity Index scores and not trait anxiety that predicts sober reactivity to hyperventilation (Asmundson et al., 1994; Donnell &

McNally, 1989; Rapee & Medoro, 1994), and Sher (1987) notes that trait anxiety does not appear to contribute to stress response dampening effects. Nonetheless, future studies should address the contributing roles of anxiety sensitivity levels as compared to trait anxiety levels in accounting for degree of sensitivity to alcohol's affective and cognitive reactivity-dampening effects in response to arousal-induction challenges.

Another possible limitation of the current study pertains to the finding that high anxiety sensitive individuals had higher blood alcohol concentration levels than their low anxiety sensitive counterparts across alcohol conditions, despite identical alcohol dose calculations and drink mixing procedures for each group. Thus, it is possible that the differences in alcohol dampening effects across the two groups may be partly attributable to differences in blood alcohol concentration levels. However, the magnitude of this difference, while statistically significant, is fairly small in absolute terms and is unlikely to entirely explain the Anxiety Sensitivity Group differences observed. Moreover, no such Anxiety Sensitivity Group differences were observed in subjective intoxication – further evidence that the observed blood alcohol concentration differences are likely not meaningful.

In summary, the present findings confirm past reports that sober high anxiety sensitive individuals display increased emotional responses to the experience of arousal-related bodily sensations. In addition, the present study suggests increased sensitivity to alcohol's dampening of fear and negative interpretation of bodily arousal symptoms among high anxiety sensitive individuals. Their increased sensitivity to these negatively-reinforcing effects may serve to promote increased alcohol consumption, and may eventually lead to alcohol abuse/dependence as high anxiety sensitive individuals

learn to use alcohol more and more for these rewarding consequences. Consistent with this idea, the present study reveals a linear effect of alcohol dose on reactivity dampening among high anxiety sensitive participants. Thus, it is possible that this population has a tendency to frequently drink to the point of legal intoxication so that they may experience larger dampening effects. Future research on the mechanisms by which alcohol exerts its fear-dampening effects among high anxiety sensitive individuals could be useful in designing targets for alcohol abuse prevention and treatment approaches for this group, as outlined in our review (Stewart et al., 1999). A fuller discussion of possible treatment approaches arising from the current results is presented in Chapter Five.

# CHAPTER THREE

## Alcohol Outcome Expectancies

A number of researchers have suggested an information processing perspective as a means of conceptualizing learned behaviour (Goldman, Darkes, & Del Boca, 1999; Rescorla, 1988; Sayette, 1999). In this model, learned behaviour is represented by templates stored in memory. These templates, also known as expectancies, are built up through an acquisition of information from environmental stimuli, and anticipate associations between behaviour and its consequences. The resulting templates or expectancies can then influence subsequent behavioural patterns. Alcohol outcome expectancies are formed when biological, psychological, and environmental information about alcohol's effects contributes to memory templates that influence alcohol-related behavioural effects and consumption (Goldman, Del Boca, & Darkes, 1999, Vogel-Sprott & Fillmore, 1999). These expectancies are thought to be stored in a semantic memory network where they can be activated automatically following presentation of alcohol cues (Goldman, Del Boca, & Darkes, 1999; Stacy, Leigh & Weingardt, 1994). In simpler terms, alcohol outcome expectancies reflect beliefs about the consequences of drinking alcohol (Goldman et al., 1987). It appears that alcohol outcome expectancies are formed very early in life, and may be acquired vicariously prior to actual alcohol consumption (see Dunn & Goldman, 1996). Reliable sets of positive alcohol expectancies have been revealed through factor analysis. These include expectancies that alcohol consumption will contribute to: global positive changes; sexual enhancement; social and physical pleasure; social assertion; arousal/aggression; and (most importantly for the present

thesis) relaxation and tension-reduction (Brown, Christiansen & Goldman, 1987; Goldman, Greenbaum & Darkes, 1997; MacDonald & Stewart, 1999).

## Alcohol Outcome Expectancies and Drinking Behaviour

### Alcohol Usage Patterns

There is increasing evidence that alcohol outcome expectancies have significant effects on alcohol consumption patterns and the behavioural consequences of drinking (see reviews by Goldman et al., 1987; Goldman, Darkes & Del Boca, 1999). Positive alcohol expectancies have proven to be powerful predictors of actual drinking behaviour. In general, expectancies of favorable outcomes of drinking alcohol have been associated with greater alcohol use (see Brown, 1993). In fact, there is accumulating evidence that expectancies are among the strongest and most robust predictors of drinking patterns (Goldman, Darkes, & Del Boca, 1999; Goldman, Del Boca, & Darkes). For example, in a study with college students, positive expectancy variables proved to be stronger predictors of drinking patterns than relevant background/demographic factors (Brown, 1985a). Positive alcohol outcome expectancies have also been shown to be more powerful motivators for drinking than negative expectancies or beliefs regarding the aversive outcomes of drinking (Leigh & Stacy, 1993). In addition, positive alcohol outcome expectancies have been shown to predict undergraduates' intentions of drinking excessively and actual excessive consumption, over and above attitudes that these individuals may have had about alcohol use (Wall, Hinson, & McKee, 1998). Longitudinal studies have provided evidence for the idea that expectancies may have a causal influence on drinking onset and consumption among adolescents (Christiansen, Smith, Roehling, & Goldman, 1989; Stacy, Newcomb, & Bentler, 1991). In line with this

idea, research with heavy drinkers has shown that educational programs designed to reduce positive expectations about alcohol use lead to a corresponding reduction in alcohol consumption (e.g., Darkes & Goldman, 1993). Similar findings have been demonstrated in college student binge drinkers (Borsari & Carey, 2000), college student social drinkers (Jones, Silvia, & Richman, 1995), and outpatient alcoholics (Connors, Tarbox & Faillace, 1994). However, the efficacy of challenging expectancies as a means of reducing alcohol consumption has recently been disputed (see review by Jones, Corbin, & Fromme, 2001). Del Boca and Darkes (2001) have responded to these criticisms by highlighting the fact that alcohol expectancies appear to have a clear causal impact on alcohol consumption, even if modifying those expectancies does not necessarily constitute an effective treatment for alcohol problems.

Drinking Problems

In addition to predicting drinking onset, frequency and consumption levels, positive alcohol outcome expectancies have also been linked to problem drinking status (see reviews by Goldman, Darkes, & Del Boca, 1999; and Vogel-Sprott & Fillmore, 1999). Positive alcohol expectancies in general have been shown to be elevated among heavy drinkers across a variety of populations, including alcoholics, medical patients, and college students (Brown, Goldman, & Christiansen, 1985). In particular, the expectancy of relaxation and tension-reduction has been noted as one of the most powerful predictors of drinking problems (Brown, 1985a) among samples of college students. Similarly, tension-reduction expectancies have been shown to predict risky drinking patterns in both men and women across late adolescence (Mann, Chassin, & Sher, 1987; Rutledge & Sher, 2001) and early young adulthood (Rutledge & Sher, 2001). In a sample of

undergraduate social drinkers, Hittner (1995) demonstrated that high frequency drinkers endorsed significantly greater tension-reduction expectancies than low frequency drinkers regardless of the participants' preferred beverage type. In addition, Brown (1985b) found that strong expectations for relaxation and tension-reduction from drinking predicted alcoholism treatment failures at one-year follow-up better than traditional variables such as low levels of social support and high stress. The relationship between positive alcohol expectancies and alcohol problems has been seen as early as adolescence (Laurent, Catanzaro, & Callan, 1997). Thus, positive alcohol expectancies in general, and expectations for tension-reduction in particular, appear to be important factors in predicting drinking levels, risk for alcohol abuse problems, and relative recovery from alcohol disorders (see reviews by Goldman, Darkes, & Del Boca, 1999; and Goldman, Del Boca, & Darkes, 1999).

Tension-Reduction Expectancies and the Anxiety-Drinking Relationship

There is growing evidence that tension-reduction expectancies may moderate the relationship between anxiety and drinking behaviour. One study showed a stronger positive association between anxiety and alcohol-use patterns in males with high vs. low tension-reduction expectancies (Kushner, Sher, Wood, & Wood, 1994). Following up on this work, this same set of researchers noted that the strength of tension-reduction expectancies predicted both drinking frequency (at least in men) and self-reported reasons for drinking (i.e., drinking for the purpose of relieving tension) (Kushner, Thuras, et al., 2000). Goldman, Del Boca, and Darkes (1999) suggested that while positive alcohol outcome expectancies, such as tension-reduction, have been shown to moderate the relationship between certain antecedent risk variables (e.g., anxious personality) and

drinking, these expectancies may also act as partial mediators of the relationship between anxiety and alcohol use. Goldman and his colleagues (Goldman, Del Boca, & Darkes, 1999) argue that expectancies can be seen as at least one causal pathway to drinking and alcoholism. In support of this point, they review a number of studies which support a mediational view, including correlational studies, studies which show acquired expectancies in children before drinking experience, prospective studies, drinking experience/expectancy acquisition, statistical support for mediation, and true experiments (see Goldman, Del Boca, & Darkes, 1999).

Anxiety Sensitivity, Tension-Reduction Expectancies and Drinking Behaviour

Theoretical Links between Anxiety Sensitivity and Tension-Reduction Expectancies

There are some compelling theoretical reasons to believe that tension-reduction alcohol expectancies may be elevated in high anxiety sensitive individuals. Study One provides evidence that high anxiety sensitive young adults show increased sensitivity to alcohol's emotional reactivity-dampening effects, replicating similar findings by other researchers in the field (e.g., Conrod et al., 1998; Stewart & Pihl, 1994). Given the greater relief that they may receive from alcohol, it is possible that high anxiety sensitive young adults may develop stronger tension-reduction alcohol outcome expectancies than their low anxiety sensitive counterparts. Since alcohol expectancies can theoretically be modified by actual experiences with alcohol (Darkes & Goldman, 1993; Goldman, Del Boca, & Darkes, 1999), it stands to reason that high anxiety sensitive young adults may develop heightened expectancies about the relaxing and tension-reducing effects of alcohol as they increase their drinking frequency in young adulthood (see reviews by Goldman, Darkes, & Del Boca, 1999; and Goldman, Del Boca, & Darkes, 1999).

Although alcohol expectancies and alcohol motives are considered to be distinct constructs (Mann et al., 1987), Goldman, Del Boca, and Darkes (1999) have suggested that self-report items which measure individual expectancies (e.g., items on the Alcohol Expectancy Questionnaire: Brown et al., 1987) may overlap with measures which tap reasons and motives for drinking (e.g., Drinking Motives Questionnaire; Cooper, Russell, Skinner, & Windle, 1992). Self-report items which measure drinking motives ask respondents why, how often, or how much they drink for various reasons, while the items for expectancies ask what they believe will happen when they drink. Goldman, Del Boca, and Darkes (1999) implied that the wording for both measures may differ, but the items may, in fact, tap into the same information set. Considering the increasing evidence that high anxiety sensitive individuals tend to drink for coping-related reasons (i.e., drinking to deal with negative affect) (Stewart, Karp, et al., 1997; Stewart & Zeitlin, 1995; Stewart, Zvolensky, et al., 2001), it is possible that these reasons may overlap with alcohol outcome expectancies for tension-reduction (i.e., drinking to feel more relaxed) or global positive changes. In partial support of this idea, O'Hare (1998) demonstrated that young adults who report high tension-reduction expectancies tend to drink more in situations in which alcohol use represents a negatively-reinforcing coping response.

Experimental Links between Anxiety Sensitivity and Tension-Reduction Expectancies

Clinical Populations Characterized by High Anxiety Sensitivity. Consistent with the theoretical notion presented above, there is growing experimental evidence showing that positive alcohol outcome expectancies in general, and tension reduction expectancies in particular, may be elevated and predictive of drinking patterns in high anxiety sensitive individuals. Kushner, Abrams, Thuras, and Hanson (2000) observed that the drinking

behaviour of panic disorder patients with no history of alcoholism was strongly predicted by tension-reduction expectancies. These expectancies emerged as more important predictors of alcohol consumption than anxiety-related personality traits such as trait anxiety. Not surprisingly, individuals with panic disorder are characterized by high levels of anxiety sensitivity (Stewart, Knize, et al., 1992). Similarly, O'Hare (1990) found that social anxiety levels significantly predicted tension-reduction alcohol expectancies in a large sample of male and female undergraduate students. These findings were replicated by Ham, Hope, White, and Rivers (2002) who found that outpatient individuals seeking treatment for social anxiety had greater tension reduction and global positive change expectancies than normal controls. Moreover, similar to the findings of Kushner, Abrams, et al. (2000), tension reduction expectancies were predictive of the quantity of alcohol consumed per month among the socially anxious participants (Ham et al., 2002). Given that panic disorder, non-clinical social anxiety, and social phobia are characterized by high anxiety sensitivity levels (Stewart, Knize, et al., 1992; Ball et al., 1995; Cox et al., 1999), high anxiety sensitive individuals may hold higher tension reduction expectancies than others in the general population, and that these expectancies may influence subsequent drinking patterns.

Direct Evidence of Associations Between Anxiety Sensitivity and Tension-Reduction Expectancies. More direct evidence for a link between high anxiety sensitivity levels and high tension-reduction expectancies is slowly starting to emerge. Karp (1993) studied a large sample of subjects diagnosed with alcohol abuse/dependence. Anxiety sensitivity levels were positively correlated with levels of a variety of alcohol expectancies (as assessed by the Alcohol Expectancy Questionnaire; Brown et al., 1987),

and strongly related to tension-reduction and relaxation expectancies in particular. In contrast, tension-reduction expectancies were unrelated to measures of trait anxiety. This suggests that fear of anxiety symptoms, as opposed to frequency of anxiety, best predicts expectations about the tension-reducing effects of alcohol, at least among alcoholics (Karp, 1993). Similarly, the lack of relationship between trait anxiety and expectations for relaxation and tension-reduction has been observed in a large college student sample in a study by Brown and Munson (1987). Preliminary work by McKay (2000) indicates that anxiety sensitivity may also be a significant predictor of tension-reduction expectancies in non-alcoholic, young adults. Using a large sample of undergraduate students, McKay examined correlations between factors on the Anxiety Sensitivity Index (Reiss et al., 1986) and various alcohol outcome expectancies, as measured by the Comprehensive Effects of Alcohol Questionnaire (Fromme, Stroot, & Kaplan, 1993). McKay (2000) found that a primary factor of the Anxiety Sensitivity Index (Reiss et al., 1986), namely, fear of bodily sensations or "physical concerns" (cf., Stewart, Taylor, et al., 1997), was significantly associated with alcohol outcome expectancies for tension-reduction.

Tension-Reduction Alcohol Expectancies and Mediation of the Anxiety Sensitivity-Alcohol Consumption Relationship

Given that tension-reduction expectancies are particularly predictive of problematic drinking patterns in adolescents and young adults (Brown, 1993; Goldman et al., 1987), that alcoholics report more positive alcohol expectancies, including tension-reduction expectancies, than non-alcoholics (Karp, 1993), and given that tension-reduction expectancies appear to be associated with anxiety sensitivity (McKay, 2000), it

is possible that tension-reduction expectancies may mediate (Baron & Kenny, 1986) the previously observed significant relationship between anxiety sensitivity levels and levels of alcohol use (Cox et al., 1993; Stewart, Peterson, et al., 1995; Stewart, Zvolensky, et al., 2001). As noted previously, heightened sensitivity to the arousal-dampening effects of alcohol among high anxiety sensitive individuals (see our review in Stewart et al., 1999) may lead to stronger expectancies of tension-reduction in this group compared to those with lower levels of anxiety sensitivity. Stewart and Kushner (2001) proposed that these expectancies could then motivate increased alcohol consumption. Thus, the existing relationship between anxiety sensitivity and alcohol consumption may be at least partially mediated by greater tension reduction expectancies among highly anxiety sensitive people. Stewart and Kushner (2001) proposed a model which incorporates both alcohol expectancies and anxiety level as mediators of the relationship between anxiety sensitivity and alcohol use. In this model, as in the mediator model discussed in Chapter One, anxiety sensitivity promotes increased anxiety levels. Information stored in memory about the fearfulness of anxiety symptoms is activated by increased state anxiety. This activation then spreads to informational templates (nodes) which are related to feared anxiety sensations, such as expectancies that alcohol reduces tension (Goldman, Del Boca, & Darkes, 1999). Tension-reduction expectancies subsequently become active which, in turn, motivate the use of alcohol for coping purposes. Thus, the relationship between anxiety sensitivity and alcohol use is mediated by elevated state anxiety and activated tension reduction expectancies. Anxiety symptoms can be seen as antecedents which activate nodes about alcohol use and associated desired outcomes (e.g., "if I drink alcohol now, then my anxiety will be relieved").

## Alcohol Expectancies and Stress Response Dampening in High Anxiety Sensitive Individuals

Using expectancies as a framework, Goldman and his colleagues (1987) have provided a cognitive model to explain the stress response dampening (Sher, 1987) properties of alcohol. Simply stated, the model predicts that relief from tension and stress will be influenced by an individual's expectancy that alcohol will lead to tension-reduction. Several studies have demonstrated that the mere expectancy that one has consumed alcohol, without actual alcohol consumption, can lead to subsequent reductions in tension/anxiety (see review by Young, Oei, & Knight., 1990). One of the best-controlled studies in this area was conducted by Wilson and Abrams (1977). They used a between-subjects, balanced placebo procedure (in which alcohol instructions are crossed factorially with beverage type) to separate a sample of male social drinkers into four cells (pharmacology, expectancy, pharmacology and expectancy, and control). A social stressor was then introduced in which participants were asked to make a positive impression on a female confederate. Physiological and self-report measures of anxiety were taken before and after the social interaction. Subjects who expected alcohol exhibited reduced physiological reactivity to the stressor regardless of the beverage they consumed. Interestingly, participants also reported less anxiety in response to the stressor as measured by the State Trait Anxiety Inventory – State subscale (Spielberger et al., 1983) and the Subjective Units of Disturbance Scale (Wolpe, 1973), but only in the expectancy (told alcohol, receive placebo) condition. The other three conditions showed no effect on self-reported anxiety. Based on these results, Wilson and Abrams (1977) suggested that learned expectations about the consequences of drinking alcohol, rather

than alcohol's pharmacological properties, determine alcohol's behavioural effects at low to moderate blood alcohol levels (0-.04%). They further argued that use of a theoretically relevant stressor (social challenge) was important for an ecologically valid examination of alcohol's effects on the anxiety response (Wilson & Abrams, 1977). Interestingly, a similar study with women yielded no effects of expectancy on self-reported anxiety but an increase in physiological arousal in response to the social stressor under the alcohol expectancy condition (Abrams & Wilson, 1979). These results were partially replicated by de Boer, Schippers, and Van der Staak (1993) using a similar paradigm. Unlike Wilson and Abrams (1977), de Boer et al. (1993) found that pure expectancy influences did not reduce self-reported anxiety in men, but did lead to anxiety reductions in women. Purely pharmacological properties of alcohol, as well as pharmacological and expectancy effects working in concert, reduced reports of anxiety in both groups. These results led de Boer et al. (1993) to suggest that cognitive factors like expectancy clearly mediate the effects of alcohol on self-reported anxiety, but that pharmacological factors may be more powerful.

Tension-Reduction Expectancies as Moderators

According to Greeley and Oei (1999), the belief that one has consumed alcohol is not as much a determining factor for tension-reduction as pre-held beliefs about alcohol effects. For example, de Boer, Schippers, and van der Staak (1994) found that women exposed to a social stressor reported reduced subjective feelings of anxiety in an alcohol expectancy condition. Interestingly, this effect only held for women who held prior beliefs that alcohol would have a positive effect on social behaviour. For women who believed that alcohol would lead to adverse effects on social interactions, no expectancy

effect was found (de Boer et al., 1994). Similarly, in another study involving a stressful social interaction, Keane and Lisman (1980) observed that socially anxious males who were led to believe that alcohol would facilitate social interactions reported less subjective tension than participants who were led to believe that alcohol would disrupt social interactions, despite the fact that each group received a placebo beverage. The moderating (Baron & Kenny, 1986) effects of pre-existing beliefs on the expectancy-anxiety reduction relationship have also been partially supported in a study of individuals who held specific beliefs about the tension-reduction effects of drinking alcohol. In a sample of social drinkers with pre-existing tension-reduction expectancies, Young, Knight, and Oei (1988; cited in Young et al., 1990) found that behavioural and self-reported tension decreased in experimental participants who were administered alcohol in a socially stressful situation. While this particular study was limited by the lack of comparison to a control group without pre-existing tension-reduction expectancies, Young et al. (1990) nonetheless have suggested that studies such as these provide an empirical link between tension-reduction expectancies, alcohol consumption, and actual relief from tension. On the basis of their review, they further suggested that the traditional tension reduction hypothesis (Conger, 1956) (a general explanation for drinking behaviour) be restricted to those individuals who hold tension-reduction expectancies.

Stress Response Dampening in High Anxiety Sensitive Individuals

Non-Clinical Young Adults. Given evidence that tension-reduction expectancies may be elevated in highly anxiety sensitive individuals (Karp, 1993; McKay, 2000), the idea that tension-reduction expectancies may directly influence alcohol-induced stress response dampening (Young et al., 1990) suggests that the anxiety relief that high anxiety

sensitive individuals experience from consuming alcohol (see Study One; also see Conrod et al., 1998; and Stewart & Pihl, 1994) could be partly attributable to expectancy factors. Although Stewart and Kushner (2001) propose alcohol expectancy activation as a possible intermediary step on the path to a pharmacologically-induced coping response in high anxiety sensitive individuals, it is also possible that some degree of relief from anxiety may be gained from activation of expectancies alone (Young et al., 1990). Thus, tension-reduction expectancies may mediate the use of alcohol as a coping response in high anxiety sensitive individuals *and* provide a coping mechanism in and of themselves. In support of this idea, a recent study has shown that anxiety sensitivity levels and tension reduction outcome expectancies predict physiological responses to alcohol cues following a hyperventilation arousal-induction challenge (Mulligan & McKay, 2001). Mulligan and McKay (2001) examined a group of non-clinical participants with high levels of anxiety sensitivity. Following a voluntary five-minute hyperventilation challenge, subjects were presented with a visual alcohol cue (a labeled alcoholic beverage). Heart rate decreased faster for participants who had higher levels of anxiety sensitivity and positive alcohol expectancies such as tension reduction. Heart rate decelerations have been shown to reflect heightened attention to alcohol cues (e.g., smell, taste, or sight of stimuli associated with alcohol) and may represent physiological indicators of a craving response for alcohol (see Carter & Tiffany, 1999; Goldman et al., 1987). Applying a modified Stewart and Kushner (2001) model to these results, one may assume that the high anxiety sensitive participants experienced heightened state anxiety as a result of the hyperventilation stressor. Subsequently, tension-reduction expectancies may have been primed by state anxiety and further triggered by the alcohol cue, leading

to reductions in physiological reactivity. Such an effect would likely be rewarding for individuals who fear the somatic sensations associated with anxiety, making future alcohol consumption and alcohol cues negatively reinforcing. Subsequent pharmacological effects of alcohol following alcohol consumption may then operate alone or could interact with expectancies to further dampen negative affect and feelings of anxiety after exposure to a stressor, as seen in Study One of the present thesis.

Panic Disorder Patients. A recent study has demonstrated that pharmacological and expectancy effects of alcohol work together to reduce anxiety in panic disorder patients, a clinical population characterized by high anxiety sensitivity levels. Lehman, Brown, Palfai, and Barlow (2000) measured responses to a $CO_2$ panic challenge in a between subjects design using a group of patients with panic disorder with agoraphobia. Participants in the study were provided with either a placebo or an alcoholic beverage prior to challenge. In addition, individual expectancies of the ability of alcohol to dampen panic sensations were measured. Alcohol was found to dampen participants' self-reported anxiety compared to placebo. In addition, regression analyses revealed that specific positive expectations about the ability of alcohol to reduce panic sensations predicted subsequent self-reported reductions in those sensations regardless of beverage condition. That is, patients who held more positive expectancies that alcohol would act as a panic-reducing agent in fact reported less severe panic sensations in response to the challenge than patients who did not hold such positive expectancies. In line with Goldman, Darkes, and Del Boca (1999) and Young et al. (1990), Lehman et al. (2000) used these results to suggest that pre-existing alcohol outcome expectancies of panic-reduction strongly influence the perceived physiological effects of a stressor following beverage

consumption. They noted that both pharmacological and expectancy stress response dampening models were supported in this clinical population, and highlighted the important role of cognitions on perceived panic symptoms. Given the strong overlap between the belief systems of panic disorder patients and non-clinical populations with high anxiety sensitivity (cf., Peterson & Plehn, 1999), expectancy factors could theoretically be just as salient for non-clinical high anxiety sensitive individuals in terms of emotional-response dampening.

Social Phobic Patients. A recent study with social phobic individuals, another anxiety disorder characterized by high levels of anxiety sensitivity, has shown stress response dampening effects which appear to be attributable to expectancy effects (Himle, Abelson, Haghightgou, Hill, Nesse, & Curtis, 1999). Himle et al. (1999) exposed two groups of treatment-seeking individuals with social phobia to a social stressor in which they were asked to give two impromptu speeches. One group received a placebo beverage prior to each speech, while the other group received a placebo prior to the first speech and alcohol prior to the second speech. While there were no significant differences on anxiety measures between the two conditions, the belief that alcohol had been consumed reduced subjective reports of anxiety and negative cognitions on the first speech in the dual placebo group. These results led Himle et al. (1999) to suggest that the pharmacological properties of alcohol do not directly reduce social phobic anxiety, but alcohol expectancy may reduce the experience of social anxiety in this clinical population. These results differ from Lehman et al. (2000) in that expectancy alone, rather than an interaction of expectancy and pharmacological effects, appeared to mediate observed stress response dampening (Himle et al., 1999).

## Testing Alcohol Expectancy Effects Empirically

### The Balanced Placebo Design

Study One demonstrated enhanced receptivity to the affective, and to a lesser extent, cognitive, reactivity-dampening effects of alcohol in high anxiety sensitive individuals, consistent with previous work in this area (Conrod et al., 1998; Stewart & Pihl, 1994). Use of a placebo control clearly indicated that the pharmacological properties of alcohol contributed strongly to these effects. Moreover, the experience of dampening may explain future development of alcohol problems in this population. However, there are several compelling reasons to extend these findings by examining the potential role of expectancy effects in producing the dampening seen in high anxiety sensitive individuals. First, there is evidence that alcohol outcome expectancies alone can produce stress response dampening effects (de Boer et al., 1993; Keane & Lisman, 1980; de Boer et al., 1994; Lehman et al., 2000; Wilson & Abrams, 1977; Young et al., 1990); this was not tested in Study One. Second, there appear to be elevations in expectancies that alcohol will produce tension reduction in both high anxiety sensitive alcoholics (Karp, 1993) and in high anxiety sensitive, non-alcoholic, young adults (McKay, 2000); this suggests that alcohol expectancies for tension reduction may be particularly important to study in the laboratory among high anxiety sensitive individuals. Third, potential links exist between alcohol expectancies and increased drinking in high anxiety sensitive individuals (Ham et al., 2002; Kushner, Abrams, et al., 2000). Thus, it is important to explore possible reasons why alcohol expectancies may promote increased alcohol consumption in individuals with high levels of anxiety sensitivity. One of these reasons may be that tension reduction alcohol expectancies could have a positive influence on alcohol dampening effects

among high anxiety sensitive individuals (Lehman et al., 2000; Mulligan & McKay, 2001), a hypothesis deserving controlled empirical study.

Study One has already demonstrated that expectancy effects do not supersede alcohol's pharmacological, anxiolytic effects. However, it is still not known if expectancy effects work together with pharmacology to create anxiolytic effects (e.g., Lehman et al., 2000), or have no discernable influence on high anxiety participants. Moreover, we do not know if expectancy alone would produce any dampening effects. Unfortunately, it is difficult to measure pure expectancy effects in experiments (like Study One) that compare an alcohol group with a placebo control, as both conditions involve expectancy activation. One paradigm that has been developed to empirically separate the contributions of alcohol expectancy from pharmacology is the balanced placebo design (see review by Hull & Bond, 1986). In this paradigm, the dose of alcohol administered (given alcohol vs. given placebo) is crossed factorially with the dose instructions provided to subjects (told alcohol vs. told placebo) to yield four cells. In the first cell, the participant is told that he/she will be administered alcohol and, in fact, receives alcohol. In this condition, both pharmacology and expectancy effects are potentially present. The second cell is one in which the participant is told that he/she will be administered alcohol and receives a placebo instead. In this condition, the pharmacological effects of alcohol are absent, and expectancy effects are potentially activated in isolation. This is the "traditional placebo" condition. The third cell has the participant being told that he/she will be administered a placebo beverage, when in fact he/she receives an alcoholic beverage. This is a "reverse placebo" condition, in which potential expectancy effects are theoretically absent and only pharmacological alcohol effects are potentially present. The

final cell is one in which the participant is told that he/she will consume a placebo beverage and does, in fact, receive a placebo, providing a control condition in which both pharmacological and expectancy effects are absent.

Modified Balanced Placebo Designs. The balanced placebo design has proven effective when relatively low alcohol doses are used, as subjects cannot correctly discriminate the traditional placebo from the reverse placebo conditions (see Bradlyn & Young, 1983). Moreover, it has been shown to accurately assess pharmacological and/or expectancy effects of alcohol in a number of domains like mood, physiological reactivity and social behaviours (see review by Hull & Bond, 1986). However, a number of researchers have highlighted difficulties in achieving effective deception in the reverse placebo condition, especially when alcohol doses in the legally intoxicating range are used (i.e., blood alcohol levels in the .08 to .10% range) (Martin, Earleywine, & Young, 1990; Rohsenow & Marlatt, 1981; Ross & Pihl, 1989). One study found that 44% of subjects in the reverse placebo condition reported a belief that they had consumed at least some alcohol (Sayette, Breslin, Wilson, & Rosenblum, 1994). In contrast, Sayette et al. (1994) found that the traditional placebo condition, in which pure expectancy effects are measured, was a very effective experimental deception, with 94% of participants believing that they had consumed alcohol. In response to this problem, some researchers have chosen to use a modified three cell version of the balanced placebo design to directly assess the effects of expectancy factors, while omitting the reverse placebo cell in which participants are told they will be receiving placebo and are administered alcohol instead (e.g., Newlin, 1989; Sayette, Smith, Breiner, & Wilson, 1993). Thus, the three remaining cells in this design are: told alcohol-given alcohol (pharmacology plus

expectancy); told alcohol-given placebo (expectancy only – the "traditional placebo" condition); and told placebo-given placebo (the control condition).

## Alcohol Expectancies and Differential Behavioural Effects

### Volitional vs. Automatic Behaviours

Previous research using the balanced placebo design to investigate alcohol effects on tension has utilized a wide range of dependant measures of "tension", including behavioural, self-report, cognitive, and physiological measures (see review by Greely & Oei, 1999). The results of these studies have been equivocal. Some studies have shown alcohol's tension-reducing effects to be purely a function of alcohol expectancy (e.g., Wilson & Abrams, 1977). Other research has revealed tension-reduction effects that appear to be wholly attributable to pharmacology (e.g., Levenson et al., 1980; Sutker, Allain, Brantley & Randall, 1982). A number of studies have also demonstrated an interaction of both expectancy and pharmacological influences on subsequent decreases in anxiety or physiological arousal (Lehman et al., 2000; Polivy, Schueneman, & Carlson, 1976). It is difficult to interpret these results definitively. However, work by Hull and Bond (1986) suggests a potential organizing principle for the diversity of effects that have been observed. Using data accumulated from a meta-analysis of balanced placebo designs, Hull and Bond (1986) argued that behaviours following alcohol consumption are affected by either the pharmacological or expectancy effects of alcohol depending on the type of behaviour. For example, behaviours which operate under a high degree of volitional control (e.g., social behaviours) are more likely to be influenced by expectancy effects, whereas behaviours which are relatively more automatic (e.g., nonsocial behaviours such as physiological arousal or psychomotor behaviours) are more

likely to be influenced by alcohol's pharmacological properties (Hull & Bond, 1986). Elsewhere we have suggested that self-reported aspects of the anxiety/tension response would be under more volitional control and might therefore be expected to show stronger expectancy influences, while physiological responses, being more automatic, would be influenced to a greater degree by the pharmacological properties of alcohol (Stewart et al., 1999).

Situational Specificity of Alcohol Expectancy Effects

We have suggested that diversity in expectancy effects may be explained by pharmacological alcohol effects on more automatic behaviours (e.g., somatic responses) and expectancy-mediated alcohol effects on more volitional behaviours (e.g., self-reported anxiety/tension) (Stewart et al., 1999). There is also increasing interest in the situational specificity of alcohol expectancies for explaining differing alcohol expectancy effects. Vogel-Sprott and Fillmore (1999) view alcohol expectancies as a series of three learned associations between four types of events. The first event is an environmental cue for alcohol, such as an alcoholic smell or the sight of a beer bottle. The second event is comprised of the drug stimulus itself, that is, administration of alcohol. The third event involves a physiological/psychological response to the drug. For example, many people report positive changes in mood following alcohol consumption. The final event is an outcome, such as facilitated social interaction or social impairment depending on the situation. The repetition of these events in the context of taking alcohol allows an individual to learn associations between pairs of successive events and yields three expectancies: for example, environmental cues may lead to an expectation of alcohol administration, alcohol administration may elicit expectations of a positive mood and

positive mood may foster expectations for improved social interactions. Vogel-Sprott and Fillmore (1999) make the point that all three expectancies are often present to attain a specific behavioural consequence.

From a cognitive-learning perspective, if certain drinking outcomes are consistently paired with certain drinking contexts, these effects should come to be expected in those contexts in the future. For example, if a high anxiety sensitive individual experiences fearful anxiety sensations (the drinking context) and is exposed to cues for alcohol (triggering the first expectancy), he may have a drink (alcohol administration – triggering the second expectancy) and experience tension-reduction effects (triggering the third expectancy), leading to an outcome of relaxation and reduced fear. With enough repetitions of these circumstances, tension-reduction expectancies should be activated when high anxiety sensitive subjects are exposed to feared anxiety sensations in the future. Vogel-Sprott and Fillmore (1999) further suggest that the *desirability* of a particular outcome influences the activation of a particular response to alcohol. Thus, in the example noted above, if alcohol cues trigger expectations for tension-reduction and subsequent relaxation, and this is a desired outcome, it is more likely that such effects would be experienced. However, if the outcome is interpreted as undesirable, individuals may actively resist responses which would lead to that outcome (Vogel-Sprott & Fillmore, 1999). For example, a socially anxious individual who is experiencing an anxiety reaction may expect alcohol to promote a tension-reduction response, leading to an outcome of decreased anxiety and relaxation. However, if they drink in a social setting, socially anxious individuals may feel that an outcome of relaxation is not desirable, in that relaxation may cause them to let their guard down and

expose themselves to social scrutiny. Conversely, resisting a tension-reduction response may lead to a more desirable outcome (i.e., maintaining self-control). Thus, the individual may become hyper-vigilant in response to feelings of tension-reduction, ultimately experiencing less relaxation than might otherwise be observed in a different setting. In contrast, when alone at home, relaxation may be a highly desirable outcome for a socially anxious individual and so he/she may not resist tension-reduction effects, thereby experiencing increased relaxation. Thus, the situational context may be vital in determining the influence of expectancies on dampening of anxious reactions in high anxiety sensitive individuals.

Maximizing Tension-Reduction Expectancy Effects in High Anxiety Sensitive Individuals

Research with university students has shown that Anxiety Sensitivity Index scores are correlated with the frequency of drinking in negatively-reinforcing drinking situations (i.e., typically solitary contexts involving physical discomfort, conflict with others, and unpleasant emotions) (Samoluk & Stewart, 1998). Conversely, Samoluk and Stewart (1998) found that anxiety sensitivity levels appear to be unrelated to frequency of drinking in positively reinforcing drinking situations (i.e., contexts involving social cues to drink, pleasant times with others and pleasant emotions) on the Inventory of Drinking Situations (Annis et al., 1987). Given the theoretical likelihood of situational specificity of expectancy-based behaviours (Vogel-Sprott & Fillmore, 1999) among high anxiety sensitive individuals and demonstrations of their drinking selectively in potentially-negatively reinforcing drinking contexts (Samoluk & Stewart, 1998; Stewart, Zvolensky, et al., 2001), it might be predicted that the hypothesized associations between anxiety

sensitivity and increased tension-reduction expectancies might be strongest when high anxiety sensitive individuals are exposed to typical drinking contexts (i.e., alone and drinking to cope with an anxiogenic stressor).

## Testing the Effects of Alcohol Expectancy on Stress Response Dampening Effects in High Anxiety Sensitive Individuals

There is empirical evidence that positive alcohol expectancies, particularly tension-reduction expectancies, may mediate stress response dampening effects (de Boer et al., 1993; de Boer et al., 1994; Keane & Lisman, 1980; Wilson & Abrams, 1977), and that expectancies may have specific influences on dampening effects in populations with high anxiety sensitivity (Himle et al., 1999; Lehman et al., 2000; Mulligan & McKay, 2001). There are also theoretical indications that expectancy factors may influence stress response dampening in general (Goldman et al., 1987, Young & Knight, 1990) and in high anxiety sensitive individuals in particular (Stewart et al., 1999). Although expectancy factors were controlled in Study One, it remains to be seen whether alcohol expectancies may have contributed directly to the reactivity dampening demonstrated among high anxiety sensitive participants. To test this idea, use of a modified balanced placebo design (e.g., Newlin, 1989; Sayette et al., 1993) would be necessary to separate potential expectancy factors from the pharmacological effects of alcohol. Moreover, following the theory of Vogel-Sprott and Fillmore (1999), three layers of expectancies would have to be present for activation of a particular outcome like stress response dampening. First, the experimental environment would have to lead participants to believe that they would be receiving alcohol. Second, cues signaling alcohol administration would have to be present (e.g., smell, taste and visual cues). Finally,

participants would have to experience some sort of physiological or psychological response to the placebo beverage. If this response (e.g., relaxation) is desirable to the participant in the experimental setting, an outcome like stress response dampening might take place. Tension-reduction expectancies would also be most strongly activated in a typical drinking context for high anxiety sensitive individuals. This would mean that they should be alone and drinking to cope with an anxiogenic stressor (Samoluk & Stewart, 1998; Samoluk et al., 1998; Stewart et al., 1999). Finally, aspects of the fear response which are theoretically under the most volitional control (e.g., cognitive and affective reactivity) (Hull & Bond, 1986) would be expected to be most influenced by expectancy factors, while more automatic components of the fear response (e.g., somatic reactivity) should be affected predominantly by the pharmacological properties of alcohol (Stewart et al., 1999). These considerations led to the development of Study Two, presented in the next chapter.

# CHAPTER FOUR: Study Two

## The Roles of Alcohol and Alcohol Expectancy in the Dampening of Responses to Hyperventilation among High Anxiety Sensitive Young Adults

While Study One demonstrated that alcohol exerted a unique emotional reactivity-dampening effect in high anxiety sensitive individuals, all comparisons made in that study were between alcohol (high or low dose) and placebo conditions. With the potential influences of alcohol expectancies (Goldman et al., 1987) controlled through the inclusion of a placebo cell, it is tempting to conclude that the emotional reactivity-dampening effects of alcohol observed in my earlier study were pharmacological in nature. However, I did not examine the possible *additional* contributions of alcohol outcome expectancies to the emotional reactivity-dampening effects of alcohol among high anxiety sensitive individuals.

The present study was designed to replicate findings of alcohol dampening of reactivity to hyperventilation challenge in high anxiety sensitive individuals as seen in Study One. In addition, I hoped to extend these findings by evaluating the potentially unique effects of alcohol expectancies on alcohol dampening in high anxiety sensitive individuals. Specifically, I employed a three-cell Beverage Condition design to test the contributions of pharmacological and expectancy factors on the emotional-reactivity dampening properties of alcohol among high anxiety sensitive young adults. The current methodology further differed from Study One in that a low anxiety sensitive group was excluded from the present study. It is now well established that, relative to sober low anxiety sensitive individuals, sober high anxiety sensitive people show greater emotional

responsiveness to a voluntary hyperventilation challenge (e.g., Asmundson et al., 1994; Donnell & McNally, 1989; Holloway & McNally, 1987; Rapee & Medoro, 1994). Moreover, high levels of anxiety sensitivity have been strongly correlated with several clinically relevant alcohol outcomes (see our review in Stewart et al., 1999). Finally, the findings of Study One added to a growing body of evidence suggesting that, compared to low anxiety sensitive individuals, high anxiety sensitive individuals show greater sensitivity to alcohol dampening of subjective emotional responses in response to laboratory challenges (Stewart & Pihl, 1994). Thus, the present study represents an initial effort to determine whether expectancies play a role in alcohol dampening of responses to hyperventilation challenge among high anxiety sensitive individuals.

The study tested three major hypotheses. First, I hypothesized that affective, cognitive, and somatic responses to a hyperventilation challenge would be dampened among high anxiety sensitive young adults administered alcohol relative to high anxiety sensitive young adults administered placebo, replicating the findings of Study One. In addition, I expected that high anxiety sensitive young adults administered alcohol would also show dampened affective, cognitive, and somatic responses compared to high anxiety sensitive young adults administered the control beverage (cf., Stewart & Pihl, 1994). Finally, high anxiety sensitive participants in the placebo condition were expected to show dampened affective and cognitive, but not somatic, responses to the hyperventilation challenge when compared to high anxiety sensitive participants in the control Beverage Condition. That is, direct expectancy effects were predicted for aspects of the response to hyperventilation that are theoretically under the greatest degree of volitional control (Hull & Bond, 1986; Stewart et al., 1999).

## Method

The methodology employed in Study Two was exactly the same as Study One with the exception of a few key changes, noted below.

### Participants

Two high anxiety sensitive participants originally run in the study were excluded from the final analysis based on DSM-IV (American Psychiatric Association, 1994) criteria for panic disorder as measured by the Panic Attack Questionnaire - Revised (Cox et al., 1992). These criteria included history of spontaneous panic attacks, recurrent attacks, at least one month of significant concerns about the implications of having future panic attacks, and panic attacks characterized by at least four of the symptoms as listed in the DSM-IV (American Psychiatric Association, 1994). They were replaced with two additional high anxiety sensitive participants with no history of panic disorder.

The final sample consisted of 48 students who met the selection criteria. There were 17 participants in the control cell (told placebo - given placebo), 17 participants in the placebo cell (told alcohol - given placebo) and 17 participants in the alcohol cell (told alcohol - given alcohol). Three subjects were excluded from the final analysis because they were not willing/able to complete the experimental protocol. Two participants were dropped from the placebo cell when they were unable to complete the hyperventilation task owing to feelings of nausea following consumption of the placebo beverages, while one was dropped from the alcohol cell after failing to consume the assigned number of beverages to achieve the target blood alcohol concentration. This resulted in cells of slightly unequal N (control: N = 17; placebo: N = 15; alcohol: N = 16). Although a greater proportion of women than men volunteered for this study (resultant sample =

70.8% women), this gender composition is consistent, and therefore comparable, with previous studies of young adults' responses to hyperventilation challenge (63.8% women (Donnell & McNally, 1989) to 81.0% women (Rapee & Medoro, 1994)) and with the gender composition seen in Study One (65.7% women). Moreover, the present gender imbalance reflects the gender composition of the population from which participants were drawn, since females are typically over-represented among the enrolment of undergraduate psychology courses (Stewart, Taylor, et al., 1997).

Materials

There were no changes in materials between Study One and Study Two.

Procedure

Participants were randomly assigned to one of the three Beverage Conditions to complete three cells of equal N (17 in each cell) and were provided with their assigned beverage (i.e., control, placebo, or alcoholic beverage). Four to five drinks (depending on total volume) were placed in front of the participants, along with a bottle of tonic water and a bottle of 100 proof vodka. Control participants were informed that the vodka bottle was present to keep the procedure standardised with that of the alcohol condition. While control participants were informed that they would be receiving non-alcoholic beverages, both placebo and alcohol participants were informed that they would be receiving alcoholic beverages. The alcohol dose used was the high dose used in Study One – specifically, 2.28 ml 50% USP units of alcohol per kilogram of body weight for females and 2.73 ml 50% USP units of alcohol per kilogram of body weight for males, mixed 1:4 parts alcohol to tonic water. Participants in the placebo and alcohol conditions were told that they would be receiving an amount of alcohol equivalent to four or five mixed bar

drinks to enhance activation of alcohol expectancies (Kushner et al., 1996). In order to further enhance the beverage deception in the placebo condition, the placebo drinks, which were matched for volume with the alcohol drinks, contained a small amount of vodka on top to provide smell and taste cues of alcohol (Kushner et al., 1996). The open bottle of vodka on the same table as the drinks served as a visual cue. A similar procedure was used in the preparation of the beverages for those in the control condition.

Participants in the control Beverage Condition were made aware of the presence of the vodka as both visual and smell/taste cues, and were told that these cues served to keep the procedure standardised with that of the alcohol condition. Since a small amount of alcohol was present in the placebo and control drinks, participants in these groups were expected to show slightly elevated blood alcohol concentrations. However, resultant blood alcohol concentrations were not expected to be significantly greater than .000% in the placebo or control group by the time the hyperventilation stressor was applied (cf., Stewart et al., 1992, 1995).

## Results

### Anxiety Relevant Measures

Means (and SDs) for the anxiety-related measures (Anxiety Sensitivity Index, State Trait Anxiety Inventory - Trait Subscale, and percentage of participants with a history of different types of panic on the Panic Attack Questionnaire - Revised) are shown in Table 4. Anxiety Sensitivity Index scores were subjected to a one-way (Beverage Condition) Analysis of Variance (ANOVA). No significant main effect of Beverage Condition emerged. State Trait Anxiety Inventory - Trait Subscale scores were also subjected to a one-way (Beverage Condition) Analysis of Variance (ANOVA).

Table 4

Means (and SDs) of Control Measures as a Function of Beverage Condition.

| | Beverage Condition | | | | | |
|---|---|---|---|---|---|---|
| | Control (N = 17) | | Placebo (N = 15) | | Alcohol (N = 16) | |
| Measure | Mean | SD | Mean | SD | Mean | SD |
| ASI | 32.00 | 7.02 | 29.40 | 4.69 | 31.75 | 5.00 |
| STAI-T | 40.82 | 7.54 | 44.27 | 8.05 | 42.13 | 8.80 |
| Panic Attacks (% uncued) | 5.88 | (n=1) | 6.67 | (n=1) | 6.25 | (n=1) |
| Panic Attacks (% cued) | 17.65 | (n=3) | 6.67 | (n=1) | 12.50 | (n=2) |
| Panic Attacks (% both) | 5.88 | (n=1) | 13.33 | (n=2) | 6.25 | (n=1) |
| Age (years) | 19.94 | 2.14 | 19.73 | 1.22 | 20.69 | 3.52 |
| Gender (% female) | 70.59 | ---- | 66.67 | ---- | 75.00 | ---- |
| Drinks per week | 11.51 | 11.72 | 11.17 | 14.93 | 5.84 | 4.94 |
| Days Since Last Menses | 13.73 | 7.58 | 18.00 | 12.94 | 14.25 | 10.69 |
| Oral Contraception (% users) | 50.00 | ---- | 20.00 | ---- | 58.30 | ---- |

ASI = Anxiety Sensitivity Index (Reiss et al., 1986); STAI-T = State-Trait Anxiety Inventory - Trait subscale (Spielberger et al., 1983). Days since last menses and oral contraceptive use are for women only.

Again, no significant main effect of Beverage Condition was noted. Panic Attack Questionnaire - Revised scores were subjected to chi-square analyses. No significant between-Beverage Condition effect was observed for the proportion of participants who had experienced only cued panic attacks (i.e., situationally bound or situationally predisposed attacks). Similarly, no significant between-Beverage Condition effects were observed for the proportion of participants who had experienced only uncued (i.e., unexpected) panic attacks, or both cued and uncued panic attacks.

Demographic Measures

A series of one-way (Beverage Condition) ANOVAs and chi-square analyses (for dichotomous variables) were performed on several demographic variables (see Table 4) to ensure equivalence between Beverage Conditions on control measures. No significant differences between Beverage Conditions were noted for any of the demographic variables.

Manipulation Check: Intoxication Levels

A one-way (Beverage Condition) ANOVA was performed on the visual analogue scale scores, revealing a significant Beverage Condition main effect, $F$ (2, 45) = 59.40, $p$ < .0001. Newman-Keuls comparisons showed that those in both the alcohol ($M$ = 61.89, $SD$ = 20.27), and placebo ($M$ = 23.81, $SD$ = 16.92), conditions rated themselves as being significantly more intoxicated than those in the control condition ($M$ = 4.54, $SD$ = 4.92) (both $p$ < .05). This indicates efficacy of the placebo (expectancy) manipulation in inducing subjective intoxication. However, those in the alcohol condition rated themselves as significantly more intoxicated than those in the placebo condition ($p$ < .05),

indicating that the placebo (expectancy) manipulation failed to induce subjective intoxication levels equivalent to those produced by actual alcohol administration.

Another one-way (Beverage Condition) ANOVA was conducted on the blood alcohol concentrations collected just prior to hyperventilation. There was a significant main effect of Beverage Condition, $F$ (2, 45) = 440.12, $p < .0001$. Newman-Keuls comparisons revealed that the alcohol condition reached a higher mean blood alcohol concentration ($M$ = .094%, $SD$ = .018%) than the placebo ($M$ = .001%, $SD$ = .001%) and control ($M$ = .001%, $SD$ = .001%) conditions (both $p < .01$). There was no statistical difference between the placebo and control conditions in terms of blood alcohol concentration achieved and neither placebo nor control conditions achieved mean blood alcohol concentrations significantly greater than .000%.

Analysis of Hyperventilation Questionnaire Baseline Scores

Hyperventilation Questionnaire pre- and post-drinking baseline scores are illustrated in Figure 1 as a function of Beverage Condition. These baseline subscale scores were subjected to a set of 3 x 2 (Beverage Condition x Time) repeated measures ANOVAs with Time (pre- and post-drinking baseline) as the repeated measure.

For the affective subscale, there was no significant main effect for Beverage Condition, and only a marginally significant main effect of Time, $F$ (1, 45) = 3.10, $p < .09$. However, a significant Beverage Condition x Time interaction emerged, $F$ (2, 45) = 5.07, $p < .05$. I examined the simple effects of Time within each Beverage Condition. While affective scores did not show significant changes from pre- to post-drinking baseline for the control or alcohol conditions, affective scores did show a significant *decrease* from pre- to post-drinking baseline for the placebo condition,

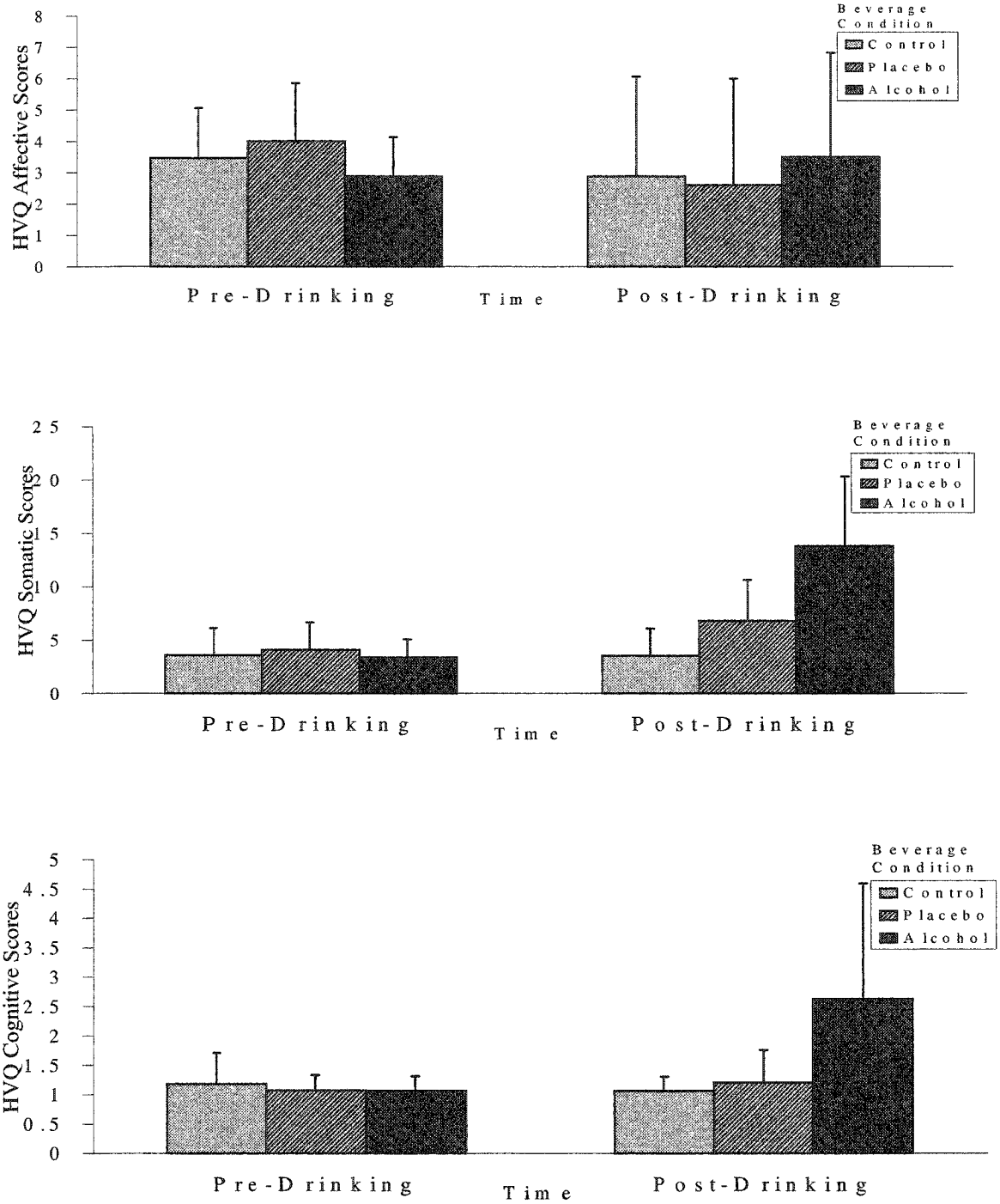


Figure 1. Means for the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) Sub-Scale Scores at Baseline as Functions of Beverage Condition (Control, Placebo, and Alcohol) and Time (Pre-Drinking and Post-Drinking Baseline). Error bars represent standard deviations.

$F$ (1, 14) = 9.44, $p < .01$ (see Figure 1, top panel). I also examined the simple effects of Beverage Condition at both pre- and post- drinking baseline. There was no significant simple effect of Beverage Condition at pre-drinking baseline or at post-drinking baseline.

For the somatic subscale, there was a significant main effect of Beverage Condition, $F$ (2, 45) = 12.89, $p < .001$ and a significant main effect of Time, $F$ (1, 45) = 46.25, $p < .001$. These effects were qualified by a significant Beverage Condition x Time interaction, $F$ (2, 45) = 24.34, $p < .001$. I examined the simple effects of Time within each Beverage Condition. Somatic scores did not show significant changes from pre- to post-drinking baseline for the control condition. In contrast, somatic scores did show a significant *increase* from pre- to post-drinking baseline for the placebo condition, $F$ (1, 14) = 10.40, $p < .01$, and alcohol condition, $F$ (1, 15) = 38.68, $p < .001$, (see Maisto et al., 1980, for a discussion of alcohol's effects in increasing baseline somatic symptoms such as "dizziness"). The increase was of larger magnitude in the alcohol condition ($\eta^2$ = 0.72) relative to the placebo condition ($\eta^2$ = 0.43) (see Figure 1, middle panel). I also examined the simple effects of Beverage Condition at both pre- and post- drinking baseline. There was no significant simple effect of Beverage Condition at pre-drinking baseline, but a significant simple effect of Beverage Condition emerged at post-drinking baseline, $F$ (2, 45) = 21.47, $p < .001$. Newman-Keuls comparisons revealed that those in the alcohol condition reported higher Hyperventilation Questionnaire somatic scores than the placebo and control conditions (both $p < .05$; see Figure 1, middle panel). Those in the placebo condition also reported higher somatic scores than those in the control condition at the post-drinking baseline ($p < .05$; see Figure 1, middle panel).

For the cognitive subscale, there was a significant main effect of Beverage Condition, $F$ (2, 45) = 6.58, $p < .005$ and a significant main effect of Time, $F$ (1, 45) = 9.40, $p < .005$. These effects were qualified by a significant Beverage Condition x Time interaction, $F$ (2, 45) = 9.42, $p < .001$. I examined the simple effects of Time within each Beverage Condition. While cognitive scores did not show significant changes from pre- to post-drinking baseline for the control or placebo conditions, cognitive scores did show a significant *increase* from pre- to post-drinking baseline for the alcohol condition, $F$ (1, 15) = 10.48, $p < .01$ (see Figure 1, bottom panel; see Maisto et al., 1980, for a discussion of alcohol's effects in increasing baseline cognitive symptoms such as "feel like passing out"). I also examined the simple effects of Beverage Condition at both pre- and post-drinking baseline. There was no significant simple effect of Beverage Condition at pre-drinking baseline, but a significant simple effect of Beverage Condition emerged at post-drinking baseline, $F$ (2, 45) = 8.62, $p < .005$. Newman-Keuls comparisons revealed that those in the alcohol condition reported higher Hyperventilation Questionnaire cognitive scores than those in the placebo and control conditions (both $p < .05$; see Figure 1, bottom panel). There was no statistical difference between the placebo and control conditions in terms of Hyperventilation Questionnaire cognitive scores at the post-drinking baseline.

Response to Hyperventilation Measured on Hyperventilation Questionnaire

There were significant Beverage Condition effects for cognitive and somatic scores at the post-drinking baseline, but not at the pre-drinking baseline. Thus, as in Study One, post-drinking Hyperventilation Questionnaire scores were selected as the most appropriate baseline for examining responses to hyperventilation because they took

into account these Beverage Condition differences prior to hyperventilation. Reactivity to hyperventilation on the Hyperventilation Questionnaire subscales was examined with relation to the initial hypotheses through a set of 3 x 2 (Beverage Condition x Time) repeated measures ANOVAs with Time (post-drinking baseline vs. hyperventilation) serving as the repeated measure. Hyperventilation Questionnaire post-drinking baseline and hyperventilation scores are illustrated in Table 5 as a function of Beverage Condition.

For the affective subscale, there was no significant main effect for Beverage Condition. However, there was a significant main effect of Time, $F$ (1, 45) = 36.38, $p$ < .001, which was qualified by a significant Beverage Condition x Time interaction, $F$ (2, 45) = 3.53, $p$ < .05. To explore the interaction, I further examined Beverage Condition x Time effects for all two-way Beverage Condition comparisons. The Beverage Condition x Time interaction was significant in the alcohol versus placebo comparison, $F$ (1, 29) = 6.59, $p$ < .05, and marginally significant in the alcohol versus control comparison, $F$ (1, 31) = 3.55, $p$ < .07. However, the Beverage Condition x Time interaction was not significant in the placebo versus control comparison (see Table 5, panel a). The two-way interactions in the alcohol versus placebo and alcohol versus control comparisons were further explored by examining simple effects of Time in each Beverage Condition. Consistent with my alcohol dampening hypothesis, affective scores did not show a significant increase from post-drinking baseline to hyperventilation for the alcohol condition (see Table 5, panel a). However, affective scores did show a significant increase from post-drinking baseline to hyperventilation in the placebo condition, $F$ (1, 14) = 16.63, $p$ < .01, and control condition, $F$ (1, 16) = 16.39, $p$ < .01 (see Table 5, panel a).

Table 5

Means (and SDs) for the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) Sub-Scale Scores as Functions of Beverage Condition (Control, Placebo, and Alcohol) and Time (Post-Drinking Baseline and Hyperventilation).

| | Time | |
|---|---|---|
| a) HVQ-Affective | Post-Drinking Baseline | Hyperventilation |
| Beverage Condition | | |
| Control | 2.88 (1.41) | 5.59 (3.18) |
| Placebo | 2.60 (0.99) | 6.40 (3.40) |
| Alcohol | 3.50 (2.22) | 4.56 (3.33) |
| b) HVQ-Somatic | Post-Drinking Baseline | Hyperventilation |
| Beverage Condition | | |
| Control | 3.53 (2.55) | 10.82 (7.58) |
| Placebo | 6.80 (3.84) | 15.47 (8.85) |
| Alcohol | 13.81 (6.50) | 16.94 (9.09) |
| c) HVQ-Cognitive | Post-Drinking Baseline | Hyperventilation |
| Beverage Condition | | |
| Control | 1.06 (0.24) | 2.06 (1.56) |
| Placebo | 1.20 (0.56) | 2.53 (2.23) |
| Alcohol | 2.63 (1.96) | 3.25 (3.02) |

Note: Values represent raw score subscale means; possible range (affective) = 0-21; possible range (cognitive) = 0-18; possible range (somatic) = 0-54.

For the somatic subscale, there was a significant main effect of Beverage Condition, $F$ (2, 45) = 7.82, $p < .01$, and a significant main effect of Time, $F$ (1, 45) = 43.11, $p < .001$. These main effects were qualified by a marginally significant Beverage Condition x Time interaction, $F$ (2, 45) = 2.92, $p < .07$ (see Table 5, panel b). To explore the interaction, I further examined Beverage Condition x Time effects for all two-way Beverage Condition comparisons. The Beverage Condition x Time interaction was significant in the alcohol versus placebo comparison, $F$ (1, 29) = 4.96, $p < .05$, and marginally significant in the alcohol versus control comparison, $F$ (1, 31) = 3.90, $p < .06$. However, the Beverage Condition x Time interaction was not significant in the placebo versus control comparison (see Table 5, panel b). The two-way interactions in the alcohol versus placebo and alcohol versus control were further explored by examining simple effects of Time in each Beverage Condition. Somatic scores increased significantly from post-drinking baseline to hyperventilation in all three Beverage Conditions (i.e., $F$ (1, 15) = 4.63, $p < .05$; F (1, 16) = 22.84, $p < .001$; $F$ (1, 14) = 17.86, $p < .005$; for the alcohol, placebo, and control conditions, respectively). Consistent with the alcohol-dampening hypothesis, pair-wise comparisons of difference scores between the post-drinking baseline and hyperventilation revealed that the increase in the alcohol condition was significantly smaller than the increase in the placebo condition ($p < .05$) and marginally smaller than the increase in the control condition ($p < .09$).

For the cognitive subscale, there was a significant main effect for Beverage Condition, $F$ (2, 45) = 3.40, $p < .05$, and a significant main effect of Time, $F$ (1, 45) = 12.54, $p < .01$ (see Table 5, panel c). The predicted Beverage Condition x Time interaction was not statistically significant (see Table 5, panel c). Similarly, none of the

Beverage Condition x Time interactions in any of the specific two-way Beverage Condition comparisons proved statistically significant. Nonetheless, in keeping with my a priori hypotheses, I examined the simple effects of Time within each Beverage Condition for the cognitive subscale as I had for the other two Hyperventilation Questionnaire subscales. Consistent with my alcohol-dampening hypothesis, cognitive scores did not show significant increases from post-drinking baseline to hyperventilation for the alcohol condition (see Table 5, panel c). However, cognitive scores did show a significant increase from post-drinking baseline to hyperventilation in the placebo condition, $\underline{F}$ (1, 14) = 5.55, $\underline{p} < .05$, and the control condition, $\underline{F}$ (1, 16) = 8.50, $\underline{p} < .05$ (see Table 5, panel c).

To ensure that evidence for alcohol-dampening was not merely secondary to increased baselines in the alcohol condition, I also examined the effects of Beverage Condition on Hyperventilation Questionnaire subscale scores at hyperventilation, after co-varying out the influence of post-drinking baseline scores. This procedure corrects for possible initial value effects (cf. Stern, Ray, & Davis, 1980). Specifically, each of the Hyperventilation Questionnaire subscale scores at hyperventilation in turn were subjected to a one-way (Beverage Condition) Analysis of Covariance (ANCOVA), with post-drinking baseline subscale scores serving as the covariate. It should be noted that this ANCOVA approach is not redundant with the repeated measures analyses reported above, as the two approaches can yield conflicting results in cases where initial baselines differ – a situation known as "Lord's Paradox" (see Locascio & Cordray, 1983; Winer, 1991; Werts & Linn, 1971). Covariate-adjusted Hyperventilation Questionnaire scores are displayed in Figure 2 as a function of Beverage Condition.

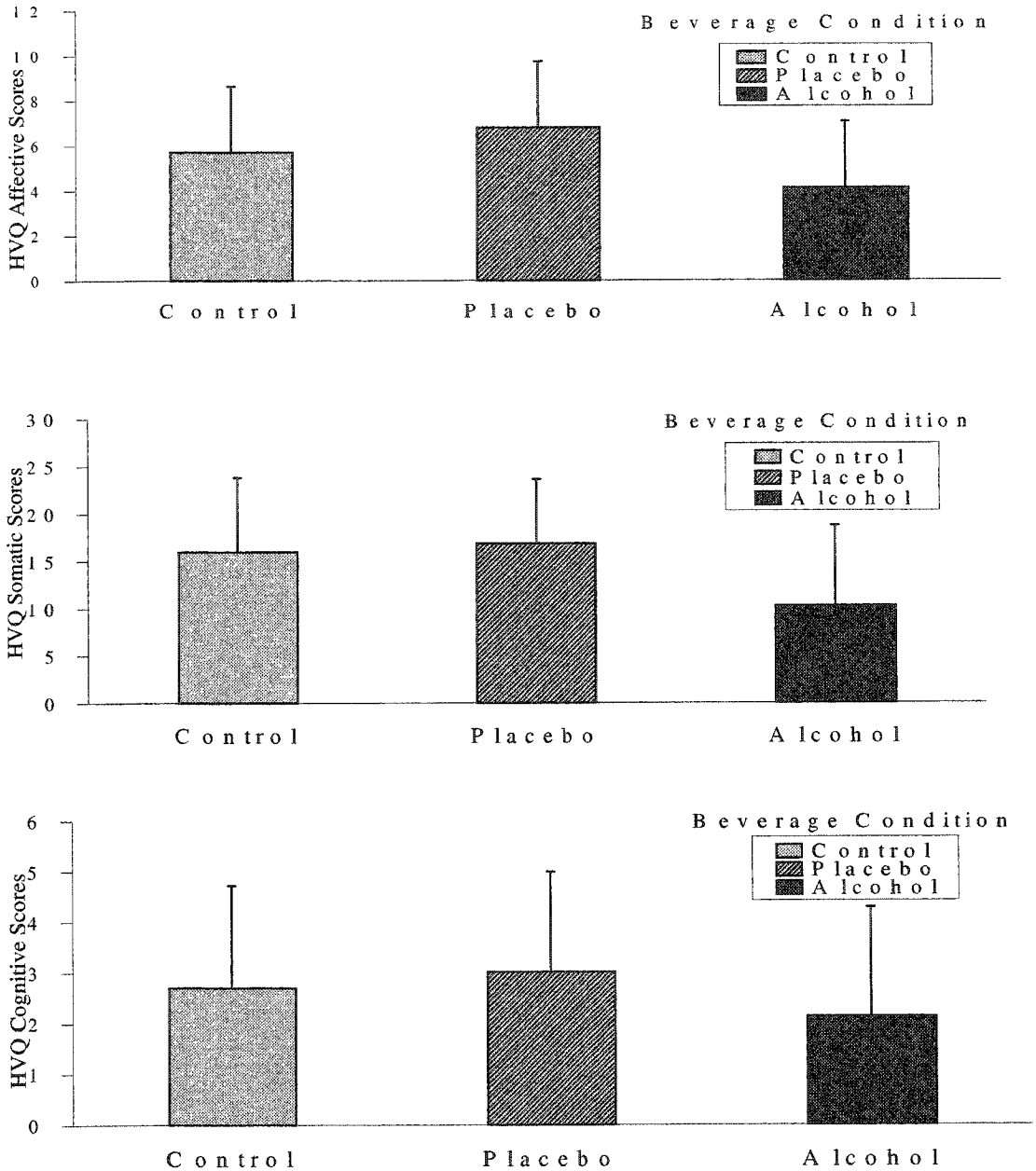


Figure 2. Covariate-Adjusted Means for the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) Sub-Scale Scores at Hyperventilation as a Function of Beverage Condition (Control, Placebo, and Alcohol). Means are adjusted for post-drinking baseline scores to control for possible initial value effects. Error bars represent standard deviations.

For the ANCOVA on affective subscale scores, a significant main effect of Beverage Condition emerged, $F$ (2, 44) = 3.21, $p < .05$. Pair-wise comparisons revealed that the covariate-adjusted affective mean for the placebo participants was significantly greater than the covariate-adjusted affective mean for the alcohol participants, $p < .05$. Effect sizes were calculated by dividing the difference between covariate-adjusted means for the two groups in question by the pooled standard deviation for the two groups, according to suggestions made by Cohen (1977). Cohen noted that effect sizes close to .80 should be considered large, effect sizes close to .50 should be considered medium and effect sizes close to .20 should be considered small. Effect size analysis revealed the difference between the covariate-adjusted affective mean in the placebo group and the covariate-adjusted affective mean in the alcohol group to be a large effect, d = .79. Similarly, the covariate-adjusted affective mean for the control condition was greater than that for the alcohol condition, but the statistical difference was only marginal, $p = .12$. Consistent with this result, the effect size for this comparison was found to be of medium magnitude, d = .50. Covariate-adjusted affective subscale scores of the placebo condition participants failed to differ from those of the control condition at hyperventilation (see Figure 2, top panel).

For the ANCOVA on somatic subscale scores, a marginally significant main effect of Beverage Condition emerged, $F$ (2,44) = 2.68, $p < .09$ (see Figure 2, middle panel). Pair-wise comparisons revealed that the covariate-adjusted somatic mean for the placebo participants was significantly greater than the covariate-adjusted somatic mean for the alcohol participants, $p < .05$. Effect size analysis revealed this to be a large effect, d = .75. Similarly, the covariate-adjusted somatic mean for the control condition was

greater than the covariate-adjusted somatic mean of the alcohol condition, although the statistical difference was only marginal, $p < .09$. Consistent with this result, the effect size for this comparison was found to be medium-large in magnitude, $d = .65$. The covariate-adjusted somatic means for the control and placebo conditions did not differ significantly at hyperventilation (see Figure 2, middle panel).

In the case of the ANCOVA on cognitive subscale scores, the pattern of covariate-adjusted means was similar to the patterns reported above for the affective and somatic subscales (i.e., less reactivity in the alcohol condition relative to the other two Beverage Conditions; see Figure 2, bottom panel). Nonetheless, no significant main effect of Beverage Condition was revealed in the ANCOVA. Consistent with this result, effect sizes were found to be small for both the placebo-alcohol comparison, $d = .33$, and the control-alcohol comparison, $d = .23$.

Post-Hoc Investigation of Expectancy Effects

While significant differences in hyperventilation responses did not appear between placebo and control conditions as predicted by the expectancy hypothesis, co-varied reactivity means were consistently (but not significantly) higher in the placebo condition than in the control condition for all Hyperventilation Questionnaire subscales (see Figure 2). The shape of the data suggests that an "inverse placebo" effect (Wilson, 1988) may have been operative. That is, placebo participants who *expected* to experience tension-reduction (in the absence of pharmacologically induced tension reduction) may have responded with increased reactivity to the stressor compared to control participants who had no such expectation.

To investigate the possible operation of an "inverse placebo" effect, I separated placebo participants into Low and High Expectancy activation groups based on a median split of their visual analogue scale ratings. Given the odd number of participants in the placebo group ($N = 15$), the resulting groups from the median split were unequal in size. As the median participant (visual analogue scale rating of 19.2) scored below the visual analogue scale mean ($M = 23.8$), the median subject was considered to be more typical of the Low Expectancy group, resulting in a Low Expectancy group containing eight participants and a High Expectancy group containing seven participants. I then re-examined scores on the Hyperventilation Questionnaire subscales in the placebo group using a series of 2 x 2 (Expectancy level x Time) repeated measures ANOVAs with Time (post-drinking baseline and hyperventilation) as the repeated measure. Hyperventilation Questionnaire scores for placebo participants are displayed in Figure 3 as a function of Expectancy level and Time.

For the affective subscale, there were significant main effects of Time, $F(1,13) = 20.03$, $p < .01$, and of Expectancy level, $F(1,13) = 4.80$, $p < .05$, qualified by a marginally significant Expectancy level x Time interaction, $F(1,13) = 3.07$, $p = .10$. There was no significant simple effect of Expectancy level at post-drinking baseline. However, a marginally significant simple effect of Expectancy level emerged at hyperventilation, $F(1,13) = 4.26$, $p < .07$, with high Expectancy level participants reporting greater affective symptoms at hyperventilation than low Expectancy level participants (see Figure 3, top panel).

For the somatic subscale, there was a significant main effect of Time, $F(1,13) = 19.37$, $p < .01$, with somatic subscale scores increasing from post-drinking baseline to

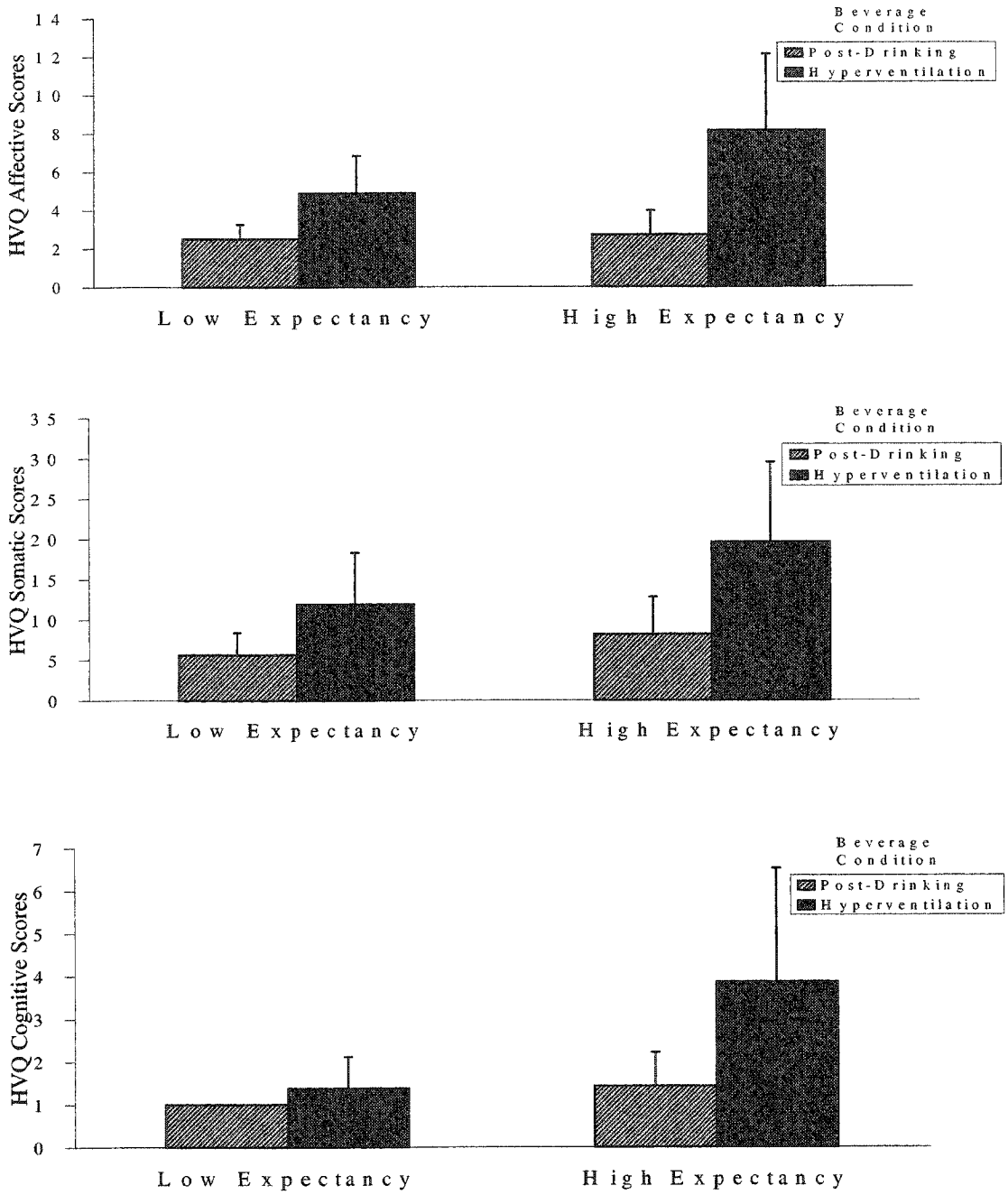


Figure 3. Means for the Hyperventilation Questionnaire (HVQ; Rapee & Medoro, 1994) Sub-Scale Scores in the Placebo Condition (N = 15) as Functions of Time (Post-Drinking and Hyperventilation) and Expectancy Level (Low and High). Error bars represent standard deviations.

hyperventilation, regardless of Expectancy level (see Figure 3, middle panel). There were no other significant effects for the somatic subscale.

For the cognitive subscale, there were significant main effects of Time, $\underline{F}$ (1,13) = 7.39, $\underline{p} < .05$, and of Expectancy level, $\underline{F}$ (1,13) = 8.34, $\underline{p} < .05$, qualified by a marginally significant Expectancy level x Time interaction, $\underline{F}$ (1,13) = 3.97, $\underline{p} < .07$. There was no significant simple effect of Expectancy level at the post-drinking baseline. However, a significant simple effect of Expectancy level emerged at hyperventilation, $\underline{F}$ (1,13) = 6.40, $\underline{p} < .05$, with high Expectancy level participants reporting greater cognitive symptoms at hyperventilation than low Expectancy level participants (see Figure 3,bottom panel).

## Discussion

In the present study, I replicated some important aspects of the results of Study One. High anxiety sensitive participants administered a moderately intoxicating dose of alcohol showed significantly dampened affective and somatic responses, and marginally dampened cognitive responses, to hyperventilation challenge relative to high anxiety sensitive participants administered placebo. Moreover, in comparisons of high anxiety sensitive participants who were administered placebo versus control beverages, I extended my previous findings by showing that expectancy factors alone did not lead to significant dampening of responses to hyperventilation challenge.

Consistent with my first hypothesis, high anxiety sensitive participants in the alcohol condition reported fewer and less intense affective and somatic sensations than high anxiety sensitive participants in the placebo condition, supportive of findings of alcohol-induced stress-response dampening seen in Study One. Similarly, cognitive

Hyperventilation Questionnaire scores increased from post-drinking baseline to hyperventilation in the placebo but not the alcohol conditions, supporting the idea that alcohol dampened negative thoughts in response to the challenge, as suggested in Study One. However, this latter effect must be interpreted with caution as the Beverage Condition x Time interaction failed to reach statistical significance and the effect size for alcohol dampening was small with the cognitive measure.

Consistent with my second hypothesis, high anxiety sensitive participants in the alcohol condition reported less affective, cognitive, and somatic reactivity than high anxiety sensitive participants in the control condition. This extends to a different stressor (i.e., hyperventilation), previous findings of alcohol-induced dampening of responses to stress in high anxiety sensitive individuals in studies that have used a no-alcohol control condition (Conrod et al., 1998; Stewart & Pihl, 1994). Although simple effects were supportive of alcohol dampening in the alcohol versus control Beverage Condition comparisons, it should be cautioned that the two-way Beverage Condition x Time interactions were only marginally significant in the case of the affective and somatic subscales, and non-significant for the cognitive subscale. Moreover, although alcohol dampening effect sizes were medium to large for the affective and somatic measures, the alcohol dampening effect size was again small for the cognitive Hyperventilation Questionnaire subscale.

The lack of significant effects for the cognitive subscale in the present study is not consistent with my finding (albeit marginally significant) of alcohol dampening of cognitive reactivity to hyperventilation challenge among high anxiety sensitive participants in Study One. The absence of a significant Beverage Condition x Time

interaction and the small effect sizes for alcohol dampening of cognitive reactivity observed in the present study support suggest that my measure of cognitive response to the hyperventilation challenge may be limited. The cognitive subscale may lack sensitivity to the specific cognitions experienced by high anxiety sensitive individuals when they are exposed to a bodily-arousing stressor. Alternative methods of measuring cognitive response should be considered in future work.

My third hypothesis was partially supported in that somatic reactivity dampening was observed in the alcohol condition relative to both the control and placebo conditions. The absence of differences between the placebo and control conditions is consistent with our suggestion that relatively more automatic aspects of the fear response (i.e., somatic reactivity) should be most highly influenced by the pharmacological properties of alcohol (Stewart et al., 1999) and least influenced by expectancies. However, subjective intoxication ratings were higher in the alcohol condition compared to the placebo condition, indicating that larger magnitude expectancy factors could have been activated in the alcohol condition and could have contributed to the observed somatic dampening effects. A reverse placebo cell (e.g., Wilson & Abrams, 1977) should be included in future work before possible expectancy effects can be ruled out definitively.

In addition, high anxiety sensitive placebo participants experienced reductions in baseline affective responding after consuming the placebo beverage. This replicates findings from Study One and could reflect a dampening of anticipatory anxiety with perceived alcohol intake (see Lehman et al., 2000). However, the failure to find reductions in baseline affective responding in the alcohol condition was unexpected and is inconsistent with this interpretation.

The second part of my third hypothesis was that alcohol expectancy factors would prove important in dampening relatively more controlled aspects of high anxiety sensitive individuals' response to hyperventilation (i.e., affective and cognitive responses to hyperventilation). This did not occur; neither affective nor cognitive dampening was seen in the placebo condition relative to the control Beverage Condition. Placebo condition participants did report significantly higher subjective intoxication ratings than control participants, indicating that the failure to support this hypothesis was not secondary to an unsuccessful expectancy manipulation.

Given a lack of significant support for direct expectancy influences, the findings of Study Two suggest that the pharmacological properties of alcohol are the primary contributors to stress-response dampening effects in high anxiety sensitive individuals. This is consistent with the conclusions reached in other studies using alcohol challenge plus anxiety-provocation protocols across a variety of anxious populations (e.g., Abrams, Kushner, Lisdahl, & Voight, 2001; Levenson et al., 1980). However, the conclusion that dampening effects are *purely* pharmacological in nature must be made cautiously considering that a reverse placebo cell (used to measure pure pharmacological effect of alcohol) was not included in the present study.

Significant differences in hyperventilation responses did not appear between placebo and control conditions. However, it is possible that differential levels of activation of tension-reduction expectancies among participants in the placebo condition may have obscured true differences between placebo and control groups. In fact, the shape of the data suggests that an "inverse placebo" effect (Wilson, 1988) may have been operative. That is, in the absence of an expected feeling of tension-reduction, high

anxiety sensitive placebo participants who had their tension-reduction expectations activated prior to hyperventilation appear to have been *more* reactive to challenge than high anxiety sensitive control participants who did not have their tension-reduction expectations activated prior to hyperventilation. This apparent pattern is consistent with previous results that placebo participants respond with elevated anxiety responses to various stressors compared to a "no-expectancy" control condition (e.g., Abrams & Wilson, 1979).

This idea was tested through a *post-hoc* examination of responses to hyperventilation in subgroups of placebo participants experiencing high versus low levels of expectancy activation, respectively. I used scores on the subjective intoxication visual analogue scale to operationalize the degree to which tension-reduction expectancies were likely to have been activated in high anxiety sensitive placebo participants just prior to hyperventilation. It was assumed that high visual analogue scale ratings (that is, ratings reflecting high levels of perceived intoxication) would correspond to greater activation of tension-reduction expectancies by the smell, taste, and visual cues of alcohol prior to hyperventilation. In contrast, low visual analogue scale ratings (ratings reflecting lower levels of perceived intoxication) were considered to correspond to less activation of tension-reduction expectancies by pre-hyperventilation alcohol cues.

*Post-hoc* analyses revealed trends in which high anxiety sensitive placebo condition participants whose tension reduction expectancies were likely most activated prior to hyperventilation ("high expectancy" participants) appeared to experience greater affective and cognitive reactivity to hyperventilation than those whose tension-reduction expectancies were less activated prior to hyperventilation ("low expectancy"

participants). This pattern of findings, while only marginally significant, is consistent with an inverse placebo effect in which high anxiety sensitive placebo participants may have responded with *increased, rather than decreased*, fear and negative thoughts in response to the induced bodily sensations.

Although expectancy-mediated stress response dampening was not observed in Study Two, the potential existence of an inverse placebo effect among high anxiety sensitive participants nonetheless raises the possibility that tension-reduction expectancies may constitute an additional risk factor for potential alcohol abuse in this population. Siegal (1983) showed that antagonistic placebo responses in animals could provide a Pavlovian conditioning mechanism to explain tolerance, craving, and relapse. Newlin (1989) further suggested that responses in a placebo condition which were opposite in direction and magnitude to responses in an alcohol condition may predict a progression to alcoholism.

Of course, speculations about an inverse placebo effect in high anxiety sensitive individuals must be tempered by the fact that the Expectancy level x Time interactions were only marginally significant for cognitive and affective subscales and cell sizes were small. Moreover, the groupings (i.e., High vs. Low Expectancy level) were not the result of random assignment. This leaves open the possibility of third variable explanations for the pattern of findings. For example, there may be a personality factor (e.g., suggestibility) which leads people to report not only stronger subjective intoxication ratings to placebo, but also stronger emotional responses to hyperventilation. It is also possible that my indirect measure of tension-reduction expectancy activation (via subjective intoxication ratings) lacks the sensitivity to tap into the complex nature of

these expectancies (Goldman, Del Boca, & Darkes, 1999). Future work should address these issues by using a larger sample size, by using a regression analytic approach to test various possible contributing variables to inverse placebo responding, and by evaluating the strength of expectancies more directly through use of a validated measure. For example, McLatchey-Gaudet and Stewart (2001) used the Expectancy Context Questionnaire (Levine, 1988) to successfully measure expectancy strength across a variety of contexts.

In this study, I focussed solely on high anxiety sensitive participants, which raises questions about the specificity of the findings to high anxiety sensitive individuals. Study One and Stewart & Pihl (1994) have provided evidence that high anxiety sensitive individuals are more sensitive than low anxiety sensitive individuals to alcohol's pharmacological stress response dampening properties. However, the design of Study Two cannot determine whether observed inverse placebo effects would be present in all individuals, or merely in high anxiety sensitive individuals, for example. The present design also cannot determine whether, unlike high anxiety sensitive individuals, low anxiety sensitive individuals might show evidence of a direct placebo effect in dampening responses to the hyperventilation challenge. Future work in this area should incorporate a low anxiety sensitive comparison group to explore these possibilities.

In summary, the present findings provide additional evidence that high anxiety sensitive individuals are sensitive to alcohol's pharmacological effects in dampening fear of bodily arousal symptoms. The present data also provide some marginal evidence of alcohol dampening of negative interpretations of bodily arousal in this population. The results taken as a whole support the idea that increased alcohol consumption may be

negatively reinforced in high anxiety sensitive individuals, which could eventually lead to alcohol abuse and dependence. Although the dampening effects of alcohol in high anxiety sensitive individuals do not appear to be influenced by alcohol expectancies, activation of tension-reduction expectancies may contribute to *heightened* fear and negative cognition among high anxiety sensitive individuals who do not experience the pharmacological changes they expect to be induced by alcohol. Sensitivity to such an "antagonistic" placebo response (Newlin, 1989) could be an additional risk factor for alcohol abuse among high anxiety sensitive individuals – a possibility which is deserving of further research attention. Implications of this finding are discussed in more detail in Chapter Five.

# CHAPTER FIVE

## Summary of Main Findings

### Sober Reactivity to Hyperventilation

As expected, Study One demonstrated that sober high anxiety sensitive individuals report more negative cognitions and fearful affect than their sober low anxiety sensitive counterparts in response to hyperventilation. This finding is consistent with the belief system that high anxiety sensitive individuals have about the meaning of bodily arousal sensations. This finding is also consistent with past research which has shown that, in a sober state, those with high anxiety sensitivity display heightened subjective-emotional reactivity to a hyperventilation stressor compared to low anxiety sensitive participants (Asmundson et al., 1994; Donnell & McNally, 1989; Rapee & Medoro, 1994; Sturges et al., 1998). Interestingly, high and low anxiety sensitive participants did not appear to differ in terms of their experience of bodily sensations on the somatic scale of the Hyperventilation Questionnaire when both groups were sober. This finding supports the idea that high anxiety sensitive individuals do not experience a different level of physical arousal than low anxiety sensitive participants, but rather, interpret that arousal differently. That is, consistent with their belief system, high anxiety sensitive participants appear to have judged their arousal as harmful or "catastrophic", leading to increased feelings of anxiety (Stewart & Pihl, 1994; Rapee & Medoro, 1994; Shostak & Peterson, 1990). The low anxiety sensitive group, not sharing this belief system, also did not appear to share the same degree of fear and catastrophic thinking in response to their own arousal. Thus, these findings provide strong support for the idea that state anxiety is

"amplified" by high levels of anxiety sensitivity (Reiss, 1991) in situations where bodily arousal sensations are present, at least when individuals are not intoxicated.

Responses to Alcohol Consumption at Post-drinking Baseline

Alcohol Increases Baseline Somatic Symptoms. Both Study One and Study Two provided results consistent with past research demonstrating the baseline stimulating properties of alcohol (Maisto et al., 1980). In other words, when in a resting state, consuming alcohol results in moderately increased arousal. Regardless of Anxiety Sensitivity Group status, participants in both studies reported elevated somatic (e.g., "dizziness") and cognitive (e.g., "feel like passing out") symptoms after consuming alcohol while in a resting state. Interestingly, although somatic sensations increased after drinking, high and low Anxiety Sensitivity Groups did not differ significantly in terms of cognitive symptoms after alcohol consumption. Assuming that low anxiety sensitive participants are not "catastrophizing" about the meaning of their alcohol-induced bodily arousal, similar levels of cognitive symptoms in high anxiety sensitive individuals may indicate some negative thinking about their somatic experience but not "catastrophic" interpretations of physical sensations. In previous work, I have shown that high anxiety sensitive individuals may actually experience lower increases in resting heart rate than low anxiety sensitive individuals after moderately intoxicating doses of alcohol (MacDonald & Stewart, 1996). Thus, it is possible that high anxiety sensitive individuals may be relatively insensitive to the baseline stimulating properties of alcohol as opposed to bodily arousal-inducing stressors such as hyperventilation, contributing to fewer and less intense catastrophic thoughts than might be expected in response to alcohol consumption. This may explain why the physically stimulating properties of alcohol do

not deter high anxiety sensitive individuals from continued drinking.

Alcohol Effects on Anticipatory Anxiety. Although the baseline measuring periods prior to hyperventilation were intended to be resting states, participants knew that they would be facing a voluntary hyperventilation challenge in the latter stages of the experiment. Thus, a certain level of anticipatory anxiety was expected in advance of the stressor. Study One revealed that, in the baseline resting state, fearful affect decreased more strongly after drinking for high anxiety sensitive participants than for the low anxiety sensitive participants in both the placebo and alcohol conditions. Similarly, high anxiety sensitive participants in Study Two experienced reductions in baseline affective responding after consuming the placebo beverage. These findings suggest that the perceived consumption of alcohol may dampen anticipatory anxiety to a greater degree in high anxiety sensitive individuals compared to low anxiety sensitive individuals (see Lehman et al., 2000). However, this explanation is qualified by two important considerations. First, Study One showed higher overall affective scores among high anxiety sensitive than low anxiety sensitive participants at the pre-drinking baseline. Thus, high anxiety sensitive participants may have had more "room to move" downwards in terms of their affective scores, while low anxiety sensitive individuals may have been subject to floor effects that prevented significant reductions in affective responding from the pre- to the post-drinking baseline. Second, Study Two failed to find reductions in baseline affective responding following beverage consumption in the alcohol condition. It is difficult to conclude that alcohol expectancy would produce reductions in anticipatory anxiety when these reductions were observed in the placebo but not the alcohol condition, especially considering that alcohol consumption did reduce affective responding in Study

One. There are no apparent procedural differences that can account for this discrepant result across the two studies. Clearly, further work is necessary to resolve the inconsistency in results for baseline alcohol effects on affective scores seen between Studies One and Two.

Responses to Hyperventilation Following Alcohol Consumption

Alcohol Dampening Effects on Somatic Reactivity. Both Study One and Two demonstrated that participants who were administered a moderately intoxicating dose of alcohol reported fewer and less intense somatic sensations in response to the hyperventilation challenge compared to participants who received a placebo beverage. Study One showed that this effect was consistent for both high and low anxiety sensitive participants. As the somatic subscale of the Hyperventilation Questionnaire was designed to tap into bodily arousal symptoms (Rapee & Medoro, 1994), the difference noted between alcohol and placebo conditions suggests that alcohol exerted some overall physiological stress-response dampening effects (Sher, 1987). This finding is consistent with the first tenant of the tension reduction hypothesis (Conger, 1956) and with Sher's (1987) stress response dampening theory, both of which contend that alcohol has direct, physiological, stress- (or tension-) reducing properties. In this case, alcohol does appear to reduce the self-reported experience of physical symptoms, at least in response to a hyperventilation stressor.

Similarly, Study Two showed that high anxiety sensitive participants in the alcohol condition reported less somatic reactivity in response to hyperventilation than high anxiety sensitive participants in the control condition. This replicates previous findings of alcohol-induced dampening of the stress response in high anxiety sensitive

individuals in studies that have used a no-alcohol control condition (Conrod et al., 1998; Stewart & Pihl, 1994), and extends these findings by using a stressor (i.e., hyperventilation) that is more theoretically relevant in the study of anxiety sensitivity. However, it should be noted that this finding, while supported by simple effects analysis, emerged from a marginal interaction effect between Beverage Condition and Time. Replication in a larger sample is necessary before the alcohol-control comparison can be used as strong evidence for alcohol's apparent physiological stress response-dampening effects (Sher, 1987).

Although high anxiety sensitive participants experienced physiological stress-response dampening effects in response to alcohol, they did not show a unique receptivity to this effect compared to low anxiety sensitive participants. Thus, while this dampening effect could reinforce the use of alcohol as a coping strategy to deal with bodily arousal symptoms in high anxiety sensitive populations, the somatic reactivity dampening findings do not do not provide support for a specific anxiety sensitivity risk model for alcohol abuse (Stewart et al., 1999).

Alcohol Dampening Effects on Affective Reactivity. Both Study One and Two demonstrated that the self-reported fearful affect of high anxiety sensitive participants in response to hyperventilation was dampened by the consumption of a moderately intoxicating dose of alcohol compared to the placebo condition. No such dampening was present for participants who consumed the placebo beverage compared to the control condition. Moreover, Study One showed that this alcohol dampening effect was specific to high anxiety sensitive participants, as low anxiety sensitive participants did not appear to experience dampening of their affective reactivity. The specificity of this affective

stress-response dampening effect to the high anxiety sensitive group has been previously demonstrated by Stewart and Pihl (1994), but the strength of the current findings is augmented by use of a between-subjects design and a more theoretically relevant stressor. It now appears that high anxiety sensitive individuals have a special receptivity to alcohol-induced dampening of fearful affect in response to stressors which heighten arousal. This supports the anxiety sensitivity risk model of alcohol abuse (Stewart et al., 1999), which contends that alcohol consumption should be negatively reinforced in high anxiety sensitive individuals who use alcohol to cope with the fear associated with bodily arousal sensations, thereby increasing their risk for developing alcohol problems.

Study Two also yielded findings of alcohol-induced affective dampening among high anxiety sensitive participants in comparison to a no-alcohol control condition. Again, this finding replicated previous research in the area which used a no-alcohol control condition (Conrod et al., 1998; Stewart & Pihl, 1994), and extended this research by use of a more theoretically relevant, hyperventilation stressor. Unfortunately, while supported by simple effects analysis, this finding was extended from an interaction between Beverage Condition and Time that was only marginally significant. Replication in a larger sample might yield a stronger interaction effect and add more weight to the finding of alcohol-induced affective-response dampening.

Alcohol Dampening Effects on Cognitive Reactivity. Study One and Two offer some support for the idea that alcohol selectively dampens cognitive components of the fear response in high anxiety sensitive individuals. Simple effects analyses indicated that high anxiety sensitive-alcohol participants did show fewer and less intense negative thoughts in response to the hyperventilation challenge compared to high anxiety

sensitive-placebo participants. However, the interactions which would support this contention were marginally significant in Study One and non-significant in Study Two. Certainly the pattern of the data suggests that high anxiety sensitive individuals are susceptible to cognitive dampening effects, and the results are consistent with affective response dampening seen in this population, but the empirical support is weak. Given these considerations, it is difficult to conclude definitively that high anxiety sensitive individuals are especially receptive to alcohol-induced cognitive reactivity dampening.

Similarly, simple effects analyses supported that notion that high anxiety sensitive participants in Study Two experienced alcohol-induced dampening of negative cognitions compared to high anxiety sensitive participants in the control condition. However, the interaction which would support simple effects testing was non-significant. Thus, the current results represent a weak replication and extension of previous work showing stress-response dampening in high anxiety sensitive individuals when using a no-alcohol control comparison (Conrod et al., 1998; Stewart & Pihl, 1994).

Limitations of Cognitive Reactivity Findings. The results of Study One and Two call into question the notion that alcohol use is strongly reinforcing for the high anxiety sensitive population owing to its ability to reduce catastrophic cognitions. While there does appear to be some degree of cognitive reactivity dampening in high anxiety sensitive individuals in Studies One and Two, the effect is not as robust as in the cases of affective and somatic reactivity dampening effects. This conclusion is borne out by findings of small effect sizes for cognitive dampening in Study Two. However, the failure to find strongly significant interaction effects in Study One and the small effect sizes for alcohol dampening of cognitive reactivity observed in Study Two may reflect, at

least in part, a measurement issue. Owing to a small number of items (6) and constrained descriptions, the cognitive subscale of the Hyperventilation Questionnaire may not be sensitive enough to assess the unique, automatic, and relatively fleeting nature of catastrophic thoughts (Beck, 1995) in response to physiological arousal induction in high anxiety sensitive individuals. Alternatively, accurate retrospective examination of thoughts following the hyperventilation challenge may simply be too difficult for high anxiety sensitive participants who are experiencing feared bodily sensations. In addition, participants were not trained in thought monitoring techniques prior to commencement of the study, nor were they taught to differentiate between catastrophic thoughts, fearful emotions, and somatic sensations. Barlow and Craske (1989) suggest that such training techniques are vital in terms of helping people to understand the components of their anxiety response. These skills might prove particularly beneficial to high anxiety sensitive individuals, whose catastrophic thoughts in response to unpleasant somatic sensations may vary widely. For example, one individual may worry that he might lose control of himself during the challenge, while another might catastrophize about being negatively judged by the experimenter. A third individual may fear a physical catastrophe like serious illness or a heart attack (see Stewart et al., 1997). Also, alternative methods of assessing cognitions should be considered as adjuncts to the Hyperventilation Questionnaire in future studies to more firmly test whether or not alcohol dampens catastrophic thinking about the meaning of bodily arousal symptoms in high anxiety sensitive individuals. For example, experimental participants could keep a simple thought record during challenge, in which they write down thoughts and rate their intensity as they occur (Barlow & Craske, 1989). Such a measure would overcome

retrospective memory problems and allow individuals to express their unique cognitions in response to the hyperventilation challenge. Finally, a short structured interview immediately following the hyperventilation challenge might allow a participant to voice the unique content and intensity of negative thoughts experienced in reaction to the challenge. These thoughts could then be coded and quantified as variables. Many of the suggestions presented here have been further elaborated in a review by Elting and Hope (1995).

Linear Dose Effects on Alcohol Dampening. Study One supports the idea that dampening of anxious reactivity seen in high anxiety sensitive individuals is influenced by the dose of alcohol consumed. That is, as high anxiety sensitive participants consumed larger quantities of alcohol, subsequently attaining higher blood alcohol concentrations, they experienced greater and greater reductions in anxious responding to the hyperventilation stressor. The largest dampening effects were seen for alcohol doses in the legally intoxicating range. This finding may explain why high anxiety individuals frequently consume alcohol to the point of legal intoxication (Stewart, Peterson, et al., 1995; Stewart, Zvolensky, et al., 2001). It appears that the more high anxiety sensitive individuals drink, the more powerful the reward they receive for drinking (i.e., increased relief from fearful affect and catastrophic thoughts). Thus, the most acute, short-term means of "self-medicating" anxiety with alcohol for this population may be to drink excessively. However, as only two doses of alcohol were investigated in the present studies, the dosage of alcohol which would be most rewarding for dampening in high anxiety sensitive individuals remains uncertain. In addition to probable long-term detrimental effects, in the short-term there is likely a point at which increasing doses of

alcohol fail to provide enhanced dampening of stress-induced responses, or even lead to increased anxiety. Greely and Oei (1999) review a number of cases in which high levels of alcohol administration increased anxiety and aggressiveness in experimental participants at higher alcohol levels. Clearly, future work in the area of alcohol challenge and anxiety provocation in high anxiety sensitive individuals should address alcohol effects at higher dosages of alcohol.

Responses to Placebo Consumption Following Hyperventilation

Lack of Expectancy Effects on Stress Response Dampening. As anticipated, alcohol dampened high anxiety sensitive participants' reports of physiological responses to hyperventilation compared to participants who received a placebo or a no-alcohol control beverage in Study Two. The absence of somatic reactivity dampening in the placebo group compared to the control group suggests that expectancy factors alone did not influence this dampening effect and that pharmacological effects of alcohol (or pharmacological factors in combination with expectancy factors) may account for it. This finding is consistent with our proposal (Stewart et al., 1999) that more automatic aspects of the fear response (e.g., physiological reactivity) would be primarily influenced by the pharmacological properties of alcohol, while expectancy factors would not prove to be as influential. However, no dampening of cognitive, affective or somatic reactivity was noted when high anxiety sensitive participants in the placebo condition were compared to participants in the control Beverage Condition. If expectancy factors were operative and actively contributing to stress response dampening of more volitional behaviours (as we suggest in Stewart et al., 1999) in the sober state, it would be expected that placebo participants would show reductions in affective and cognitive reactivity to the

hyperventilation challenge compared to control Beverage Condition participants. Meanwhile, participants in the alcohol condition did experience affective dampening effects, and to a lesser extent, cognitive dampening effects, compared to placebo participants in Study One, and compared to both placebo and control participants in Study Two. This indicates that pharmacological dampening properties of alcohol led to diminution of negative thoughts about bodily arousal and fearful feelings related to that arousal. What remains unknown is whether expectancy factors may have interacted with these pharmacological effects. However, expectancy factors do not appear to act in isolation to produce dampening effects seen in high anxiety sensitive individuals.

Potential "Inverse Placebo" Effects. *Post hoc* analyses suggested that "high expectancy" participants in the placebo condition, whose tension-reduction expectancies were theoretically most greatly activated, appeared to experience more intense fearfulness and catastrophic thoughts in response to hyperventilation compared to "low expectancy" participants in the placebo condition, whose tension-reduction expectancies may not have been as highly activated by ingestion of the placebo. This finding may represent an "inverse placebo" effect, in which high anxiety sensitive participants who strongly expect tension-reduction prior to hyperventilation respond with increased anxiety when actual, alcohol-induced tension-reduction does not occur. This finding is consistent with previous research which has demonstrated elevated anxiety responses to various stressors in placebo conditions compared to "no-expectancy" control conditions (Abrams & Wilson, 1979; Sutker et al., 1982). However, this finding must be interpreted with caution considering that the pattern of results was only marginally significant, the sample sizes involved were small, participants were not randomly assigned to "high" and

"low" expectancy groups, and no direct measure of tension-reduction expectancy activation was administered. Future work could test for the presence of inverse placebo effects more directly amongst high anxiety sensitive participants by increasing the size of the placebo group and by evaluating the strength of alcohol expectancy activation both prior to and immediately following the hyperventilation stressor using a validated self-report scale, such as the Expectancy Context Questionnaire (Levine, 1988). This scale has already been used successfully to measure the strength of tension-reduction expectancies in different contexts (MacLatchy-Gaudet & Stewart, 2001). Linear regression analyses could then ascertain the degree to which increasing levels of expectancy activation vary with increased somatic, cognitive, and affective reactivity.

## Theoretical Implications of Main Findings

### Support for the Anxiety Sensitivity Risk Model of Alcohol Abuse

The anxiety sensitivity risk model of alcohol abuse (Stewart et al., 1999) suggests that high anxiety sensitive individuals are at increased risk of abusing alcohol because they are more sensitive than others to negatively reinforcing effects of alcohol consumption, namely, dampening of the fear associated with bodily arousal sensations. Important elements of the model are supported by the findings in Studies One and Two of the present dissertation. First, demonstration of significant sober emotional hyper-reactivity in high anxiety sensitive participants compared to low anxiety sensitive participants in response to a theoretically relevant stressor suggests that the relationship between anxiety sensitivity and state anxiety levels is significant and positive. That is, when high anxiety participants are exposed to a stressor which creates heightened bodily arousal, such as hyperventilation in this case, they respond with increased state anxiety.

Stewart and Kushner (2001) noted that this anxiety sensitivity-state anxiety relationship is a necessary prerequisite in a state anxiety mediator model of anxiety sensitivity and alcohol abuse. This model states that anxiety sensitivity acts as a personality variable which predicts alcohol use and abuse, with state anxiety acting as a mediator of the anxiety sensitivity-alcohol use relationship. Thus, the increased emotional reactivity seen in high anxiety sensitive individuals, compared to low anxiety sensitive individuals, might make them more likely to turn to alcohol as a coping response for dealing with feared bodily-arousal sensations, owing to alcohol's perceived tension-reducing properties (see Young et al., 1990). As an analogy, picture the reaction that fair-skinned people have to the sun compared to dark-skinned people. Exposed to the sun for the same length of time, both would experience pigmentation changes in their skin. However, the fair-skinned people would be more likely to burn, while the dark-skinned people would be more likely to tan. Thus, the fair-skinned people would probably start using, and continue using, sunscreen to avoid a painful burn, while the dark-skinned people would not feel a need to use sunscreen as often, if at all. Additionally, the sober reactivity findings indicate that high anxiety sensitive participants not only experienced increased somatic sensations in response to hyperventilation compared to their post-drinking baseline scores, but also that they found these symptoms to be more affectively and cognitively aversive than their low anxiety sensitive counterparts. Sher (1987) has suggested that anxiety that is experienced as aversive or debilitating is more likely to lead to use of alcohol as a coping response.

In addition to sober hyper-reactivity, Studies One and Two demonstrate significant alcohol-induced dampening of physiological responding and fearful affect in

high anxiety sensitive participants following hyperventilation. There is also a trend towards alcohol-induced dampening of catastrophic thoughts about the anxiety experience among high anxiety sensitive participants. Furthermore, dampening of fearful affect, and to a lesser degree, dampening of negative cognitions, appears to be specific to high anxiety sensitive individuals. These findings meet the second of Stewart and Kushner's (2001) criteria for a mediator model, namely, that alcohol consumption is potentially rewarding because it offers an effective escape response from feared anxiety symptoms. The finding that this dampening appears to be more powerful in high anxiety sensitive individuals compared to low anxiety sensitive individuals supports the idea that alcohol consumption may be differentially (negatively) reinforced in the high anxiety sensitive population. The apparently unique receptivity to affective and cognitive alcohol-dampening effects in high vs. low anxiety sensitive individuals also highlights anxiety sensitivity as an individual difference variable which could be a predisposing factor in the relationship between alcohol use and stress response dampening (Greely & Oei, 1999). In addition, the finding that increasing doses of alcohol appear to enhance alcohol's reactivity-dampening effects in high vs. low anxiety sensitive participants indicates that excessive alcohol consumption would be encouraged in this population, as high anxiety sensitive individuals learn to drink more to experience more powerful and rewarding dampening effects. Finally, the reductions in affective responses at the post-drinking baseline for alcohol and placebo participants in Study One, and for placebo participants in Study Two, may indicate a potentially negatively reinforcing effect of perceived or actual alcohol intake in terms of reducing anticipatory anxiety (i.e.,

reactivity prior to the stressor). However, this conclusion is not definitive for the reasons mentioned previously.

Overall, my findings support a mechanism whereby high anxiety sensitive individuals are at particular risk for alcohol abuse for three reasons. First, owing to higher levels of cognitive and affective reactivity in high vs. low anxiety sensitive individuals, high anxiety sensitive individuals may develop more unhealthy drinking motivations (i.e., drinking to cope) (Comeau et al., 2001; Cooper, 1994; Cooper, Russell, Skinner, & Windle, 1992; Stewart et al., 1999). Second, given their special receptivity to the dampening properties of alcohol in conditions of heightened bodily arousal, high vs. low anxiety sensitive individuals would be expected to drink in more risky situations (i.e., negatively reinforcing contexts) (Cunningham et al., 1995; Samoluk & Stewart, 1998). Finally, the linear effects of dose on alcohol-dampening in high anxiety sensitive individuals place them at risk for excessive use of alcohol (McNally, 1996; O'Neill et al., 2001; Sher, 1991; Stewart et al., 1999). Unhealthy drinking motivations, drinking in risky situations, and excessive use of alcohol have all been described as particular risk factors for high anxiety sensitive individuals to develop alcohol problems (see our review in Stewart et al., 1999).

The third experimental test of the anxiety sensitivity risk model for alcohol problems, namely, examining whether high anxiety sensitive individuals show an instrumentally conditioned response to consume more alcohol than low anxiety sensitive individuals under conditions of stress, was not tested explicitly in the present dissertation. However, some of our previous research in this area has generated mixed results. In a study I was involved with prior to commencing this thesis research (Samoluk et al.,

1999), we made use of a mock taste-rating task (Higgins & Marlatt, 1975) as an unobtrusive measure of alcohol consumption in high and low anxiety sensitive individuals in a lab-based setting. Participants were asked to rate the taste of a variety of presented beverages, some of which contained alcohol. The dependent variable of interest was the amount of alcohol consumed. Participants in the Samoluk et al. (1999) study were non-clinical male and female young adults assigned to either a solitary or a social (presence of confederates) drinking context. In both contexts, the participants were given a visual-motor task. In the solitary context, high anxiety sensitive participants consumed more alcohol than low anxiety sensitive participants. High anxiety sensitive participants in the solitary condition also consumed more alcohol than high anxiety sensitive participants in the social context. Alcohol consumption in the social context was equal for both high and low anxiety sensitive groups. In addition, measures of negative affect were higher overall in the high anxiety sensitive group than in the low anxiety sensitive group. Moreover, negative affect was positively correlated with alcohol consumption, but only among high anxiety sensitive students. We (Samoluk et al., 1999) argued that these results provided experimental support for previous self-reports of coping-motivated drinking among young anxiety sensitive adults (Stewart, Karp, et al., 1997; Stewart & Zeitlin, 1995). Specifically, findings of increased drinking in response to negative affect among high anxiety sensitive individuals could indicate the activation of a learned response to consume alcohol under conditions of stress within high anxiety sensitive students.

Another study in this area did not yield results that were as promising as those presented in Samoluk et al. (1999). Samoluk and Stewart (1996) attempted to

experimentally manipulate affect in an endeavor to elicit coping-motivated drinking in high anxiety sensitive university students. High and low anxiety sensitive participants were told to prepare for one of two types of self-disclosing interviews while engaged in a similar taste-test procedure to the one used in the Samoluk et al. (1999) study. One type of interview was designed to be anxiety relevant (e.g., questions about anxiety symptoms) while the other was designed to be anxiety irrelevant (e.g., questions about favorite foods) (cf., Maller & Reiss, 1987). While the high anxiety sensitive students drank more alcohol overall in anticipation of their assigned interview compared to low anxiety sensitive students, the two anxiety sensitive groups did not differ in their level of alcohol consumption in anticipation of the anxiety relevant interview. Surprisingly, on the anxiety irrelevant interview, high anxiety sensitive students consumed more alcohol than their low anxiety sensitive counterparts.

Several possibilities have been raised to explain the ineffectiveness of the mood manipulation in eliciting coping-motivated drinking in the Samoluk and Stewart (1996) study (Stewart et al., 1999). For the purposes of this discussion, the most prominent of these involves the apparent ineffectiveness of the stressor. Anticipatory anxiety specific to the anxiety-relevant interview was not appreciably higher in the high anxiety sensitive group compared to the low anxiety sensitive group (Samoluk & Stewart, 1996), and this was despite this procedure being previously shown to be effective in differentially inducing anxiety in high vs. low anxiety sensitive individuals (Maller & Reiss, 1987). However, Maller and Reiss (1987) examined the effectiveness of this procedure for inducing anxiety during the actual interviews/speeches, whereas we modified the procedure to induce anticipatory anxiety as participants prepared for the

speeches/interviews. As noted previously, an experimental setting may require the use of a stressor which has a more direct effect on eliciting the types of somatic sensations that are so aversive for the high anxiety sensitive population (McNally, 1996) in order to test for the existence of negatively reinforced alcohol consumption responses. For example, high anxiety sensitive participants could be challenged by a stressor that specifically leads to bodily arousal (e.g., oral ingestion of yohimbine, $CO_2$ inhalation, or voluntary hyperventilation) and informed prior to the stressor about the types of symptoms that they could expect (and find aversive) in response to the stressor, such as rapid heartbeat, breathlessness, faintness, stomach upset, and nervousness. In anticipation of the stressor, they could be asked to engage in an imaginal exposure exercise (Beck, 1995) in which they think about experiencing upcoming anxiety symptoms during the stressor and predict how frightening each of those symptoms might be.

In addition to potential problems with the stressor in the Samoluk and Stewart (1996) study, it may be difficult to demonstrate the presence of an instrumentally conditioned drinking response in non-clinical young adults with high anxiety sensitivity. While sensitivity to alcohol's stress response dampening effects may be present, development of the operant response may require a certain level of experience with the rewarding effects of alcohol consumption. Young drinkers may lack the necessary number of experiences with alcohol to exhibit a learned response. Finally, there are a number of difficulties in measuring naturalistic drinking behaviour in a lab-based setting. It is possible that lab-based settings may lead participants to believe that alcohol consumption is not a good coping response to stress, especially if they feel that drinking alcohol in the lab might lead to an undesirable outcome (Vogel-Sprott & Fillmore, 1999).

For example, Greely and Oei (1999) noted that experimental participants may inhibit drinking to avoid negative social scrutiny. Greely and Oei (1999) also suggested that the restricted context of the laboratory may limit the particular type and level of stress required to elicit increased drinking. In addition, they noted that the taste-rating procedure may not lead to the quantity of drinking required to see particular alcohol effects. Finally, Sher (1987) noted that drinking behaviour is initiated as a coping response only when other, more preferable options for dealing with stressful situations are absent (e.g., leaving the situation, engaging in relaxation strategies, etc.).

Stewart and Kushner (2001) suggested that tension-reduction alcohol expectancies may prove to be a partial mediator in the relationship between anxiety sensitivity and alcohol usage. The present findings are supportive of the idea that high anxiety sensitive individuals have a heightened sensitivity to the arousal-dampening effects of alcohol. Given the role of actual experiences with alcohol in strengthening alcohol expectancies (Goldman, Del Boca, & Darkes, 1999), it is reasonable to assume that the special receptivity to dampening effects seen in high anxiety sensitive participants may lead to enhanced tension-reduction alcohol expectancies in this population compared to low anxiety sensitive individuals. Again, although not tested explicitly in Studies One and Two, these expectancies could motivate increased alcohol consumption among high anxiety sensitive individuals even if these expectancies themselves are not capable of eliciting tension-reduction effects. Stewart and Kushner (2001) envision tension reduction expectancies as part of the pathway between anxiety sensitivity and alcohol consumption, in that anxiety sensitivity promotes heightened state anxiety levels, which in turn may activate alcohol expectancies for tension reduction,

leading to the increased use of alcohol for coping purposes. Thus, the relationship between anxiety sensitivity and alcohol use may be mediated by elevated state anxiety and tension reduction expectancies. Future research could evaluate this model more clearly by measuring the presence and intensity of relevant alcohol expectancies at various times during an anxiety challenge procedure (see MacLatchy-Gaudet & Stewart, 2001, for relevant methodology of assessing situational shifts in alcohol expectancies) and examining the relationship between expectancies and subsequent alcohol consumption.

Lack of Support for Expectancy Mediated Stress Response Dampening

As outlined in Chapter Three, a model involving expectancy mediation of stress response dampening effects (Sher, 1987; Young et al., 1990) offers a possible explanation for the affective and cognitive dampening effects seen in the high anxiety sensitive participants in Studies One and Two. In particular, the anxiety sensitivity-expectancy model of dampening proposed by Stewart et al. (1999) predicts that components of the fear response theoretically under the greatest degree of volitional control, such as affective and cognitive responses, would show the greatest influence of expectancy factors (Hull & Bond, 1986). Components of the fear response which are considered more automatic, such as somatic responses, would theoretically be most influenced by pharmacological properties of alcohol.

Study Two met several criteria to allow for effective testing of this model. First, a modified (three cell) balanced placebo procedure (e.g., Newlin, 1989; Sayette et al., 1993) allowed for examination of expectancy factors in isolation from the pharmacological properties of alcohol. The placebo deception was demonstrated to be

effective, as subjective intoxication ratings were significantly higher than zero in Study One and higher than ratings in the no-alcohol control condition in Study Two. Second, the elements required to see a situationally-specific, expectancy-mediated outcome of stress response dampening (Vogel-Sprott & Fillmore, 1999) were present. Participants were led to believe that they would be receiving alcohol, they were administered an active placebo beverage with smell, taste, and visual cues for alcohol administration and they experienced somatic, affective, and cognitive changes in the resting baseline state as a result of consuming the placebo beverage. Moreover, the experimental context was consistent with typical, negatively reinforcing drinking contexts for the high anxiety sensitive population. For example, participants were left alone during the alcohol absorption period, consistent with Samoluk and Stewart's (1998) assertion that high anxiety sensitive individuals may be more motivated than others to drink in solitary vs. social contexts. In addition, participants consumed alcohol while anticipating an anxiogenic hyperventilation stressor, consistent with previous research which has demonstrated that high anxiety sensitive individuals tend to drink in situations in which they are experiencing physical discomfort/negative emotional states (Samoluk & Stewart, 1998) or in which they are drinking for coping-related reasons (i.e., to deal with negative affect) (Stewart & Zeitlin, 1995; Stewart, Zvolensky, et al., 2001). Given the mutually reinforcing nature of alcohol consumption and alcohol expectancies (Goldman, Del Boca, & Darkes, 1999), such a context should be predictive of tension-reduction expectancy activation in high anxiety sensitive individuals.

The lack of purely expectancy-mediated dampening of somatic reactivity to hyperventilation, combined with evidence of alcohol-induced dampening of physical

sensations in response to hyperventilation in the alcohol condition, supports one aspect of Stewart et al.'s (1999) model for stress response dampening among high anxiety sensitive individuals. That is, relatively more automatic components of the fear response (e.g., physiological responding) appear to be dampened primarily through the pharmacological properties of alcohol. It is tempting to conclude that somatic reactivity dampening may have been produced by the pharmacological properties of alcohol alone. However, the lack of a reverse placebo cell (Wilson & Abrams, 1977) in the design of Study Two precludes the interpretation that somatic dampening was *purely* influenced by the pharmacological properties of alcohol. Instead, the effects in this cell could be the result of an interaction of pharmacology and expectancy. In fact, higher ratings of subjective intoxication for high anxiety sensitive participants in the alcohol condition indicate that there may have been a larger activation of tension-reduction expectancies for these participants relative to placebo (expectancy only) participants. These increased expectancies, in turn, may have interacted with the pharmacological properties of alcohol to produce the somatic reactivity reductions seen in Study Two. That is, the specific interoceptive and exteroceptive cues elicited by sufficient alcohol consumption may have activated alcohol expectancies for tension-reduction (see review by Goldman et al., 1987). This second interpretation is consistent with the assertion by Goldman and his colleagues that *all* alcohol effects in the real world result from some level of interaction of pharmacological and expectancy factors. The purpose of controlled alcohol challenge work is to merely break down the relative contributions of expectancy and pharmacological factors.

The findings of Study Two do not support the hypothesis that cognitive and affective responses to hyperventilation, theoretically under more volitional control, were dampened via expectancy factors. Given a lack of significant support for direct expectancy influences, the findings of Study One and Two suggest that stress-response dampening effects in high anxiety sensitive individuals may be achieved through the pharmacological properties of alcohol, or some interaction of pharmacological and expectancy factors. This conclusion is consistent with previous research findings in other studies using alcohol challenge plus anxiety-provocation protocols across a variety of anxious populations (e.g., Abrams et al., 2001; Levenson et al., 1980; Sutker et al., 1982). However, pure expectancy influences on dampening cannot be definitively ruled out, as outlined in the following section.

Factors which may have Obscured True Expectancy Effects on Alcohol Dampening. While there is little support from the present findings to suggest that expectancy factors alone contribute to stress response dampening factors, the conclusion that dampening effects are purely pharmacological in nature must be made cautiously. There are several possible reasons why predicted expectancy effects on cognitive and affective reactivity may not have been observed in the current studies. First, it is possible that expectancy factors worked in concert with pharmacological properties of alcohol to produce cognitive and affective dampening effects in high anxiety sensitive participants. In both Study One and Two, participants in the alcohol condition reported significantly greater subjective intoxication ratings than both placebo and control conditions. Thus, increased feelings of being intoxicated may have contributed to increased tension-reduction expectancy activation. Goldman et al. (1987) have suggested that expectancy

activation may require a certain level of gustatory (e.g., smell, taste and tactile feelings of alcohol in mouth and throat), interoceptive (nervous system effects), and exteroceptive (altering of perceptual system) stimuli which may have only been sufficiently present in the alcohol condition. While the placebo beverage did contain a small amount of alcohol, the gustatory, interoceptive, and exteroceptive stimuli sufficient for expectancy activation may have been absent at such a small dosage (placebo participants in both studies only reached a blood alcohol concentration of 0.001%). Several researchers have indicated that stress response dampening effects are likely produced by the interaction of alcohol and expectancy factors (Goldman, Del Boca, & Darkes, 1999; Lehman et al., 2000; Polivy et al., 1976; Young et al., 1990). This idea could be examined by adding a fourth, reverse placebo condition, to the design of Study Two, so that alcohol conditions with and without the presence of potential expectancy factors could be compared directly. However, given the realistically high dose of alcohol used in Study Two, concerns about achieving an effective deception in the reverse placebo condition would remain (Martin et al., 1990; Rohsenow & Marlatt, 1981; Ross & Pihl, 1989).

It is still theoretically possible that expectancy effects contribute to stress dampening without appreciable contributions from the pharmacological properties of alcohol. However, the design of Study Two may have precluded observations of this potential effect, because the small dosage of alcohol in the placebo beverage may not have been high enough to produce the necessary cues (Goldman et al., 1987) for tension-reduction expectancies to be activated. Alternatively, work by Hittner (1995) suggests that tension-reduction expectancies may be differentially activated and influenced by variables such as gender and alcoholic beverage type. In a lab-based study, Hittner (1995)

noted that women endorsed significantly fewer tension reduction expectations in response to mixed drinks than men. Moreover, there was a trend in both genders towards greater endorsement of tension reduction expectancies in response to beer compared to mixed drinks. Given that the current studies consisted of a sample of 65-70% women drinking a placebo beverage which was perceived to be a mixed drink (vodka and tonic), tension-reduction expectations may not have been strongly activated by the placebo beverage as they might have been had the study used beer as the alcohol beverage and low alcoholic beer ("near beer") as a placebo (cf., Martin et al., 1990; Roehrich & Goldman, 1995). Also, anecdotal reports from participants in Studies One and Two indicated that a majority of experimental participants were relatively unfamiliar and inexperienced with vodka and tonic as a mixed drink. Thus, the alcohol cues generated from an active placebo beverage which was unfamiliar may not have had sufficient similarities to a "prototypical" drinking stimulus (Goldman et al., 1987) for these participants. Thus, tension-reduction expectancies may not have been sufficiently activated in the placebo condition to allow for observation of pure, direct expectancy effects on alcohol dampening of high anxiety sensitive participants' responses to hyperventilation. Moreover, without the inclusion of a validated measure of alcohol expectancies (e.g., the Expectancy Context Questionnaire: Levine, 1988), it is difficult to determine whether expectancies were absent, weak or in a different direction than anticipated.

Other Interpretive Models of Alcohol Dampening Effects

Enhanced GABA-ergic Transmission. Although expectancy effects and expectancy/pharmacology interactions cannot be discounted, alcohol-induced dampening of fear responses in high anxiety sensitive individuals in the current studies appears to be

strongly influenced by pharmacological factors. There are several possible mechanisms by which the pharmacological properties of alcohol may have produced dampening effects. First, it has been suggested that the physiological actions of alcohol may increase transmission of the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) in brain regions which have been associated with the production of anxiety (Sher, 1987; Stewart, Pihl, & Padjen, 1992). The resulting enhanced inhibition may act directly to reduce anxiety responses. It is possible that high anxiety sensitive individuals are particularly receptive to enhanced GABA transmission and therefore show increased alcohol-induced dampening of affective, and to a lesser extent, cognitive, reactivity to hyperventilation compared to low anxiety sensitive individuals. Gray (1982) has suggested that individuals who are over-reactive to anxiety-provoking stimuli may have a special receptivity to GABA inhibition from alcohol and other anxiolytic drugs.

Cognitively Mediated Appraisal Disruption. Another model has been suggested which incorporates cognitive processes as mediators of anxious fear-response dampening. In this model, the pharmacological effects of alcohol interfere with the appraisal of stressful information as harmful or threatening (Levenson et al., 1980; Sayette, 1999). Threat appraisal is disrupted when alcohol constrains the spread of activation among nodes which store information about fear and danger in memory and diminishes the power of the stressor (e.g., hyperventilation) to activate these nodes. This model contends that alcohol's stress response dampening effects will be strongest when consumed before onset of a stressor, and when information about the upcoming stressor is vague prior to beverage consumption (Greely & Oei, 1999; Sayette, 1999). Both of these conditions were met in Studies One and Two, in that alcohol administration occurred before the

hyperventilation stressor and participants were not likely to have had prior experience with voluntary hyperventilation. Moreover, considering that it is fearful interpretation of anxiety sensations which theoretically elevates state anxiety levels in high anxiety sensitive individuals (Reiss & McNally, 1985), it is reasonable to assume that high anxiety sensitive participants would be most affected by a process which disrupts appraisal of a stressor as dangerous or threatening. High anxiety sensitive individuals may interpret arousal-related bodily sensations as less dangerous after drinking and thus show a dampened emotional response to the experience of bodily arousal sensations. Low anxiety sensitive individuals, who do not share this belief system about the fearfulness of anxiety sensations, would not be expected to experience the same degree of change in anxious responding as a result of consuming alcohol. The findings from Study One are consistent with this model, in that high anxiety sensitive participants showed increased receptivity to alcohol-induced affective and cognitive dampening effects relative to low anxiety sensitive individuals.

Somatically Mediated Stress Response Dampening. A further model which holds some promise in explaining the dampening effects seen in high anxiety sensitive individuals in the present thesis involves the direct, physiological stress-response dampening effects of alcohol (Sher, 1987). With somatic symptoms of arousal diminished by alcohol, high anxiety sensitive individuals may simply have fewer anxious sensations to fear, and thus exhibit dampened reports of anxious reactivity. Studies One and Two provide some support for this explanation, in that alcohol appears to have overall dampening effects on somatic sensations in response to hyperventilation. However, Study One, which compares low and high anxiety participants, reveals that

patterns of affective responding are more similar to patterns of cognitive responding than patterns of somatic responding across Groups, Beverage Conditions, and Test Times. For example, somatic stress response dampening effects in Study One were not specific to high anxiety sensitive individuals but rather were observed among high and low anxiety sensitive groups alike. In contrast, affective and cognitive dampening effects were restricted to the high anxiety sensitive group. Thus, there is some preliminary support that dampening effects seen in high anxiety sensitive individuals may be cognitively, not somatically, mediated.

Of course, none of the models presented here are mutually exclusive and they may, in fact, overlap. High anxiety sensitive individuals could be particularly sensitive to enhanced GABA-ergic transmission, threat appraisal disruption, *and* reduced fear as a result of pharmacologically dampened somatic sensations after consuming alcohol. Future research should be devoted to teasing these models apart empirically.

The Potential Role of Inverse Placebo Effects

Despite a lack of evidence for expectancy influences on stress response dampening effects in high anxiety sensitive participants, the potential existence of an inverse placebo effect among these participants suggests that tension-reduction expectancies could be playing a somewhat different role than originally hypothesized in terms of risk for alcohol abuse and dependence in high anxiety sensitive individuals. Working with animals, Siegal (1983) demonstrated that placebo responses which were opposite in direction to drug effects could be conditioned in response to the drug-taking environment, ultimately contributing to physiological drug tolerance and craving. He extrapolated his work to humans, noting that "drug-opposite" effects could explain

withdrawal symptoms in drug addicts and present a risk factor for potential relapse. Newlin (1989) further suggested that responses in a placebo condition which were opposite in direction and magnitude to alcohol effects could be a result of prior conditioning and could predict a progression to alcoholism. Extending this idea to the present findings, it is possible that inverse placebo responding seen in high anxiety sensitive participants may place them at higher risk for alcohol abuse. First, as drinking experience grows, tension-reduction expectancies for alcohol may be strengthened (Goldman, Del Boca, & Darkes, 1999) in high anxiety sensitive individuals. Second, if these individuals are exposed to cues associated with alcohol consumption, tension-reduction expectancies may become activated. Third, if this activation occurs before administration of alcohol, these expectancies may contribute to increased physiological and emotional reactivity ("drug opposite" effects) in the absence of pharmacological dampening from alcohol. Thus, high anxiety sensitive individuals might be motivated to consume more and more alcohol to alleviate these aversive "drug opposite" effects that occur in response to drug cues prior to drug administration. Considering that anxiety sensitive individuals are already more fearful of aversive physical sensations than the general public, one could imagine that they might be at particular risk for alcohol problems if, in fact, their tension-reduction expectancies contribute to inverse placebo responding. Alternatively, it is possible that tension-reduction expectancy activation is not necessary for "drug opposite" effects to be experienced by high anxiety sensitive individuals. Mere exposure to alcohol cues could elicit these aversive effects which high anxiety sensitive individuals could then theoretically "self-medicate" through consumption of alcohol.

The potential existence of inverse placebo effects in high anxiety sensitive participants also raises interesting methodological issues. First, future research which seeks to compare alcohol to placebo responding in anxiety-provocation paradigms should include both a control and a placebo beverage condition, especially in stress response dampening studies. If the inverse placebo effect proves to be reliable, then the difference between alcohol and placebo groups may be artificially inflated due to inverse placebo responding in the placebo condition, an effect that could only be detected through inclusion of a control beverage condition. Second, researchers often make the implicit assumption that leading a participant to believe that he/she has consumed alcohol is sufficient to activate expectancies which produce outcomes in the same direction as alcohol effects (Goldman et al., 1987). The current findings suggest a more complex situation. Specifically, the belief that one has consumed an alcoholic beverage may interact with interoceptive and exteroceptive cues generated by an anxiety-provoking stressor (e.g., hyperventilation), leading to unexpected outcomes such as the inverse placebo response observed here. From this perspective, placebo effects which are in the same direction as alcohol-induced stress-response dampening should only occur when participants are strongly convinced that they have consumed alcohol and when strong, contradictory physiological cues are absent. Furthermore, situational factors may influence the ultimate outcome of activated expectancies, depending on whether a particular outcome (e.g., relaxation) is desirable in that situation (e.g., experimental setting) (Vogel-Sprott & Fillmore, 1999). Future research should make an attempt to explore this intriguing possibility.

It should be noted, however, that firm conclusions about an inverse placebo effect in high anxiety sensitive individuals are premature given the marginal significance of the findings and relatively small number of participants assigned to each group in the *post hoc* analyses in Study Two. Replication in a larger sample is required before definitive conclusions can be drawn. Moreover, lack of random assignment to "high" and "low" expectancy groups raises the possibility that another variable may have been responsible for the findings. For example, it is possible that participants in the "high" expectancy group were simply more suggestible than participants in the "low" expectancy group. Higher levels of suggestibility might lead not only to higher subjective intoxication ratings but also to heightened subjective-emotional reactions to hyperventilation. Future alcohol challenge and anxiety-provocation work could isolate expectancy-related placebo and potential inverse placebo effects by experimentally manipulating expectancies (e.g., Breslin & Wilson, 1993; Keane & Lisman, 1980) in high and low anxiety sensitive individuals. That is, participants in different expectancy groups could be told that alcohol is known to either increase or decrease physiological arousal, and then their responses to placebo administration could be examined. It is also possible that my indirect measure of tension-reduction expectancy activation (via subjective intoxication ratings) lacks construct validity in terms of its ability to measure and evaluate expectancies which are quite complex in nature (Goldman et al., 1987; Goldman, Darkes, & Del Boca, 1999; Goldman, Del Boca, & Darkes, 1999). The strength of tension-reduction expectancies could be directly measured using a validated self-report instrument administered at pre-drinking baseline and immediately prior to the hyperventilation challenge, to more accurately assess the degree of activation of tension-reduction expectancies in placebo

participants. Previous research has made use of the Expectancy Context Questionnaire (Levine, 1988) to measure the strength of tension-reduction expectancies in different contexts (MacLatchy-Gaudet & Stewart, 2001). The Expectancy Context Questionnaire is preferable to other self-report inventories such as the Alcohol Expectancy Questionnaire (Brown et al., 1987), in that it assesses situational fluctuations in expectancies.

## Limitations and Directions for Future Research

### Alternatives to Anxiety Sensitivity in Producing Results

Trait Anxiety. One potential limitation of Study One is that groups which differed in terms of anxiety sensitivity level also differed in trait anxiety. This raises the possibility that trait anxiety could theoretically be responsible for increased sober reactivity exhibited by high anxiety sensitivity individuals compared to low anxiety sensitive participants in response to hyperventilation (McNally, 1996; Taylor, 1996; Borden & Lister, 1994; Lilienfeld et al., 1989). However, there is strong evidence to suggest that it is anxiety sensitivity levels, and not trait anxiety, that predict sober responses to a hyperventilation challenge (Asmundson et al., 1994; Donnell & McNally, 1989; Rapee & Medoro, 1994; Sturges et al., 1998).

It is also possible that trait anxiety, as opposed to anxiety sensitivity, could be responsible for increased sensitivity to alcohol dampening. However, a number of research findings argue against this idea. In his review, Sher (1987) noted that alcohol-induced stress response dampening effects of alcohol do not appear to be any greater in anxiety-prone individuals than others. In fact, he noted that trait anxiety and stress response dampening effects of alcohol may be negatively related. These findings are also

supported by Karp's (1993) work demonstrating significant, positive relationships between tension-reduction expectancies and anxiety sensitivity, but not trait anxiety, in an alcoholic sample. Given evidence that expectancies can be generated and strengthened by the effects of alcohol (see review by Goldman, Del Boca, & Darkes, 1999), it seems likely that alcohol dampening effects specific to highly trait anxious individuals should be accompanied by elevations in tension-reduction expectancies.

Future studies should explicitly compare the potential role of trait anxiety to the role of anxiety sensitivity levels in examining both sober reactivity and receptivity to alcohol's stress response dampening effects following an anxiety -provoking challenge. This could be accomplished in an unselected sample through the use of a regression design (cf. Rapee & Medoro, 1994). Alternatively, independent groups of high vs. low trait anxiety participants and high vs. low anxiety sensitive participants could be examined via a 2 x 2 ANOVA design (cf. Donnell & McNally, 1989).

Panic Attack History. There have also been some suggestions in the literature that heightened reactivity to stressors could be a result of a history of panic attacks (Donnell & McNally, 1990), given the relationship between elevated anxiety sensitivity and panic history (e.g., Taylor et al., 1992). But there has been research to show definitively that it is high anxiety sensitivity and not panic history that accounts for sober hyper-reactivity to hyperventilation (i.e., Donnell & McNally, 1989). Nonetheless, to protect against this possibility, participants who met DSM-IV (American Psychiatric Association, 1994) diagnosis of panic disorder were excluded from the analysis in the present set of studies. Moreover, I took care to ensure that groups did not differ in terms of panic attack history (either cued or spontaneous panic attacks) as assessed by the Panic Attack Questionnaire

– Revised (Cox et al., 1992). The equivalence between high and low anxiety sensitive groups in terms of panic attack history is somewhat puzzling, considering that several links have been established between anxiety sensitivity and panic attacks (see review by Cox et al., 1999). For example, Schmidt et al. (1997) have provided longitudinal findings that high anxiety sensitivity leads to later development of panic attacks. It is possible that I did not see predicted differences between groups in terms of panic attack history owing to the relatively young age of the sample (mean age = 21.9 years ). That is, they simply may not have experienced panic attacks yet. Another possibility that should be considered is a self-selection bias which may have reduced the numbers of high anxiety sensitive individuals with a history of panic attacks. High anxiety sensitive individuals who had experienced panic attacks in the past may have declined to participate in the study when they learned about the hyperventilation component, while others, who had not had such experience, were more willing to volunteer. It may also be an issue of power since the present sample size may not have been large enough to detect real differences in rates of panic attacks. Nonetheless, the absence of high vs. low Anxiety Sensitivity Group differences in panic history in Study One reduces the likelihood that sober reactivity and alcohol dampening effects attributed to Anxiety Sensitivity Group membership were actually due to group differences in panic attack history.

Despite careful experimental control, the possibility remains that sober reactivity or the alcohol dampening effects seen in the high anxiety sensitive group could be accounted for by latent or "prodromal" panic disorder. Schmidt et al. (1997) have demonstrated that anxiety sensitivity is a risk factor for the future development of panic attacks, with the usual age of onset of panic disorder occurring in the mid- to late-20's

(DSM-IV; American Psychiatric Association, 1994). As noted previously, the relatively young age of my sample in Studies One and Two might mean that some members of the high Anxiety Sensitivity Group could be predisposed to developing panic disorder later in life, especially if they had previously experienced spontaneous panic attacks, and their resulting reactivity to hyperventilation could be influenced by developmental precursors to panic disorder. Similarly, alcohol dampening of reactivity effects could be specific to individuals who fall into a "prodromal" panic disorder group.

Similar to recommendations made about testing trait anxiety levels, future studies should contrast the potential role of panic attack history with the role of anxiety sensitivity levels in examining sober reactivity and receptivity to alcohol's stress response dampening effects after an anxiety-provoking stressor. As with trait anxiety, an unselected sample could be studied with regression techniques (cf. Rapee & Medoro, 1994) or independent groups of spontaneous panickers vs. non-panickers, and high vs. low anxiety sensitive participants could be examined in a 2 x 2 ANOVA design (cf. Donnell & McNally, 1989).

Blood Alcohol Concentration Differences. Another potential limitation of Study One involves the finding of higher overall blood alcohol concentrations in the high vs. low anxiety sensitive group following alcohol absorption, despite identical alcohol dose calculations and drink mixing procedures for each group. This finding raises the possibility that differences in blood alcohol concentration, and not anxiety sensitivity group status, may have influenced dampening effects seen in the high anxiety sensitive group. However, the magnitude of the difference in blood alcohol concentration, while statistically significant, was small in absolute terms (0.054% vs. 0.044%), and is unlikely

to completely explain post-drinking reactivity differences between Anxiety Sensitivity Groups. The differences in blood alcohol concentrations may be partly attributable to drinking rate. While total volume and alcohol content of the beverages was strictly controlled, the rate of drinking during the consumption period was not regulated. Anecdotal observation suggested that high anxiety sensitive participants tended to consume drinks faster than low anxiety sensitive participants, on average. Consistent with this observation, current qualitative interview work with high anxiety sensitive teens indicates that this group reports drinking quickly in social situations in order to obtain the calming effects of alcohol more rapidly (Comeau, Stewart, Conrod, & Loba, 2002). This differential rate of drinking could account for higher blood alcohol concentrations among high anxiety sensitive participants overall, given findings that suggest that the rate of drinking is linked to peak blood alcohol concentration achieved (e.g., Miller, 1991). A faster rate of drinking among high anxiety sensitive participants is also consistent with increased risk for alcohol abuse and suggests a "riskier" type of drinking behaviour in high vs. low anxiety sensitive participants (Samoluk & Stewart, 1998; Stewart, Peterson, et al., 1995; Stewart & Zeitlin, 1995; Stewart, Zvolensky, et al., 2001). Future research should examine not only the amount of beverage consumed by high vs. low anxiety sensitive individuals in response to experimental manipulations (Samoluk & Stewart, 1996; Samoluk et al., 1999) but also the rate of drinking as a dependent variable in alcohol challenge studies that involve anxiety provocation. It would also be interesting to replicate Study One with strict controls on drinking rate to determine whether the present findings persist.

Potential Gender Influences

A further potential limitation of both Studies One and Two pertains to gender imbalance in the sample (i.e., 65.7% women and 70.8% women respectively). This imbalance is to be expected, in that potential volunteers were originally screened from undergraduate psychology classes where females are typically over-represented (Stewart, Taylor, et al., 1997). As a result, the findings may or may not apply equally to both men and women. Previous research suggests that gender can exert important effects in alcohol challenge work. In some cases, men and women can even show responses in the opposite direction from one another (e.g., de Boer et al., 1993; Newlin, 1989). It is also possible that high anxiety sensitive men and women may differ in terms of alcohol-induced stress response dampening effects. For example, Stewart, Taylor, et al. (1997) noted that high anxiety sensitive males tend to be most concerned with psychological (e.g., insanity) and social (e.g., embarrassment) outcomes in response to anxiety experiences, while high anxiety sensitive females appear to have greater concerns about the potential physical consequences of anxiety symptoms (e.g., serious illness, heart attack). Thus, alcohol might have enhanced dampening effects on women vs. men when the stressor is designed to arouse bodily symptoms of anxiety (e.g., hyperventilation). Conversely, alcohol may exert stronger dampening effects on men vs. women for social stressors (e.g., trying to make a good impression on a member of the opposite sex; cf. Keane & Lisman, 1980). Lab-based alcohol challenge work has provided some support for this idea. In examining a female-only sample, Stewart and Pihl (1994) noted that high anxiety sensitive participants showed a heightened sensitivity to alcohol-induced subjective-emotional responses to a loud noise burst compared to low anxiety sensitive participants. In

contrast, Conrod et al. (1998) tested an all-male sample and found that alcohol-induced dampening of subjective-emotional responses to unavoidable shock was uniform for both high and low anxiety sensitive participants. Similarities between the sexes have also emerged in alcohol challenge work with high anxiety sensitive participants. For instance, increased sober reactivity to stressors has been demonstrated in high vs. low anxiety sensitive participants in both women (Stewart & Pihl, 1994) and men (Conrod et al., 1998). Although it clearly would have been preferable to include gender as an additional independent variable in the current set of studies, my relatively small number of male volunteers did not provide sufficient statistical power to do so. Given the inconsistencies noted above, future alcohol challenge studies should explore the potential moderating effects of gender on the current study findings.

Dependent Measure Issues

Although the Hyperventilation Questionnaire (Rapee & Medoro, 1994) assessed several components of anxious reactivity (somatic sensations, fearful affect, and negative cognitions associated with bodily arousal) in response to the hyperventilation challenge, it represents but one dependent measure in a single response domain (introspective self-report). However, despite some of the limitations of self-report methodology, an introspective account of feelings and thoughts associated with anxiety at least offers a direct measurement of the variables of interest. Self-reports of tension, stress, anxiety, fear, frustration, anger, and physiological state have all been used as dependent measures in alcohol-induced stress response dampening studies (see reviews by Greely & Oei, 1999; Sher, 1987; and Stewart et al., 1999). Importantly, Rapee and Medoro (1994) were able to differentiate high from low anxiety sensitive participants in terms of their

response to hyperventilation by using the Hyperventilation Questionnaire, making it an effective tool for tapping various relevant domains of response to challenge in the present set of studies. In particular, the Hyperventilation Questionnaire appears to be a strong indicator of the fear and negative cognitions associated with high anxiety sensitivity individuals' responses to arousal-induction challenges (Reiss, 1991; Reiss & McNally, 1985; Taylor & Fedoroff, 1999), and thus may prove to be a good measure for tapping into the unique responses of high anxiety sensitive individuals when they are confronted with the sensations that accompany hyperventilation. Moreover, the Hyperventilation Questionnaire, with items including "fear" and "anxiety", offers a manipulation check for efficacy of the threat manipulation used in the present set of studies, in line with Sher's (1987) suggestions about assessing the validity of a stressor.

There has been lively debate over the use of the correct dependent variables in stress response dampening research. Physiological measures such as heart rate (Sayette, 1993), blood pressure (Vogel & Netter, 1989), digital pulse volume amplitude (Stewart, Finn, et al., 1992), electrodermal activity (Finn, Zeitouni, & Pihl, 1990), catecholamine levels (Vogel & Netter, 1989), and coding of facial expression (Sayette et al., 1993) have the benefit of objectivity and provide a direct measurement of somatic responses to a stressor. Unfortunately, there is some uncertainty about what these measures actually mean, given that the relationship between these measures and the construct of "tension" or "anxiety" in not well understood (see review in Greely & Oei, 1999). For example, if heart rate increases, can this be equated with feelings of anxiety? A number of researchers (e.g., Levenson et al., 1980) have demonstrated heart rate increases following alcohol consumption even when the experimental participant reports feeling decreased

feelings of tension. Greely and Oei (1999) caution against using heart rate as an index of anxious responding owing to the fact that heart rate increases after the immediate consumption of alcohol (e.g., Stewart, Finn, et al., 1992), followed by a return to homeostasis, could be misinterpreted as a stress response dampening effect. Regardless, given the often poor correspondence between various dimensions of anxious responding (Rachman & Hodgson, 1974), future research in this area should include direct physiological (e.g., Stewart & Pihl, 1994) and/or behavioural (e.g., Kushner et al., 1997) measures of anxious responding during challenge as supplements to self-report (see Rachman & Hodgson, 1974). Even if physiological/behavioural recordings are inconsistent, they may lead to some interesting conclusions about which of these reactions is most closely linked to the subjective experience of anxiety and which aspects are dampened via alcohol.

Sample Issues

Another potential limitation of Study Two involves the exclusive use of high anxiety sensitivity participants. While the particular receptivity of high anxiety sensitive individuals to alcohol-induced stress response dampening was well established in Study One and in previous research (see Stewart et al., 1999), the design of Study Two prevents the examination of placebo vs. control responses in low anxiety sensitive individuals. Given that low anxiety sensitive individuals do not share the same belief system as their high anxiety sensitive counterparts about the fearful nature of anxiety symptoms, they may not experience inverse placebo effects following hyperventilation. In fact, they may show evidence of traditional placebo effect responding to the hyperventilation challenge.

The only way that this idea can be explored definitively is for future alcohol challenge-anxiety provocation research to include a low anxiety sensitive control group.

Power Issues

Owing to relatively small sample sizes in Study One (17 participants per cell) and Study Two (15-17 participants per cell), it is possible that my analyses may have lacked sufficient statistical power to detect true effects. For example, if more low anxiety sensitive participants had been included in Study One, the increased sample size may have revealed a pattern of results in this group which was more similar to that seen in the high anxiety sensitive group. However, given the similarity of the current results to past, well controlled research in this area (i.e., Conrod et al, 1998, Stewart & Pihl, 1994), it does not appear that true effects were obscured by the present sample size. In fact, the presence of fairly robust effects in the high anxiety sensitive group, despite the small sample size, suggests that the current differences observed between high and low anxiety sensitive groups might become *more* significant if sample sizes were increased.

Effectiveness of Placebo Deception

Participants in the placebo condition gave subjective ratings of intoxication that were significantly higher than zero in Study One. Placebo condition participants also rated themselves as more intoxicated than control condition participants in Study Two. These findings are indicative of a successful placebo deception – placebo condition participants believed that they were consuming alcohol. However, the placebo deception was not perfect, as subjective intoxication ratings in the placebo conditions were not as high as in the actual alcohol Beverage Conditions. This finding is quite common in studies which use a realistically intoxicating dose of alcohol and subjective measures of

intoxication (e.g., Kushner et al., 1996; Polivy et al., 1976; Ross & Pihl, 1989; Wilson & Abrams, 1977). As a result, it is possible that alcohol expectancies may not be as strongly activated in the placebo condition as in the alcohol condition. This poses a difficult methodological problem with no easy solutions. Increasing the alcohol content in the active placebo beverage might improve the deception, but it might also contaminate pure expectancy effects with increased pharmacological effects of alcohol. Alternatively, a different beverage could be utilized for the placebo and alcohol conditions, such as nonalcoholic beer ("near beer") vs. regular beer. Martin et al. (1990) suggest that nonalcoholic beer may be more effective than other placebos in achieving effective alcohol expectancy owing to the close overlap in sensory cues between the nonalcoholic and alcoholic varieties of beer. However, given the relatively low alcohol percentage in most types of beer, there may be practical limitations to its usage in experimental work given the volumes that would have to be consumed to achieve the realistically high blood alcohol concentrations seen in the current studies (cf. Stewart, Peterson, et al., 1995). It appears that we are left with an imperfect methodology that, nonetheless, does appear to provide some level of deception about the true nature of the placebo beverage.

Procedural Issues

As noted in Study One, there was a one year delay in collecting data for the high alcohol dose group. The group was added one year later owing to an inability to effectively target a blood alcohol concentration in the legally intoxicating range during the first year of the study. Thus, participants in the "high dose" beverage condition were not randomly assigned, but rather, were chosen to match the participants in the placebo and low dose groups as closely as possible in terms of demographics, drinking

characteristics and anxiety relevant variables. It is possible that non-random assignment may have introduced uncontrolled error factors into the study. For example, there may have been undetectable cohort differences in the sample from one year to the next which could have influenced the results. However, owing to close matching, a strict standardized testing procedure, experimenter blindness and a lack of significant differences between groups on relevant demographic, drinking and anxiety-relevant variables, potential uncontrolled factors were minimized as much as possible.

## Clinical Implications and Applications

The findings of Studies One and Two have interesting implications in terms of alcohol dependence treatment and research. It appears that the presence of high anxiety sensitivity may contribute to the risk for "self-medicating" anxiety with alcohol (Norton, Malan, Cairns, Wozney, & Broughton, 1989). That is, individuals who fear the experience of anxiety might be more motivated to learn to use alcohol as a coping mechanism because they have an increased sensitivity to alcohol's stress dampening effects. Thus, alcohol use would become rewarding as a means of avoidance or escape from fearful sensations, increasing their alcohol use and placing high anxiety sensitive individuals at higher risk of developing alcohol problems. Given evidence that anxiety sensitivity may be a contributing factor to alcoholism, the current findings therefore hold promise for designing and evaluating primary prevention programs for alcohol abuse among the high anxiety sensitive population.

If non-alcoholic adolescents and young adults who exhibit high levels of anxiety sensitivity could be identified early enough through standardized screening processes, their risk for developing problems with alcohol might be ameliorated through cognitive

behavioural interventions aimed at reducing anxiety sensitivity (see Conrod, Stewart et al., 2000). Consistent with this idea, Comeau et al. (2002) are currently developing a cognitive behavioural treatment plan for reducing coping-related motivations for drinking in high anxiety sensitive teens. Such a treatment program would first involve psycho-education about anxiety sensitivity and its links to increased risk for alcohol abuse. Specifically, at risk individuals could be informed about the catastrophic thinking processes and fearful affect associated with bodily arousal symptoms (as seen in the sober reactivity findings in the present thesis). They could then be educated about the particular receptivity that high anxiety sensitive individuals have to the stress response dampening properties of alcohol, as demonstrated in the current findings. This information would be followed up by a discussion about the risks of developing alcohol problems if high anxiety sensitive individuals become motivated to drink for those reasons.

Next, high anxiety sensitive individuals could be taught thought monitoring techniques to identify cognitive distortions (Beck, 1995) which contribute to fear of anxiety sensations, such as "catastrophizing" about the meaning of bodily arousal sensations as seen in Studies One and Two. Once specific cognitive distortions were identified, individuals could then be taught to challenge this type of thinking through logical analysis in a procedure known as cognitive re-framing (Beck, 1995). For example, the validity of the "fear of having a heart attack" (an item on the Hyperventilation Questionnaire in the present thesis) could be examined by having the at risk individual determine whether chest discomfort becomes more painful with exertion, whether they have a history of heart problems, and calculating the odds of a young adult or adolescent

experiencing a heart attack as a result of feeling anxious (Barlow & Craske, 1989). Once the individual has concluded that an actual heart attack is unlikely, they could start to look at more realistic, alternative interpretations of their physical symptoms (e.g., "My anxiety sensitivity is making me misinterpret a common and harmless byproduct of the anxiety response.")

In addition to challenging and changing anxiety sensitive-specific cognitions, high anxiety sensitive individuals could be taught specific strategies for reducing their physiological level of arousal (as seen in the sober reactivity findings of the present thesis) through breathing re-training and progressive muscle relaxation (Barlow & Craske, 1989). Such training would provide high anxiety sensitive individuals with more confidence in their ability to control their anxiety symptoms and the outcome of those symptoms. For example, individuals who experienced "breathlessness" or "tight muscles" (as seen in sober reactivity measures in the present thesis) could restore themselves to a more relaxed state through breathing and relaxation techniques, thereby reducing the somatic experiences which lead to negative cognitions and fearful affect.

Finally, systematic exposure to interoceptive cues associated with anxiety could help high anxiety sensitive individuals to moderate their fear of bodily arousal symptoms. Techniques like rapid breathing, spinning, and step-ups (Barlow & Craske, 1989) replicate many of the sensations feared by high anxiety sensitive individuals. As high anxiety sensitive individuals consistently experience an absence of catastrophic outcomes in association with self-generated bodily arousal, the conditioned association between fear and anxiety symptoms could be extinguished. Ironically, the voluntary hyperventilation procedure used in Studies One and Two, which elicited anxious

reactivity in high anxiety sensitive participants, could actually become an effective treatment agent if participants were exposed to it often enough! Thus, psycho-education, cognitive re-framing, relaxation techniques, and interoceptive exposure exercises could offer individuals a more effective and healthier means of dealing with anxiety sensitive-related fears before coping-motivated drinking is firmly ingrained. The efficacy of this approach for reducing problematic alcohol consumption patterns and lowering risk for future alcohol use disorders in high anxiety sensitive individuals could be evaluated through longitudinal studies. Treatment and control groups of non-alcoholic adolescents and young adults who exhibited high anxiety sensitivity levels could be compared and contrasted to see if differences emerged in terms of future use of alcohol as a coping mechanism and subsequent alcohol problems.

## Concluding Thoughts

Alcohol abuse and dependence are issues which have a profound impact on our society. The costs to individuals, families, friends, employers, the legal system and the health care system are enormous. Research aimed at identifying high risk populations, isolating the mechanism which places them at risk and providing directions for prevention and treatment is absolutely essential if the human and societal impacts of alcohol abuse are to be addressed. In keeping with this objective, the findings of the present thesis have built upon past work which indicates that a certain segment of the population, namely, high anxiety sensitive individuals, may be at risk for alcohol abuse. In a more specific sense, the current findings illuminate a mechanism by which a high anxiety sensitive individual may turn to alcohol as a coping response for dealing with bodily arousal symptoms owing to a particularly strong receptivity to the stress response

dampening properties of alcohol. Identification of this mechanism may hold the key to preventative treatment strategies for the high anxiety sensitive segment of the population through early screening, targeting beliefs about the meaning of anxiety sensations, and providing these individuals with alternative coping responses which do not involve the consumption of alcohol. High anxiety sensitive individuals already have enough to fear from their physical symptoms of anxiety. Hopefully this work will help to eliminate a far more real and dangerous threat for this population – alcohol abuse and dependence.

Appendix A: Anxiety Sensitivity Index

DIRECTIONS: Read each item and decide which response best represents the extent to which you agree with the item. If any of the items concern something that is not part of your experience, (i.e., "It scares me when I feel shaky" for someone who has never trembled or had the "shakes") answer on the basis of how you expect or think you might feel if you had such an experience. Otherwise, answer all items on the basis of your own experience. Be careful to make only one choice for each item and please answer all items.

1. It is important to me not to appear nervous.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

2. When I cannot keep my mind on a task, I worry that I might be going crazy.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

3. It scares me when I feel "shaky" (trembling).
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

4. It scares me when I feel faint.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

5. It is important to me to stay in control of my emotions.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

6. It scares me when my heart beats rapidly.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

7. It embarrasses me when my stomach growls.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

8. It scares me when I am nauseous.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

9. When I notice that my heart is beating rapidly, I worry that I might have a heart attack.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

10. It scares me when I become short of breath.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

11. When my stomach is upset, I worry that I might be seriously ill.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

12. It scares me when I am unable to keep my mind on a task.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

13. Other people notice when I feel shaky.
VERY LITTLE A LITTLE SOME MUCH VERY MUCH

14. Unusual body sensations scare me.

VERY LITTLE A LITTLE SOME MUCH VERY MUCH

15. When I am nervous, I worry that I might be mentally ill.

VERY LITTLE A LITTLE SOME MUCH VERY MUCH

16. It scares me when I am nervous.

VERY LITTLE A LITTLE SOME MUCH VERY MUCH

## Appendix B: Telephone Screening Script for Potential Participants

We are conducting an experiment to study the effects of alcohol on the response to a breathing task.. If you are interested in participating you will be asked to complete several psychological measures, possibly consume alcohol and participate in a breathing task. Part of the experiment will be videotaped.

Because the experiment involves alcohol consumption, there are a few things I must ask of you:

- First, are you 19 years of age or older?
- (for women) Are you pregnant or planning to conceive in the near future?
- Is there any reason why you cannot consume alcohol or participate in a breathing task, such as medication you might be on or allergies?
- If no, I just want to verify that you are not taking any of the following medications:

amphetamines, analgesics/narcotics, antianginal preparations, antibiotics, anticoagulants, anticonvulsants, antidepressants, antidiabetic drugs, antihistamines, antihypertensive drugs, aspirin/ASA, barbiturates, diuretics, penicillin, sedative-hypnotics, major or minor tranquilizers.

- Also, do you have any of the following medical conditions:

peripheral vascular disorder, hypertension, gastrointestinal disorder, neurological disorder, pulmonary disease, cardiac disease, arterial disease, diabetes, seizure disorder or liver disease.

- Now I am going to ask you a few questions about your normal drinking habits (Quantity and Frequency Questionnaire): Eligible participants must have consumed at least one alcoholic beverage within the last month.
- I'm going to ask you a few more questions about your drinking (Brief MAST): Anyone scoring over 5 must be excluded

There are a few things that I need you to do before your appointment:

- You must abstain from alcohol for 24 hours prior to testing
- You must not eat anything 4 hours prior to testing, but you should eat something 5 hours prior to testing so you don't come in with an empty stomach
- You will be needed for approximately 2-6 hours depending on how quickly your body metabolizes the alcohol.
- We'd also ask that you not drive for 2 hours following the appointment, due to the effects of alcohol on driving ability. You will be compensated either monetarily at a rate of $5 per hour or with credit points at a rate of 1 point per hour, whichever you would prefer.

Would you be willing to participate?

- If yes, assign a participant number and attach to ASI
- Coordinate date and time
- Review abstinence and fasting requirements and tell subject: If you do not meet these requirements I will have to reschedule your

appointment. So please do your best. If for any reason you are unable to meet these requirements or make your appointment please let us know prior to your appointment (Lab#: 494—3793).

Appendix C: Brief Michigan Alcoholism Screening Test

1. Do you feel you are a normal drinker? Yes No 2

2. Do friends and relatives think you are a normal drinker? Yes No 2

3. Have you ever attended a meeting of Alcoholics Anonymous (AA)? Yes 5 No

4. Have you ever lost friends or girlfriend/boyfriend because of drinking? Yes 2 No

5. Have you ever gotten into trouble at work because of drinking? Yes 2 No

6. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking? Yes 2 No

7. Have you ever had the delirium tremens (DTs), severe shaking, heard voices, or seen things that were not there after heavy drinking? Yes 2 No

8. Have you ever gone to anyone for help about your drinking? Yes 5 No

9. Have you ever been hospitalized because of drinking? Yes 5 No

10. Have you ever been arrested for drunk driving or driving after drinking? Yes 2 No

Appendix D: Panic Attack Questionnaire - Revised

A panic attack is the sudden onset of intense apprehension, fear, or terror, often associated with feelings of impending doom. Some of the most common symptoms experienced during an attack are: dizziness, shortness of breath, chest pain or discomfort, and trembling or shaking.

1. To the best of your knowledge, have any of the following members of your family experienced panic attacks? If you do not have a son or daughter, etc., please check 'not applicable'. Please indicate if any of these persons (or you) are adopted.

| YES | NO | NOT APPLICABLE | |
|---|---|---|---|
| _____ | _____ | _____ | mother |
| _____ | _____ | _____ | father |
| _____ | _____ | _____ | brother(s) |
| _____ | _____ | _____ | sister(s) |
| _____ | _____ | _____ | son(s) |
| _____ | _____ | _____ | daughter(s) |

2. Have YOU ever had one or more panic attacks? YES _____ NO _____

If you have experienced one or more panic attacks in the PAST YEAR please answer ALL the remaining questions. If you have not experienced a panic attack or have only experienced a panic attack in a life threatening situation, please go on to the next questionnaire.

a) In the PAST YEAR approximately how many panic attacks have you had? (please circle)

0 1 2 3 4 5 6 7 8 9 10 more than 10

If more than 10, how many? _____

b) In the PAST FOUR WEEKS how many panic attacks have you had?

0 1 2 3 4 5 6 7 8 9 10 more than 10

If more than 10, how many? _____

c) In the PAST WEEK how many panic attacks have you had?

0 1 2 3 4 5 6 7 8 9 10 more than 10

If more than 10, how many? _____

3. a) For approximately how many MONTHS OR YEARS have you been experiencing panic attacks?
_____ years _____ months.

b) What age were you when you had your first panic attack? _____

4. a) Have panic attacks occurred MORE frequently at some time in the past?
YES_____ NO_____

b) Do you think the panic attacks are becoming more frequent?
YES_____ NO_____

c) Do you think the panic attacks are becoming more intense?
YES_____ NO_____

5. What types of places or situations are you avoiding specifically because of fear of having a panic attack?

6. Please indicate how severely you experience each of the following symptoms WHEN YOU ARE HAVING a panic attack?

| | DOES NOT OCCUR | MILD | MODERATE | SEVERE | VERY SEVERE |
|---|---|---|---|---|---|
| a) difficulty breathing | 0 | 1 | 2 | 3 | 4 |
| b) heart pounding | 0 | 1 | 2 | 3 | 4 |
| c) chest pain or discomfort | 0 | 1 | 2 | 3 | 4 |
| d) choking or smothering sensations | 0 | 1 | 2 | 3 | 4 |

| | DOES NOT OCCUR | MILD | MODERATE | SEVERE | VERY SEVERE |
|---|---|---|---|---|---|
| e) dizziness, vertigo, or unsteady feelings | 0 | 1 | 2 | 3 | 4 |
| f) feelings of unreality | 0 | 1 | 2 | 3 | 4 |
| g) tingling in hands and feet | 0 | 1 | 2 | 3 | 4 |
| h) hot and cold flashes | 0 | 1 | 2 | 3 | 4 |
| i) sweating | 0 | 1 | 2 | 3 | 4 |
| j) faintness | 0 | 1 | 2 | 3 | 4 |
| k) trembling or shaking | 0 | 1 | 2 | 3 | 4 |
| l) fears of death or serious illness | 0 | 1 | 2 | 3 | 4 |
| m) fear of going crazy | 0 | 1 | 2 | 3 | 4 |
| n) fear of doing something uncontrolled | 0 | 1 | 2 | 3 | 4 |
| o) feeling of nausea | 0 | 1 | 2 | 3 | 4 |
| p) visual difficulties (e.g., blurring) | 0 | 1 | 2 | 3 | 4 |
| q) auditory difficulties (e.g., ringing in ears) | 0 | 1 | 2 | 3 | 4 |

| | DOES NOT OCCUR | MILD | MODERATE | SEVERE | VERY SEVERE |
|---|---|---|---|---|---|
| r) difficulty concentrating | 0 | 1 | 2 | 3 | 4 |
| s) extremely rapid heartbeat | 0 | 1 | 2 | 3 | 4 |
| t) fear of causing a scene | 0 | 1 | 2 | 3 | 4 |
| u) feeling of anger | 0 | 1 | 2 | 3 | 4 |
| v) thought of escape from scene of panic attack | 0 | 1 | 2 | 3 | 4 |
| w) flushing | 0 | 1 | 2 | 3 | 4 |
| x) fear of drawing attention to oneself | 0 | 1 | 2 | 3 | 4 |
| y) mouth feels dry | 0 | 1 | 2 | 3 | 4 |
| z) feeling of helplessness | 0 | 1 | 2 | 3 | 4 |

other symptoms (please describe)_____

_____

7. a) What is the <u>most severe</u> panic symptom or symptoms you experience?

b) What is the first panic symptom you notice? _____

c) What is the most frightening panic symptom or symptoms for you?

d) Please list any other feelings or sensations that signal the onset of a panic attack for you.

8. In which type of situation have panic attacks occurred? (Please circle one)

   a) unexpectedly, "out of the blue"

   b) in specific situations such as in life threatening situation, being watched or stared at, speaking or acting to an audience, prior to or during test or exams (please specify) _____

   c) both (a) and (b)

9. When a panic attack occurs, generally what is the time speed between the onset of the attack and when the panic is most intense?

   a) very rapid (less than 10 minutes)

   b) moderately rapid (10-30 minutes)

   c) moderately slow (30 minutes - 1 hour)

   d) slowly (more than one hour)

10. How long, on average, does a panic attack last (start to finish)?

    a) a few minutes (0- 10 minutes)

    b) 10-30 minutes

    c) 30 minutes to an hour

    d) several hours

    e) more than one day

11. How much control do you think you have in preventing the OCCURRENCE of any panic attack? (Please circle a number)

| No Control | | | | Some Control | | | | | Total Control |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

12. How much control do you think you have in limiting the SEVERITY of any panic attack? (Please circle a number)

| No Control | | | | Some Control | | | | | Total Control |
|---|---|---|---|---|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |

13. How much distress do the panic attacks cause in your life?

| None At All | Mildly Distressing | Moderately Distressing | Very Distressing | Extremely Distressing |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |

14. To what degree have the panic attacks caused you to change or restrict your lifestyle (e.g., everyday activities, places you go)?

| No Change | Some Change | A Moderate Amount of Change | Quite a Bit of Change | Extreme Change |
|---|---|---|---|---|
| 1 | 2 | 3 | 4 | 5 |

Appendix E: Demographics Questionnaire

Information Sheet

Participant No: _____

Date: _____

Age: _____years Sex: M___ F___

What year of university are you in ? (please circle one):

1 2 3 4

Salary of Family of Origin (present) - Please circle one:

1. up to $10,000

2. $11,000 - $20,000

3. $21,000 - $30,000

4. $31,000 - $40,000

5. $41,000 - $50,000

6. $51,000 - $60,000

7. $60,000 or more

When was the starting date of your last menstrual period?_____

Are you currently taking oral contraceptives? yes no

How many occasions per week do you normally consume alcohol?

_____

If less than one occasion per week, how many occasions per month?

_____

It less than once per month, how many occasions per year?

_____

How many alcoholic beverages do you normally consume per drinking occasion? (Note that one alcoholic beverage = one 12-oz (355 ml) bottle/can of beer, or one 4-oz (118 ml) glass of wine, or one 1-oz (29.6) shot of hard liquor, either straight or with a mixer.)

_____

Appendix F: State Trait Anxiety Inventory – State Subscale

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then choose the response that indicates how you generally feel. 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 how you generally feel.

**Please select your responses from the choices below:**
**1 = Almost Never**
**2 = Sometimes**
**3 = Often**
**4 = Almost Always**

1. I feel pleasant.....1 2 3 4
2. I feel nervous and restless .....1 2 3 4
3. I feel satisfied with myself .....1 2 3 4
4. I wish I could be as happy as others seem to be .....1 2 3 4
5. I feel like a failure .....1 2 3 4
6. I feel rested .....1 2 3 4
7. I am "calm, cool, and collected" .....1 2 3 4
8. I feel that difficulties are piling up so that I cannot overcome them..1 2 3 4
9. I worry too much over something that really doesn't matter .....1 2 3 4
10. I am happy .....1 2 3 4
11. I have disturbing thoughts .....1 2 3 4
12. I lack self-confidence .....1 2 3 4
13. I feel secure .....1 2 3 4
14. I make decisions easily .....1 2 3 4
15. I feel inadequate .....1 2 3 4
16. I am content.....1 2 3 4
17. Some unimportant thought runs through my mind and bothers me...1 2 3 4
18. I take disappointments so keenly that I can't put them out of my mind..... 1 2 3 4
19. I am a steady person .....1 2 3 4
20. I get in a state of tension or turmoil as I think over my recent concerns and interests .....1 2 3 4

## Appendix G: Hyperventilation Questionnaire

Instructions: Please rate the maximum degree to which you are experiencing the following feelings at the present time by placing a circle around the appropriate number.

| Feeling | Not at all | | | Markedly |
|---|---|---|---|---|
| Numbness in extremities | 0 | 1 | 2 | 3 |
| Breathlessness | 0 | 1 | 2 | 3 |
| Buzzing in the head | 0 | 1 | 2 | 3 |
| Feeling distant | 0 | 1 | 2 | 3 |
| Feeling unreal | 0 | 1 | 2 | 3 |
| Fatigue | 0 | 1 | 2 | 3 |
| Fear | 0 | 1 | 2 | 3 |
| Pounding heart | 0 | 1 | 2 | 3 |
| Feeling trapped or helpless | 0 | 1 | 2 | 3 |
| Hot or flushed | 0 | 1 | 2 | 3 |
| Anxiety | 0 | 1 | 2 | 3 |
| Headache | 0 | 1 | 2 | 3 |
| Rising agitation | 0 | 1 | 2 | 3 |
| Feeling of suffocation | 0 | 1 | 2 | 3 |
| Dizziness | 0 | 1 | 2 | 3 |
| Feeling of losing control | 0 | 1 | 2 | 3 |
| Worrying that your actions are damaging to your health | 0 | 1 | 2 | 3 |

| Feeling | Not at all | | | Markedly |
|---|---|---|---|---|
| Tingling in the face | 0 | 1 | 2 | 3 |
| Tight or stiff muscles | 0 | 1 | 2 | 3 |
| Blurred vision | 0 | 1 | 2 | 3 |
| Weakness | 0 | 1 | 2 | 3 |
| Relaxation | 0 | 1 | 2 | 3 |
| Nervousness | 0 | 1 | 2 | 3 |
| Racing heart | 0 | 1 | 2 | 3 |
| Feel like passing out | 0 | 1 | 2 | 3 |
| Nausea | 0 | 1 | 2 | 3 |
| Tingling in extremities | 0 | 1 | 2 | 3 |
| Fear of a heart attack | 0 | 1 | 2 | 3 |
| Band across head | 0 | 1 | 2 | 3 |
| Tension | 0 | 1 | 2 | 3 |
| Feel like panicking | 0 | 1 | 2 | 3 |

Appendix H: Visual Analogue Scale For Subjective Intoxication

Instructions: Please rate the degree to which you feel intoxicated (drunk) by placing a mark on the line below at the appropriate place.

---

**Not at all** **Extremely so**

## Appendix I: Calculating Alcohol Dose

Fisher et al. (1987) suggested a formula for calculating blood alcohol concentrations based on gender and body mass, given the effects of these variables on alcohol absorption rate and blood alcohol concentrations achieved. In particular, they suggested a gender conversion factor to address differential rates of alcohol metabolism between men and women (Julien, 1996). However, pilot testing in the Stewart alcohol lab revealed that this formula overestimated blood alcohol concentrations for relatively young adults in a university population, possibly as a result of not accounting for potentially faster metabolism rates in younger adults. Thus, modifications to the formula were necessary to account for age in addition to gender and body mass. Further pilot testing indicated a conversion factor of 1.82 to account for age in calculating alcohol doses that would bring people to a desired approximate blood alcohol concentration.

The modified formula for targeting a specific blood alcohol concentration is as follows:

$[(C * M * F * B * W)/80]/P$

C = conversion factor = 1.82

M = conversion factor for finding fluid measure = 1.291

F = conversion factor for sex = .583 for males, .485 for females (Fisher et al., 1987)

B = target blood alcohol concentration (in mg per 100 ml blood)

W = weight in kilograms

P = percentage of alcohol you are using

For example, if you were targeting a blood alcohol concentration of .05% using 40% alcohol for a 60 kg female:

C = 1.82

M = 1.291

F = .485

B = 50

W = 60

P = .4

[(1.82 * 1.291 * .485 * 50 * 60)/80]/.4

= 106.8 ml of 40% alcohol would be required.

A mixture of four parts tonic water to one part 50% USP vodka was determined to be a adequate beverage for consumption. The mixed drink was determined to be more naturalistic and palatable than administration of undiluted ethanol.

Appendix J: Informed Consent Form

**NOTE: SUBJECTS MUST READ THIS FORM AND SIGN THE FOLLOWING PAGE TO CONFIRM THAT THEY UNDERSTAND AND ACCEPT CONDITIONS BEFORE EXPERIMENT CAN BEGIN**

**INFORMED CONSENT FORM**

Prepared by the Human Subjects Ethics Committee on behalf of the senior investigator.

Name of staff member responsible: Dr. Sherry H. Stewart

Code Number: 797 – 30 – SS

TO ALL SUBJECTS:

The Human Subject Ethics Committees of the Faculty of Medicine, which have approved this project, require that research using human subjects conform with ethical guidelines currently suggested by most professional and research granting agencies. These guideline require:

(1) That you be informed of the purpose of the research program and any attendant inconvenience risk, or benefits.
(2) That the character of the task required be explained to you.
(3) That you be made aware that Participation is voluntary and that you may decline to continue as a participant at any point during the course of the research project, without loss of expected compensation
(4) That you be assured that all information assembled is entirely confidential.

Please read the following which provides these details about the current research project.

Purpose of the research project: The purpose of the study is to examine the effects of alcohol on breathing (specifically, a paced breathing task).

Task requirements: As a subject, I will be required to complete a series of psychological questionnaires pertaining to my mood and my normal drinking habits. Some of these questionnaires will be administered several times during the course of testing. Next, I will have a series of electrodes affixed to my fingers and chest for recording of my sweating, heart rate, and breathing during the paced breathing task. Next, the experimenter will inform me about the beverage group to which I have been assigned: alcohol or control. If I am assigned to the alcohol group, I will be administered a quantity of alcohol winch is equivalent to 3-4 mixed drinks. If I am assigned to the control group, I will be administered a set of drinks which contain only a tiny amount of alcohol. I will be asked to consume my assigned beverages over a period of 25 minutes. Then, I will be asked to

relax for 30 minutes to allow my body to absorb the beverage. After I consume and absorb the drinks, I will be asked to participate in a paced breathing task for three minutes where I will follow taped instructions regarding the speed and depth of my breathing. The entire session will be videotaped and my physiological responses will be monitored through the recording electrodes attached to my fingers and chest. Following the study, I will be compensated either with came credit points or money.

Hazards, risks, inconveniences, or benefits associated with participation: I may be asked to consume an amount of alcohol-containing beverages equivalent to approximately 4-5 mixed bar drinks. As a result, I may experience mild to moderate physical sensations associated with alcohol intoxication (e.g., dizziness, lightheadedness, and nausea). If I consume alcohol, I will be required to remain in the laboratory following testing until my blood alcohol level reaches half the legal limit (i.e., breathalyzer reading of 0.04% or less); I will be invited to watch a movie on the VCR during this time. Since I may be consuming alcohol-containing beverages during the study, I agree not to drive, or operate machinery for at least 2 hours after completing the experiment.

I will also be asked to participate in a paced breathing task for three minutes, where I will be asked to breathe more quickly and deeply than usual. I understand that this paced breathing task is not dangerous, but that it can produce a variety of sensations in different individuals. I will be permitted to stop the paced breathing exercise at any point during the testing procedure if I find it too discomforting. Also, I understand that one of the experimenters will remain in the same room with me during the paced breathing exercise, should I have any questions or concerns about the procedure.

In addition to completing a variety of pencil and paper psychological measures during the study, I will also have my physiological responses (heart rate, breathing rate, and sweating) recorded prior to and during the paced breathing exercise For these measures, I will be required to wear three electrodes on my fingers and one around my chest. These procedures will cause me no risk or discomfort. I may choose to have the electrodes removed at any point during testing, should I wish.

Compensation: I will be provided with either $5 per hour or 1 credit point per hour towards my grade in psychology, at the end of the study as compensation for my participation. Thus, I may earn from $5-20, or 1-4 credit points, for my participation depending on the amount of time necessary for testing (1-4 hours). Should I choose to withdraw my consent to participate at any point during testing, I will still receive this compensation for my time.

Confidentiality: All of the information that I provide will be treated with the strictest of confidence. My data will be identified only with a code number and not my name, and my records will be kept in a locked filing cabinet. Only the principal investigator (Dr. Stewart) and her assistant(s) will have access to my data.

Please sign below to confirm that you understand the information provided above, and that you are aware that all records are entirely confidential, and that you may discontinue participation at any point in the study. Feel free to address any question you may wish to

the investigator either now or after you have participated. Individuals with specific ethical concerns should contact either Dr. Stewart (494-3793) or a member of the Department of Psychology Human Subjects Ethics Committee.

Participant's Signature:
_____Date:_____

Witness Signature:
_____Date:_____

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